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**THE**

**Science and Practice of Medicine.**

**VOLUME II.**





THE  
SCIENCE AND PRACTICE  
OF  
MEDICINE.

BY  
WILLIAM AITKEN, M.D., EDIN.,  
PROFESSOR OF PATHOLOGY IN THE ARMY MEDICAL SCHOOL.

SECOND AMERICAN, FROM THE FIFTH, ENLARGED AND CAREFULLY  
REVISED, LONDON EDITION,

ADOPTING THE NEW NOMENCLATURE OF THE ROYAL COLLEGE OF PHYSICIANS OF LONDON.

WITH LARGE ADDITIONS,

BY  
MEREDITH CLYMER, M.D.,  
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FORMERLY PHYSICIAN TO THE PHILADELPHIA HOSPITAL; ETC. ETC.

IN TWO VOLUMES.

WITH A MAP, LITHOGRAPHIC PLATE, AND NUMEROUS ILLUSTRATIONS  
ON WOOD.

VOL. II.

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## ADVERTISEMENT.

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THE Preface of the Editor, having been printed whilst the second volume of this work was passing through the press, contains an under-estimate of the additions made by him to the present American Edition. They would amount to *over five hundred pages* of the last London Edition.

Besides the large additions made to the Author's text, *thirty-six* (and not *twenty-two*, as stated in the Preface) articles have been written by the Editor, several of them on subjects now introduced for the first time in any text-book on the Practice of Medicine:

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|--|--|
| 1. Camp Measles.   | 20. Auscultation in Health and in Disease.                                 |
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| 3. Prognosis and Diagnosis of Typhoid Fever.                     | 22. Disease of the Heart, how far a Disqualification for Military Service? |
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| 17. Acute Centripetal Paralysis.                                 | 36. Statistics of Tracheotomy.   |
| 18. Myo-Sclerotic Paralysis.                                     |  |
| 19. Physical Diagnosis of Diseases of the Cerebro-Spinal System. |  |



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# THE SCIENCE AND PRACTICE OF MEDICINE.

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## PART III.—CONTINUED.

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### CLASS II. CONSTITUTIONAL DISEASES.

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#### CHAPTER I.

##### GENERAL REMARKS ON THE PATHOLOGY OF CONSTITUTIONAL DISEASES.

WHILE the GENERAL diseases which have been named ZYMOTIC become developed in the human body under the direct influence of agents acting from *without*, the CONSTITUTIONAL diseases, by contrast, may be said to become developed under the influence of agents generated *within* the body itself, and acting through the continuous exercise of its functions. The original organization of the human body may be of such a kind that the continued and prolonged exercise of its functions, in place of preserving the system in a healthy state, ultimately brings about diseases of various kinds in the daily course of development and growth, which (from this mode of origin and development) have received the name of CONSTITUTIONAL DISEASES. All of them are eventually attended with more or less local manifestations of disease, some of so fixed a character, and so strongly expressed, that the local lesions which may prevail are sometimes looked upon (although erroneously) as constituting distinct diseases; such, for instance, as the joint or heart affection in *rheumatism*, or the bone affection in *rickets*.

The local manifestations of those diseases which belong to the *constitutional* class are invariably the expression and result of a pre-existing unhealthy state of the system, without which no outward, extraneous, or accidental condition could call them into existence; although such conditions might determine (as a stimulus would)

the local development, external manifestation, or expression constitutional disease.

The CONSTITUTIONAL DISEASES are, therefore, all associated with what is termed a *cachectic* state (*cachexia*), or, in common language, "a bad habit of body." They are sporadic diseases, and are sometimes discovered to be hereditary. The lesions which attend them are observed to be rarely limited to one part or organ; and, death ensues, several organs, tissues, or apparatus, not necessarily contiguous to each other, become diseased, and new material of heterologous nature may grow up in their substance.

In the local lesions are to be found those elements which are the "anatomical characters" or "anatomical signs" of the several constitutional diseases.

We do not yet know why one organ or texture should be earlier or more constantly affected than another. It cannot be explained why tubercle selects at one time the bronchial gland as its main *nidus*, and the lungs at another; nor why cancer is by preference the mamma and the uterus; nor why rheumatism affects the white fibrous tissues; nor why rickets affects the bone. The predilections of constitutional diseases to express themselves through lesions of certain organs rather than others are still unexplained.

The course of *constitutional diseases* is generally a prolonged one, with the tendency to repeated attacks or paroxysms of morbid action which exists throughout life; and the local expressions of disease ultimately assume a persistent or chronic type, complicated by, and complicating diseases of another kind, and mutually aggravating each other.

The primary implication of the constitution is, in the greater number of cases, sometimes demonstrable; but sometimes it can be stated only as a matter of fair induction, that the constitution is affected through the blood and the relation of the nutritive processes to it. Pyrexia, when present, is secondary, and generally dependent on the irritation produced by the effort to eliminate some morbid material. The reabsorption of the elements of morbid material, or a persistence of its existence in the blood, tends to affect the blood secondarily; and this secondary blood affects the solids, tends to produce secondary changes in the solids. Thus the blood changes, and the changes in the solids continue to act and react on each other. Thus, also, the constitutional diseases are not traceable to the extrinsic action of a *virus*, and have none of the properties attached to infection. The constitutional diseases are thus apparently generated, developed, and sustained under the influence of an intrinsic blood poison, and the result of perversion of the nutritive or assimilative functions of the individual, and are frequently terminated by congenital constitution or hereditary tendency. "It seems strongly probable," writes Dr. Walshe, "that each member of the group has its specific morbid principle in the blood, unchangeable with the rest, just as any one *virus* is uninterchangeable with others; and that, further, there may exist for each constitutional disease its specific curative agent—an antidote for



poison. In the greater number of these diseases a more or less obvious disposition may be traced to symmetrical arrangement of the anatomical characters of the local lesions, whether these be external or internal."

External physiognomical differences may generally be recognized as distinguishing one man from another, and due to the peculiarities of his own constitution—"personal peculiarities." These are due to what have been called "*temperament*," combined with that character of the constitution which tends to the repeated expression of some form of ill-health, always in the same way, and to which the name of *diathesis* has been given. Such external differences between man and man—such *personal peculiarities*—are known to be transmitted from parent to child, and are then said to be due to *hereditary transmission*. The *tendency* to the expression of certain forms of disease, being thus born with such children, is said to be due to *hereditary predisposition*, and this tendency may be strong and evident, or it may be but feebly and faintly marked. In the former instance it will become expressed in the midst of circumstances even the most favorable to health. Another remarkable feature in the pathology of constitutional diseases requires special notice—namely, that the transmission of the hereditary tendency may fail to be expressed in the *children* of a family liable to diseases known to be so transmitted; and yet the tendency may appear in the *grandchildren*. The tendency is thus expressed in *alternate generations*—the law of "*atavism*," as it has been called. The tendency, thus failing to appear in one generation, may lie dormant, and at last burst forth "in some collateral branch of the family tree;" thus proving that *tendencies* not obviously *expressed* by the parent may nevertheless be *transmitted* by him. A person therefore cannot be considered free from the inheritance of constitutional maladies simply because his parents may not have suffered from any of them; and now it is admitted that, under at least *three generations*, the investigation of hereditary tendency is uncertain.

When one only of the parents is the victim of constitutional disease, the tendency to similar constitutional disease is most obviously expressed in those children who most resemble that parent in physical conformation and appearance; and it has been observed that, when both parents suffer, the tendency will sometimes be expressed more often in the daughters of the family than in the sons, or more often in the sons than in the daughters.

It is especially to be noticed that a marked distinction ought to be made between those which are about to be described as "CONSTITUTIONAL DISEASES" and those which are sometimes called "BLOOD DISEASES." The diseases of the zymotic class, already described, may emphatically be termed "blood diseases"—diseases in which a poison from without affects the blood and establishes a specific disease; but the constitutional diseases about to be noticed are "*blood diseases and something more*." They not only exhibit an aptitude, possessed by those who suffer from them, to assume peculiar forms of morbid action, but their existence stamps upon every other morbid state with which they may be combined a most un-

toward effect. Exudations no longer proceed to healthy resolution. A chill, which otherwise in a healthy man would do no harm, is followed by rheumatic pains and swollen joints in one whose constitution tends to rheumatism; or the growth of tubercle may commence in the lungs of one whose constitution is tuberculous. A bruise is followed by inflammation and an unhealthy suppuration which has been termed scrofulous, in those who are stamped with the diathesis of such a constitutional disease; while the influence of a constitution disposed to attacks of gout or rheumatism is unfavorable for recovery from wounds, injuries, and other diseases. On the other hand, it is to be remembered that local inflammations depending on a constitutional cause are sometimes remarkably fugitive and transient. So much is this the case in idiopathic inflammation, such as a pneumonia or bronchitis, as we know, for several days; but it occasionally happens that affections having all the characters of inflammation will be suddenly established in persons laboring under constitutional diathesis or cachexias, and these inflammatory affections will run their course and terminate in as many hours as the others may take days—they appear at first even more formidable—but the very fact that they occur in a person suffering from rheumatism or gout enables us to give a more favorable prognosis of the immediate result, which would perhaps be warrantable in the case of a person otherwise healthy. This may appear paradoxical; but it is known as a matter of experience that when visceral inflammations appear in the train of rheumatism, for example, they run a materially more favorable course, as a general rule, than if generated under any other influence (GRAVES, WALSH). This fact it is necessary to bear in mind during the examination of all cases of constitutional diseases, for it may help to explain many apparent anomalies amongst the diseases of this class.

Dr. Walshe has specially called attention to the very common and dangerous forms of disease which result when miasmatic diseases (or, as he terms them, acute specific diseases) complicated with the diathesis of constitutional diseases. He shows, further, that constitutional diseases and miasmatic diseases when they coexist, exercise an unfavorable reciprocal influence upon each other (*Med. Times*, 1855).

The reader will therefore be pleased to observe and remember that the term "CONSTITUTIONAL," applied to disease as used in this text-book, is not synonymous with GENERAL or EXTENSIVE, as is by some writers.

The diseases of this class are conveniently arranged into the following orders, namely: (1.) *Diathetic diseases*; and (2.) *Scrofulous, tubercular, phthisical, or wasting diseases*.

## CHAPTER II.

## DETAILED DESCRIPTION OF THE DIATHETIC ORDER OF CONSTITUTIONAL DISEASES.

DISEASES belonging to this order are represented by *rheumatism* and *gout*, also by those affections in which an alteration in the proportions or qualities of the elements of the blood is the obvious and prominent fact, such as *anæmia*, *leucocythemia*, *cyanosis*, and probably the *morbus Addisonii*; and, lastly, by those diseased states of the constitution which manifest themselves by such alterations of the urine as are sufficiently indicated by the *oxalic acid*, the *xanthic oxide*, the *cystic oxide*, the *lithic acid diathesis*, the *diabetic state*, *Bright's disease*, *beriberi*, *rickets*, *asthma*, and the like.

With reference to the term "diathesis," it is to be explained that there are certain states of the human body which physicians have called "morbid dispositions" (*diathesis morbosæ*). The term *diathesis* is used extensively, and sometimes vaguely. By it pathologists have meant to designate often an unknown, impalpable, undefined state of the human constitution, the existence of which is sometimes assumed, or is sometimes legitimately inferred by inductive reasoning, and which then is made to explain the characteristic tendency of some morbid constitutional state, which betrays itself by repeated local manifestations of morbid action (CRAIGIE). A *rheumatic diathesis*, a *gouty diathesis*, a *cancerous diathesis*, a *lithic acid diathesis*, are all thus spoken of. It is now, therefore, generally understood to imply—(1.) The existence of *latent* conditions in the constitution of the body itself, necessary for the development of peculiar diseases; (2.) A tendency to the development of special and peculiar diseases during the course of the nutrition and other morphological changes between the solids and fluids of the body, and which are only influenced by the operations of agents from without, acting as stimuli or excitants to the morbid development.

## ACUTE RHEUMATISM—Syn., RHEUMATIC FEVER.

LATIN Eq., *Rheumatismus acutus*—Idem valet, *Febris rheumatica*; FRENCH Eq., *Rhumatisme articulaire aigu*; GERMAN Eq., *Acuter Gelenk-rheumatismus*—Syn., *Rheumatisches Fieber*; ITALIAN Eq., *Reumapira*—Syn., *Febbre reumatica*.

**Definition.**—*Febrile excitement of a specific kind, probably due to a morbid state of the system by constitutional development, and expressed by inflammation of a peculiar and non-suppurative kind in the parts about or surrounding the joints, especially in the white fibrous tissues—such, for instance, as the sheaths of the muscles and muscular fibres, tendons, aponeuroses, bursæ, capsular ligaments, periosteum, and pericardium. Many joints may be affected at the same time or in succession. The va-*

rious local phenomena of the disease have a tendency to shift from part to part, the most remote from each other; and the febrile state is accompanied by profuse acid excretions from the skin, by the separation, in some cases, of large quantities of uric and sulphuric acid through the kidneys, and by a highly fibrinous condition of the blood.

**Pathology.**—Various opinions have been entertained from time to time regarding the nature of this disease. It was believed to have a miasmatic origin; but the most recent inquirers into the nature of acute rheumatism (BASHAM, GARROD, FULLER) show that it acknowledges no general external source, and it is not even yet demonstrated that any definite offending matter or poison pervades the system. It is presumed, however, by inductive reasoning, that some morbid principle is generated by and within the bodies of those in whom the disease is fully developed, and that it is not absorbed from without.

Evidence of the poisoned state of the blood exists in—(1.) The symmetrical development of the local symptoms—a phenomenon which obtains in all disorders connected with a vitiation of the circulating fluid (BUDD); (2.) The constancy of premonitory fever, or of fever associated with the progressive development of the disease, with a large number of local symptoms, and lesions of internal organs, occurring simultaneously; (3.) A series of observations by Chomel (*Leçons de Clinique Médicale*) tend to show that the internal inflammations in rheumatism, like those of *smallpox*, *typhus*, *scarlatina*, and the like, are referable to an alteration of the animal fluids, and more especially of the blood; (4.) The phenomenon of metastasis, when it does occur, points to a morbid matter in the blood itself as an explanation of the occurrence (HOLLAND, FULLER). The most constant morbid condition of the blood in rheumatism is the abnormal relative proportions which become developed between the *fibrine* and the *saline* elements; the mean of the *fibrine* to *saline* elements being in the relative proportion of 7.163 to 8.47 in 1000. In normal blood the *fibrine* rarely exceeds 3 in 1000.

The following circumstances point to the constitutional origin of this disease—(1.) The victims of rheumatism are apt to experience symptoms clearly denoting at an early period of life certain functional derangement, which leads to impairment of general health (FULLER, TODD, and CHOMEL). (2.) Rheumatic patients are sensitive to atmospheric vicissitudes, prone to perspire, and their perspiration has a sour, disagreeable odor, whilst their urine, though usually clear when passed, not unfrequently deposits, on cooling, a red, buff-colored sediment of lithates and lactates. By such constitutional signs the “rheumatic diathesis” is indicated. (3.) The health of such persons is irritable, and prone to take on inflammatory action, and its nutrition is apt to become perverted. (4.) A change of mode of living has been observed to induce the rheumatic diathesis. (5.) The long continuance and frequent recurrence of symptomatic paroxysms of the disease, also point to a constitutional origin of the *materies morbi* of rheumatism.

It was first suggested by Dr. Prout that all the phenomena of acute rheumatism are referable to the presence of lactic acid developed too freely or abundantly in the system—a suggestion adopted

Drs. Furnivall and Todd, and subsequently by Drs. Headland, Fuller, and Mr. Spencer Wells. Before the starch of the food can be applied to the maintenance of animal heat, it has to be converted into lactic acid, which then combines with oxygen to form carbonic acid and water; and whatever tends to interfere with this normal series of changes from taking place may lead to the accumulation of lactic acid, or other allied acids, in the system.

Dr. Fuller believes the poison of rheumatism to be identical with some natural excretion of the skin, and he grounds his belief chiefly on the following circumstances: (1.) That when the skin's action is interfered with, especially in old people, pains or stiffness of a rheumatic character are generally the consequences; (2.) That the perfect development of the means which nature adopts to relieve these symptoms suggests a relationship between rheumatism and cutaneous excretion. "No sooner is a person attacked by the disease," writes Dr. Fuller, "than excessive perspiration is set up, as if with the view of getting rid of some peccant matter, and the secretion is most profuse at the very part where local inflammation is taking place." Nevertheless, although rheumatism is undoubtedly stamped with a peculiar and specific character, yet the material morbid matter is as yet undiscovered. The exact conditions of the system under which the disease becomes developed are hitherto undetermined. Both Dr. Craigie and Dr. Watson hold that rheumatism implies inflammation of a peculiar and specific kind; and the latter goes a step farther, and writes that "Rheumatism is a blood disease,—that the circulating fluid carries with it a poisonous material which, by virtue of some mutual or elective affinity, falls upon the fibrous tissues in particular, visiting and quitting them with a variableness that resembles caprice, but is ruled, no doubt, by definite laws to us as yet unknown."

Dr. W. B. Richardson made some very important experiments to try whether the theory that superabundance of lactic acid in the system induces pathological phenomena of the rheumatic type admits of any direct demonstration. He injected into the peritoneum of a healthy cat seven drachms of a solution of *lactic acid*, with two ounces of *distilled water*. Two hours after the operation the action of the heart became irregular. The animal was left for the night about six hours after the operation, and in the morning was found dead. The inspection showed *no peritoneal mischief*, but the most marked *endocarditis* of the left cavities of the heart. The mitral valve, thickened and inflamed, was coated on its free borders with firm fibrinous deposit. The whole endocardial surface of the ventricle was intensely vascular. This experiment was afterwards repeated on two healthy dogs. The first dog died on the second day after the experiment, and the inspection revealed the most striking pathological signs of *endocarditis*. The tricuspid valve was inflamed, and swollen to twice its ordinary size. The aortic valve, swollen and inflamed, was coated on its free border with fibrinous beads. The endocardial surface was generally red from vascularity. The pericardium was dry and injected. As before, the *peritoneum escaped injury*. The joints were not affected, but



there was distinct *scleritis* in the left eye. These experiments demonstrate that *endocarditis* may be physiologically produced by lactic acid (*Medical Times and Gazette*, July 18th, 1857). Similar experiments, with similar results, are reported by Dr. Brinkley, and have been made about eleven years previously by Mr. Simon, at St. Thomas's Hospital (*Transactions of Med. Soc. of London*, Jan. 23d, 1858, reported in various Journals).

Whatever, therefore, the poison may be which induces the rheumatic state, it is one which appears to be generated within the system as the result of faulty metamorphosis—that many agents may conduce to the formation of the poison, and to its retention within the system. Whatever the poison may be, it does not seem to be absorbed from without. It is inbred, and not derived from extrinsic sources. The researches of Dr. Parkes lead to the conclusion that it is some substance rich in sulphur.

The parts affected in rheumatism are for the most part the joints, *ments*, *fasciæ*, *aponeuroses*, *periosteum*, *perichondrium*, *tendons*, *bursæ*, and *serous membranes of the heart and brain*; but the joints and surrounding structures are the parts most frequently affected; the heart, the kidneys, and the arteries all sometimes suffer. A red and injected state of the vessels of some of these structures is the only evidence after death of the existence of any approach to inflammatory action, especially in the synovial membranes, the pericardium, or membranes of the brain. This diffuse redness and injection of the vessels may terminate by resolution, or serum may be effused. *Serous* inflammation is extremely common, and is evidenced by the swollen state of the bursæ and parts external to a joint, often by an evident fluctuation within the cavity of a joint; should the patient die, the cavities of the arachnoid and of the pericardium may be found loaded with serum, the latter often to the extent of many ounces.

One of the most frequent results of acute rheumatism is the tendency to thicken parts, and to cause opposing surfaces to adhere. The connective tissue surrounding the diseased articulation is not only found thickened, but infiltrated with a loose coagulum of lymph. The tendinous sheaths and capsular ligaments sometimes exhibit the same alterations. After a time the new material comes consolidated, and in this manner parts are bound down, the motion of joints greatly and sometimes permanently impaired. The alterations of the synovial membrane are not the least curious of the changes which occur in rheumatic joints from the adhesion process. The tissue is not only often thickened, but villous processes, like the papillæ of the tongue of herbivorous animals (soft and red), and dipping into the depressions around the neck of the bone, are occasionally formed, which are intractable even after long treatment, and often lead to destruction of the joint. The strongest evidence of the tendency to thickening, consolidation, and adhesion of parts may be seen in the immense effusion of lymph which takes place in *rheumatic pericarditis*, sometimes covering the whole surface of the heart and pericardium with a layer of lymph half an inch in thickness, and whose irregular surface

been compared to a honeycomb, a calf's stomach, or to the external aspect of a pine-apple.

It is rare that the exudative process in acute rheumatism advances to suppuration. It has been observed, however, sometimes in the muscles, but more commonly within the capsules of the joint (STOHL, CHOMEL, MOREAU, PIORRY, CRUVEILHIER, BOUILLAUD, MACLEOD).

Ulceration of texture in some parts affected is by no means unusual in cases of rheumatism, sometimes perforating capsular membranes or destroying ligaments, but more frequently eroding cartilages and the ends of bones. A remarkable change sometimes takes place in the bones of rheumatic joints when the cartilages have been lost. They become enlarged, and almost eburnified from increased ossific deposit, causing not only a change of form in the articular extremity, but presenting a mechanical obstacle to the motion of the joint. When the hip-joint is affected, the acetabulum becomes deeper and wider than natural, and the head of the femur, flattened and expanded, assumes something of the shape of a turnip.

The parts most commonly affected are the joints, and such textures as are composed principally of the white fibrous tissue. Accordingly, the valvular apparatus of the heart, its fibro-serous covering, the strong white glistening sac of the pericardium, are parts which most frequently suffer. Some joints are more prone than others to be attacked, such joints also being more exposed, as the knees, the feet, the ankles, the wrist, and the hands, are the parts most commonly implicated; next in order, perhaps, follow the elbows, and then the shoulders and the hips. The larger joints suffer more frequently than the smaller, and the small joints of the hands and fingers more commonly than those of the feet. Joints which have been injured, such as those which have been sprained, are more apt to suffer than others; so also those parts which are exercised violently, when that exercise is long continued. There is a tendency to the symmetrical implication of joints.

The constitutional expression of the disease may persist without the implication of any external organ; but not unfrequently the investing or lining membrane of the heart becomes inflamed, and so do the lungs or pleuræ—complications whose existence had been long unnoticed, or at least unconnected with the state of rheumatism.

The heart affections embrace both the immediate and the subsequent dangers in an attack of rheumatism. The occurrence of the heart affection was at one time considered as due to a retrocession of the disease from the external parts, and its consequent transfer to the membranes of the heart. But endocardial or exocardial inflammation may occur as the first, and be for some time the only local symptom of the disease (WATSON, HOPE, HACHE, GRAVES, TAYLOR, FULLER, and others). *Acute rheumatism* and *pericarditis* may also coexist without any articular affection (HACHE, GRAVES, TAYLOR); and the cardiac complication sometimes even *precedes* by *several days* the access of articular redness and swelling;

and even in cases where it does not take place until after inflammation of the joints has been set up, it is rarely preceded or accompanied by subsidence of the previously existing articular inflammation; other words, in the great majority of cases it has been shown no connection can be traced between the two sets of actions, but their origin in one common source of mischief—in one poison which excites inflammation now at one spot and now at another; at one time attacking several joints simultaneously, or in succession then the investing or lining membrane of the heart; at another time reversing the order of attack, and exciting inflammation first of the heart and then of the articular structures. Although rheumatic inflammation of the heart may possibly be connected in some rare instances with the sudden subsidence of articular inflammation and the transfer of irritation from external parts, it must be regarded in most instances as a coincidence, and as an extension of the local manifestations of the disease (FULLER).

As a general rule, like the severe cases of articular rheumatism, the occurrence of cardiac complication may be expected in cases which are remarkable for the severity of their general symptoms. But from the nature of this disease *all cases are liable*; and the cardiac symptoms may for a time be the only ones. Hence has arisen the apparent difference of opinion as to which are the cases peculiarly prone to heart disease. Dr. Latham and Dr. Watson state that “pericarditis is not more looked for when the disease is severe than when it is mild;” but, according to the experience of Dr. Fuller, it is consistent to believe that it often occurs in cases which are characterized by active symptoms of the disease. “Whenever we have met it,” he writes, “even though the articular inflammation may have been slight or evanescent, the febrile disturbance has always been severe, and accompanied by profuse and sour-smelling perspiration.” His experience expressed numerically stands as follows: “That whereas pericarditis occurs *once* in about every 66.5 patients suffering from *acute* rheumatism, it does not accompany the disease above one in every 66.5 cases of the subacute form;” and his experience in this respect is consistent with that of M. Bouillaud, Macleod, William Budd, and Copland. In many instances the heart remains unaffected throughout the attack; and though it does sometimes suffer even in the milder cases, it is most commonly damaged in those instances which are marked by unusual severity of their general symptoms, by the number and intensity of the articular inflammations, and by the rapidity and frequency of their migration. It is found in youth, in women rather than in men, in those persons who have been weakened by illness, or by large repeated bleedings, and in those peculiar states of the system marked by a deficiency of red globules in the blood, when the heart’s irritability is much increased, and palpitation is readily induced. These are the cases in which cardiac inflammation is most liable to arise during an attack of rheumatism (FULLER).

The forms of disease from which the heart is apt to suffer in the cases of rheumatism are—(1.) Inflammation of the pericardium



endocardium; (2.) Inflammation of the substance of the heart itself; (3.) Fibrinous vegetations on the valves and on the lining membrane of the heart, independent of endocardial inflammation.

These vegetations, like other exudations, are doubtless common in endocarditis, but they are essentially independent of endocardial inflammation, and may take place without its occurrence, while endocardial inflammation does not necessarily produce them (KIRKES, ORMEROD, FULLER). These fibrinous deposits seem to be more immediately connected with the abnormal condition of the circulating fluid; for they are almost entirely confined to cases accompanied by acute and wide-spreading inflammation, and by conditions productive of unusual quantities of fibrine in the blood, and which tend to impair its solubility. The statistics of Dr. Barclay, in the *Med.-Chir. Trans.*, vol. xxxi, confirm these observations.

But although endocardial inflammation may fail in the first instance to produce the fibrinous deposits on the valves, yet such endocardial attacks leave behind them a tendency to endocardial degeneration. The most common lesion which thus results is the fibroid degeneration of the valves—a rheumatic lesion which may occur without any articular symptoms. It slowly advances from year to year, with a gradual but constant and more undoubted expression of symptoms; and ultimately the implication of important viscera (*e. g.*, fibroid degeneration of the kidneys, œdema and anasarca) ensues.

Rheumatism is often protracted and rendered complex by other lesions besides those of the heart. The different muscles of the body, their fascia, or tendons, in addition to the joints, or independent of them, are often the seat of rheumatism, and there are few structures of this kind that entirely escape. The scalp, for instance, is often affected. The muscles of the eye are occasionally so: Stohl quotes one case in which the patient squinted while the disease lasted. Rheumatism of the face is by no means unfrequent, and the muscles of the larynx are occasionally affected, causing aphonia. Everybody is familiar with the rheumatic affection termed “crick in the neck,” “*stiff neck*,” and which at the same time may affect the articulations of the clavicle and intercostal muscles. Rheumatism of the abdominal muscles is by no means rare, the principal pain being at their insertions into the *crista* of the ilium. *Lumbago* is well known as a rheumatic affection of the sheaths of the fleshy mass of the lumbar muscles, on one or on both sides of the loins, extending often to the ligaments of the sacrum. The pain is increased by every movement of the back, or pressure on the muscles implicated. The insertion of the *tendo Achillis* into the *os calcis* is another frequent seat of rheumatism; but no parts are more often or more painfully affected than the tendinous structure of the soles of the feet. When the neurilemma of the sciatic nerve is affected, such rheumatism is one form of *sciatica*. *Pleurodynie* designates the rheumatism of the intercostal muscles, or the fibrous fascia which incloses them. These forms of muscular rheumatism are seldom accompanied by any swelling or other external symptom.

When catarrhal affections are prevalent, inflammation of the

lungs or their investing membranes may be expected. An inflammation of the sclerotic coat of the eye is not uncommon; it appears to be most of all liable to occur in cases where a tendency to gout prevails as well as the rheumatic condition. Inflammation of the brain, or of the investing membranes, is the most frightful but happily rare complications. Cases of maniacal delirium, sympathetic and symptomatic, of cardiac pulmonary disease, or of the general vitiated condition of the circulating fluid, have also been recorded.

"It is worthy of note," writes Dr. Fuller, in conjecturing the reason why constant lesions of particular textures occur in rheumatism, "that the textures most commonly implicated are examples of the albuminous and gelatinous tissues, from the composition of which, in the wear and tear of the body, are formed those secondary organic compounds, the lithic and uric acids, with which gout and rheumatism are intimately connected and probably, also, he might have added, the increase of fibrin which John Simon has conclusively indicated. It may be for this cause, therefore, that the rheumatic poison has a special affinity for fibrous and fibro-serous textures throughout the body, and more especially upon those which are in any way subject to irritation. According to the intensity of the febrile disturbance measured by the thermometer, so, *cæteris paribus*, would appear to be the liability to inflammation, whether of the joints, the heart, or any other part of the body; and the increase of temperature above 98.4° Fahr. will give a tolerably accurate measure of the amount of the poison present in the system, and of the patient's susceptibility to its influence. The number and intensity of articular inflammations, and the proneness they exhibit to recur in their quarters, serve also as guides to the probability of heart disease or other internal affections; and the extreme liability to cardiac inflammation, engendered by the repression or rapid subsidence of the articular inflammation, is explicable by the greater quantity of the poison which is thus suddenly thrown into the blood's current." (FULLER).

Rheumatism may be acute or chronic; but the proportion of cases of the latter is infinitely greater than of the former.

The varieties of rheumatism are as follows: (1.) *Acute Rheumatism*—(2.) *Subacute Rheumatism*—*a disorder analogous to the acute, but of moderate intensity, with little or no febrile disturbance;* (3.) *Gonorrhœal Rheumatism*—*an analogous affection associated with gonorrhœa;* (4.) *Synovial Rheumatism*—*a rheumatic affection in which an accumulation of non-purulent fluid occurs in the synovial sacs, and especially those of the knee-joints;* (5.) *Muscular Rheumatism*—*pain in the muscular structures, increased by motion.* The local varieties are—(a.) *Lumbago*, (b.) "*Stiff neck*," (c.) *Chronic Rheumatism*—*chronic pain, stiffness and swelling of various joints.*

**Symptoms.**—Acute rheumatism expresses itself by a severe inflammation of the feet, of the hands, or of the larger joints, as the wrist, ankle, knee, hip, elbow, and shoulder-joint, or of one or more of these parts, and this is usually accompanied by severe

inflammatory fever. These affections often constitute the whole disease; but in some cases, either with or without the subsidence of the articular inflammation, the heart or pericardium, or the membranes of the brain, become the seat of rheumatic inflammation. The proportion of persons whose heart is thus affected probably varies according to the treatment, and other circumstances noticed in the former section. Bouillaud estimates the number at more than one-half, or as 64 in 114 cases, and Dr. Macleod at one-fifth. The affection of the membranes of the brain is much more rare, but the proportionate number is not determined.

In an attack of acute rheumatism the fever often precedes by twenty-four or forty-eight hours the inflammation of the joints; but this is not constant, for in some instances the local and general symptoms are contemporaneous, while in others the inflammation of the joints is established before the accession of the fever.

The fever which attends acute rheumatism is well marked and striking, and symptoms of functional derangement present themselves long before its full development. Before actual fever is established, the patient feels "out of sorts," and unusually sensitive to atmospheric vicissitudes; he looks pale, with a sallow, unhealthy complexion, and a dull eye with yellowish conjunctivæ. The chilliness or shivering with which, in common with other acute fevers, it is ushered in, speedily passes away, and is followed by great heat of the skin, and by copious but partial perspiration, almost invariably acid, reddening litmus paper, and of a disagreeable sour odor. It is a mistake to suppose that much perspiration is useless. It is nature's cure for the disease. It may be "wasting and enfeebling," as excessive perspiration always is, but it is highly sanative; and if it does not occur, the pains are always more excessive, and the constitutional symptoms become more severe if perspiration should unexpectedly cease. The *materies morbi* is obviously got rid of by the sweating, and the natural cure of the disease is effected by these profuse sour-smelling perspirations. Those perspirations are only useless when they are not of this characteristic sour description. They are then emphatically "useless, wasting, and enfeebling," and ought to be arrested. The urine in *acute rheumatism* presents the strongest type of the so-called febrile urine (PARKES, *l. c.*, p. 286). It is scanty, of high specific gravity, deeply pigmented, and deposits, *on cooling*, deep-colored sediments of urates. It very much resembles pea-soup. The water is lessened; the total solids augmented (chiefly by increase in urea and pigment); and the urea, in most cases, is very considerably augmented. Dr. Parkes has observed an excess of *one-fourth* or *one-fifth* over the physiological amount; and the amount is greater in equal periods during the day than during the night; and when the very spare diet of rheumatic patients is considered, the great disintegration of nitrogenous tissue is obvious,—represented, as it is, by a daily excess of from ten to twenty *grammes* of urea. The uric acid at the same time is somewhat increased; and there is usually a great quantity of water passed during the height of the disease, and at the commencement of

improvement, or some days afterwards. The chlorides are diminished, and sometimes disappear. Dr. Parkes has found a very great increase in the elimination of sulphuric acid, sometimes double that passed during convalescence. The amount of others have shown no increase. The pigment and extra are always greatly increased. Albumen appears in some generally small in amount, and very transient; the kidneys much less frequently, and much less profoundly, than in other diseases where the system generally is so much out of order. The pulse rises to 90, 100, and 110, and is large, full, and strong, and if the temperature rises above  $104^{\circ}$  Fahr., the chances of danger increase, and the case is a severe one. The tongue is usually covered with a creamy-like covering, and is greatly loaded with a yellowish-white mucus; the bowels sluggish; the evacuations dark and offensive. There are many remarkable differences between this and the phenomena of continued fevers; for it runs its given course, is not marked by changes of the tongue, nor by great depressing action; while delirium and headache even are a rare occurrence.

Dr. Sydney Ringer, the Professor of Materia Medica in University College, has made some very accurate and extremely interesting observations on the range of temperature in three cases of acute rheumatism, and he has kindly permitted me to make use of his MS. notes.

The concurrent phenomena in one of his cases (a man twenty-three years of age) may be shortly stated as follows:

On the evening of the *third day*, two days after rigors, the temperature in the axilla indicated  $102.6^{\circ}$  Fahr.: at ten o'clock at night of the same day it had fallen to  $101.4^{\circ}$ . The face was flushed, eyes bright, the lips rather dry, the skin very hot to the feel, dry, the tongue very red and rather dry; and thirst was greatly complained of. The bowels, hitherto confined, had been opened by medicine. Pulse, 150; respiration, 36. Some dulness under the right clavicle. Enlargement of the liver.

On the morning of the *fourth day* (at nine A.M.) the temperature had fallen to  $100^{\circ}$  Fahr.; by noon of the same day it again rose to  $103^{\circ}$ ; in the afternoon it fell to  $102.6^{\circ}$ ; and by ten o'clock at night of the same day it had again risen to  $103.4^{\circ}$ .

He had passed a quiet night, without delirium, sweated much, looked better in the morning, was less flushed, and had a moist skin. Much pain and tenderness in both shoulders for the first time yesterday. Seized with pain in the left knee during the night, which was very severe. Left knee is greatly swollen; the swelling is so great as to push the patella before it, rising some distance above and resting on that bone. No local redness, but some heat; has had frontal headache for several days; tongue red and rather dry at the tip; thirst less; bowels opened several times during the night; edge of liver rather rounded, can be felt as low as the umbilicus; heart apparently displaced somewhat towards the middle line—sounds normal—no friction; pulse, 120, weak; respiration not hurried; no action of the nares; urine contains a large quantity of lithates and a small

amount of albumen. No casts or blood seen on microscopic examination.

On the morning of the *fifth day* (at nine A.M.) the temperature had fallen to  $103^{\circ}$ , where it remained until three o'clock P.M. At seven in the evening it had fallen to  $102^{\circ}$ , after which it began to rise, and continued to rise till ten o'clock at night, when the thermometer indicated  $104.6^{\circ}$  Fahr.

He had slept badly during the night, and perspired freely. His complexion has acquired a sallow appearance, and the conjunctivæ are jaundiced. *Sordes* exist about the teeth and gums. Pain in both shoulders, in elbows, and in left knee; no pain in other joints, nor in the head. Tongue red and dry; much thirst; bowels open twice; heart healthy. About ten o'clock at night he complained of great pain over the lower part of the sternum; and friction is heard with the systole at the base of the heart; and there is pain in the chest, which is rather increased by breathing.

On the morning of the *sixth day* (at nine A.M.) the temperature had fallen to  $101^{\circ}$ , and by noon had only risen  $\frac{1}{8}$ ths of a degree; but by four P.M. the thermometer indicated  $104.8^{\circ}$ , after which it continued to fall till 9.30 P.M., when it indicated  $100^{\circ}$  Fahr.

He had passed a very restless night, and was delirious. The features appear shrunken and very sallow; the conjunctivæ yellow. *Sordes* exist on the gums and teeth; sudamina on the chest; sweated freely; pain in the right shoulder and elbow, none in the left. No redness, no swelling, and no tenderness save only on movement; but the pain and swelling still continue in the left knee; pulse 106, weak; friction at the base of the heart, the same as the previous night; bowels moved once daily, the motions being liquid, and of a pale yellow color.

On the morning of the *seventh day* (at nine A.M.) the temperature was  $104^{\circ}$ ; at noon of the same day it had fallen to  $102^{\circ}$ ; in the evening (six o'clock) it registered  $101.6^{\circ}$ , after which the temperature continued to rise slightly till nine at night, when the thermometer indicated  $102.2^{\circ}$ .

He had been delirious during the night. *Sordes* exist on the teeth and lips; face and chest distinctly but not intensely yellow; perspired much during the night. No pain when at rest, but on movement there is pain in the right shoulder and elbow, also in both knees. Right knee is now tender, but not swollen; tongue dry and red; motions pale; much thirst; heat as before; slight cloud of albumen in the urine.

On the morning of the *eighth day* (at nine A.M.) the thermometer indicated a temperature as high as  $104.2^{\circ}$  Fahr.; in the afternoon (two P.M.) it had fallen to  $103^{\circ}$ , and rose again slightly in the afternoon.

Emaciation has advanced rapidly; he lies in an apathetic state, with the eyes partially open, and mucus begins to cover the cornea. Conjunctivæ finely injected. Delirium during the night, but replies to questions reasonably in the morning; yellowness increased; right knee much more swollen, the left less so; tongue very dry; no in-



crease of dulness over the heart; slight friction-sound still at the base; no endocardial murmur; urine high-colored, no albumen.

On the morning of the *ninth day* the thermometer indicated 101.4° Fahr. By noon of the same day it fell to 101.4°. It rose again in the afternoon, and remained at 102° Fahr. at nine P.M.; is getting worse and losing flesh rapidly. He lies with his eyes almost closed, noticing nothing, but answering questions correctly; yellow more marked in the conjunctiva; right knee swollen; swelling of right ankle; liver much less enlarged—a distance of four fingers breadth between its margin, as now felt, compared with the distance which it formerly reached; motions liquid and dark-colored; no friction-sound.

On the morning of the *tenth day* (at nine A.M.) the temperature had risen to 103°, but fell towards noon to 102°. In the afternoon it had again risen to 103.2°, after which it again began to fall.

On the morning of the *eleventh day* (nine A. M.) the thermometer indicated 102.8°, and by noon it had fallen to 102°. During the afternoon it rose again steadily, and by ten at night it registered 103.4°.

On the morning of the *twelfth day* (at nine A.M.) it had fallen to 101°, but by noon it had risen to 103.3°, after which it fell in the afternoon to 102.2°, but rose in a short time again to 103.4°, fell in an equally short time to 102°, where it remained at eight o'clock at night.

On the morning of the *thirteenth day* (nine A.M.) the thermometer had risen to 105.4° Fahr., when he was moribund; and it remained at that high temperature till death in the afternoon.

At the post-mortem examination an abscess was found in the right sterno-clavicular articulation, and much clear tenacious fluid of a yellow color was found in many of the joints.

Universal pericarditis surrounded the heart, but no fluid effusion existed to any extent.

The liver weighed eighty ounces, the left lobe being enormously enlarged, and the right diminished. Its substance was dense and notched, giving a lobulated appearance to the organ. On section it is very firm and rather pale. The lobules are very much enlarged, and distinct from each other. An excess of fibrous tissue is seen by the microscope to surround the lobules, the cells of which look pale and free from granules. The spleen was much enlarged but normal. The kidneys were much enlarged and congested; the pyramids were marked by opaque streaks of fat.

This case and others indicate remarkable vacillations of temperature, occurring irregularly during the day; and they show a high temperature, met with occasionally before death—in some cases as high as 109.6° and 110°.

If the student will project upon paper, in the same way that the records have been already indicated in diagrams, the ranges of temperature so minutely recorded by Dr. Ringer, he will be better able to appreciate, at sight, the vacillations of temperature referred to.

These phenomena are associated with acutely painful, hot, swollen joints; thus the local symptoms which accompany the inflammation of the articulations are pain, heat, redness, and tenderness.

faction. The *pain* is generally active and severe, although in a few cases it is latent—that is, the patient is at ease, unless the joint or limb be moved. At first the pains may wander capriciously from limb to limb, and produce more or less temporary stiffness. It has many degrees of intensity, being in a few instances trifling, but more commonly atrocious and agonizing; and, though generally constant, it is sometimes intermittent. In all cases in which it exists it is greatly augmented by pressure, so that the slightest touch—even the weight of the bed-clothes—is insupportable; it usually somewhat remits during the day, and is aggravated at night. The *heat* of the inflamed joint is constantly increased, the thermometer indicating a temperature of 100°, 105° Fahr., or even more. *Redness*, though not universally present, is nevertheless the rule, and the affected joint is surrounded by a rose-colored blush, evanescent on the slightest pressure, yet returning on its removal. The *tumefaction* of the part is generally so considerable that the shape of the hand, the ankle or other joint is completely destroyed. In affections of the knee the patella is often more or less displaced by effusion into the cavity of the joint; and this, together with the swelling of the external parts, obliterating all the markings of its healthy state, renders the knee misshapen and rounded, “the surrounding skin becomes dry, tense, and shining—so much so, indeed, that experience alone enables us to predict that suppuration will not take place.”

One of the most remarkable and suggestive facts in regard to rheumatism is, that “the fever and constitutional distress are not always commensurate with the extent and intensity of the local symptoms.” The rheumatic inflammation of the joints is very frequently *preceded* by febrile disturbance; and sometimes the fever runs so high before any local symptoms have been established, that even cautious and intelligent practitioners may mistake the nature of the impending attack. Moreover, when febrile symptoms do thus precede the establishment of local inflammation, they are not only not increased by its occurrence, but they are very generally relieved, the pulse becoming calmer, the countenance less anxious, and the patient altogether easier (SYDENHAM, FULLER).

Such are the general and local expressions of a diseased state of the system in acute rheumatism; and at the height of the disorder it is difficult to conceive a more complete picture of helplessness and suffering than that to which the patient is reduced. A strong and powerful man, generally unused to disease, lies on his back motionless, unable to raise his hand to wipe the drops which flow fast from his brow in the paroxysms of pain, or the mucus which irritates his nostril. Indeed, he is so helpless that he is not only obliged to be fed, but to be assisted at every operation of nature. The sweat in which he lies drenched seems to bring him no relief; his position admits of no change; if he sleeps, his sleep is short, and he wakes up with an exacerbation of suffering which renders him fretful, impatient, and discontented with all around him.

The duration of acute rheumatism is various; in some cases both the fever and local pains are gone in three or four days, but in the

majority of instances they continue till about the tenth or fourteenth day, when the fever disappears and the pains subside; and towards the close of the third week, or the beginning of the fourth, the patient is recovered, and generally without any permanent mischief to the joints affected. In almost all cases, however, the pains continue till after the fever is gone, and sometimes for a very long period afterwards. Dr. Fuller's observations lead him to conclude that, under ordinary methods of treatment, the disease continues from four to five weeks, and patients are generally able to leave hospital about the end of the sixth week; and he considers the average duration of an uncomplicated attack may be reduced by judicious treatment from a month or five weeks to ten days or a fortnight. The patient, though recovered, is liable to relapse, and often suffers from it.

The symptoms which indicate cardiac mischief are pains and tenderness all over the chest, increased on pressing between the intercostal spaces, and also on taking a deep breath. The patient is restless, his countenance anxious, and occasionally he coughs. On applying the stethoscope to the chest a murmur may be heard, but it is not permanent, and evidently arising from some irregular contraction about the orifices of the heart or from some affection of the pericardium. Many physicians believe that the exact pathological state of the pericardium can always be determined. Thus, if the inflammation be diffuse, we shall have a crackling sound like that of new leather, the parts being dry; or if serum be effused, we shall find the heart moving in a larger space than usual. Again, if lymph be effused, we shall have a rubbing sound; and, lastly, if pus be effused, it will be determined not only by the greater space in which the heart moves, but by the sudden collapse and rapid sinking of the patient. (See "Pericarditis," &c.)

If the disease be severe and neglected, the patient may die in three or four days, of this secondary affection; but under proper treatment the complication seldom continues beyond a week. If this attack be altogether neglected, and the patient survives, the pericardial surfaces either become adherent, or the valves of the heart become permanently diseased; and the ulterior effects of these lesions are dropsies, asthma, or affections of the lungs, which exhaust all the resources of our art, and ultimately they are to be reckoned among the most fatal maladies incident to humanity.

The pain in chronic rheumatism is often latent, unless the patient be moved, and then the agony is severe. In many cases it is absent during the day, but it is extremely acute during the night. This pain has a great tendency to shift from joint to joint, and is subsiding and again recurring. Redness is rarely present in chronic rheumatism; and, indeed, pain is the character by which it is commonly indicated; so that the term "pains" is not infrequently understood to mean "chronic rheumatism." In military practice much caution and circumspection is necessary in ascribing to "pains" alone their proper significance.

It is often impossible at first sight to state positively, from the appearance of a soldier, whether a man who complains of "pains"



is or is not suffering from chronic rheumatism, expressing itself by "pains" amongst the extensive white fibrous tissues which constitute the sheaths of the muscles, the covering of the bones, and the ligaments of the joints. The man may really suffer excruciating pains in the muscles of a limb, or in the fibrous tissues round a joint, and may be quite incapable of using his limbs in any way, or at all events of performing the active duties of a soldier; and yet there may be no appreciable heat or local swelling—no material disturbance of the system at the time—no altered state of the tongue or disturbed condition of the pulse, to indicate what he really suffers. Such cases must have the benefit of the doubt, with due caution to prevent "pains" becoming epidemic amongst the men. The military medical officer must therefore be prepared to meet with two classes of cases, namely,—(1.) Old soldiers who have previously suffered from rheumatism, or who are predisposed to the disease, and, it may be, "worn out" by previous disease and mode of life, and who suffer from chronic rheumatism, expressed by "pains" diffused over all the joints and general fibrous tissues of the body. The continued exercise of military duties induces so much fatigue that the digestive functions become impaired, and "pains" are induced. Such men must not be regarded as schemers. (2.) Another class of men present themselves with "pains," who have resolved, by all and every means in their power, to escape as much duty as possible, and endeavor to make the hospital a house of refuge. "Pains" are complained of in the knees, hips, shoulders, or loins; yet the men may be in good health. Such men, in the first instance, must also have the benefit of the doubt till, by care and patient investigation of all the phenomena of life in those men, the nature of the case is fully made out (Gordon, in *Indian Annals*, 1860). Records of temperature taken regularly and continuously will be found especially useful in coming to a conclusion regarding such cases.

The lesions of motion vary from mere stiffness to an entire binding down of the joint. In this manner the hip and shoulder may be so firmly fixed that the arm cannot be extended or the leg raised. The knee and elbow-joints are generally semi-fixed and cannot be straightened; while the fingers, if straightened, cannot be bent, or, if bent, cannot be straightened. When the joint is fixed, the muscles of the limb often become atrophied, sometimes partially so. Chronic rheumatism sometimes disappears in a few hours or in a few days, but it may last many weeks or months, or even years.

**Diagnosis.**—The only diseases with which acute rheumatism, when attended with swelling and redness, can be confounded, are erysipelas, gout, and *trichinous* disease (see page 843, vol. i). Chronic rheumatism is also often of difficult diagnosis when it attacks the intercostal spaces or diaphragm. It is apt to be confounded with pleuritic pains, or with other affections of the chest. It may also be confounded with many neuralgic affections, as well as with pleuritic diseases.

**Prognosis.**—The number of deaths from acute rheumatism hardly exceeds one out of every thousand deaths for all causes; whence it

is manifest that this disease is seldom fatal, although one most common, painful, and severe diseases of this country; and perhaps the number of unsuccessful cases hardly exceeds one per cent. But although this disease is rarely immediately fatal, a considerable number of persons ultimately die from diseases of the heart, apparently resulting from the action of the rheumatic poison.

Very few deaths occur from chronic rheumatism, so that the numbers that die bear but a very small proportion to those more or less constantly suffer. The constitutional state associated with rheumatism is one in which attacks of bad health culminate at last in more or less severe paroxysms, after which the patient for a time appears to have improved in health, and been better than his illness.

[Some interesting observations on the state of the urine in acute rheumatism, as bearing upon the prognosis of the disease, have been made by Dr. Thomas Stevenson (*Guy's Hospital Reports*, 1866). His observations are: (1.) When the excretion of solid materials in the urine is large, the patient makes, other things being equal, a rapid recovery; on the other hand, in lingering cases the excretion of solids is usually scanty. (2.) The urine being invariably scanty in bulk, but generally (from the cause only) of high density, a useful guide to the progress of the disease may probably be found by diluting the urine to the normal bulk, and ascertaining its specific gravity. According now as it is of high or low density will the progress of the disease probably be favorable or unfavorable. (3.) Though the excretion of urea is usually greater during the height of the disease than during convalescence, this is not invariably the case; the reverse sometimes occurs. Though the excretion of urea is greater during the disease than during the early stage of recovery, urea in the former stage seldom very much exceeds in amount the normal physiological excretion. (4.) The uric acid is always much increased while the disease continues. (5.) The phosphoric acid is generally in a greater amount during the progress of the disease than during recovery, but the quantity of this substance is rarely much in excess of the quantity secreted in health. (6.) The excretion of sulphuric acid is generally increased, and often largely, but it is variable.]

**Causes.**—If we look to the course and antecedent phenomena of acute rheumatism, it differs from all ordinary inflammations in the tendency it has to subside in one part and to appear in another—phenomena explicable by the laws of morbid poisons of constitutional origin, but which are opposed to all we know of the laws of ordinary inflammation. An attack of acute rheumatism is a series of febrile paroxysms, during which, while one joint gets well, another becomes affected; and this is especially likely to be the case in anæmic constitutions, or those which are enfeebled from any cause. Supposing this view of the case should ultimately prove correct, it will follow that cold and wet, by lowering the vitality of the system, greatly assist or promote the action of the poison, but are not great agents in the production of this disease. Any more extensive investigation into the remote or predisposing causes of rheumatism is extremely unsatisfactory. They are generally supposed to be identical with those causes which produce catarrh. Those, how-

who refer catarrh to the vicissitudes of temperature only, attribute rheumatism to this cause alone; but the returns of rheumatism occurring in the different commands of our army effectually blast such an hypothesis. It is not in the coldest climates that rheumatism is most prevalent, but at those seasons and in those climates remarkable for damp and variable weather; and "thus," says Sir A. Tulloch, "we find in the mild and equable climate of the Mediterranean or the Mauritius the proportion of rheumatic affections even greater than in the inclement regions of Nova Scotia and Canada;" and though some of the provinces of the Cape of Good Hope have occasionally been without rain for several years, yet rheumatism is more frequent in that command than in the West Indies, where the condition of the atmosphere is as remarkably the reverse. Exposure to wet, however, would appear to have much influence in causing that impairment of health which induces rheumatism, for we find the returns of the navy shew a considerably larger proportionate number of attacks than in the army; the number *per thousand annual mean strength* attacked in the Mediterranean fleet being 63.9; in the West India and North American station, 69; and in the South American station, 72.3. A predisposing cause of rheumatism lies not so much in the abstract degree of cold as in the range of atmospheric vicissitudes; and Dr. Haygarth has estimated that the number of persons attacked with rheumatism in summer is to those attacked in winter in the ratio of five to seven.

A very small number of children suffer from rheumatism. Out of 73 cases given by Chomel, 2 only were attacked under fifteen years—35 for the first time between fifteen and thirty—22 from thirty to forty—7 cases from forty-five to sixty—and 7 cases after sixty. At whatever age, however, rheumatism occurs, one attack, while it indicates the existing diathesis, yet does not establish a predisposition to another; and although many are always martyrs to the affection, yet a well-developed rheumatic paroxysm and a complete recovery tends to establish, under attention to diet and the prevention of exposure to cold and wet, a greater or less protection for a time; but anything which exercises a prolonged depressing influence upon the system, especially if there is at the same time an hereditary tendency to the disease, may induce the development of a severe rheumatic affection. The disease is distinctly hereditary. Dr. Fuller has traced the hereditary taint in nearly 29 per cent. of the cases admitted into St. George's Hospital. Men are believed to be more liable than women to rheumatism; but women, after menstruation has ceased, become more liable to rheumatism than males about the same age.

**Treatment.**—It seldom if ever happens that the cure of rheumatism can be safely intrusted to any single remedy; and the nature of the disease, as already indicated, points to various methods and combinations of treatment as most likely to lead to a successful issue.

*Venesection, calomel combined with purgatives, and opium,* are the three remedies which have been most generally made use of in the treatment of rheumatism; but many other remedies have also been

and are still advocated exclusively by not a few. These especially *vapor and hot-air baths, antimony, cinchona, colchicum, guaiac sulphur, nitre, lemon-juice, alkalies, and their salts.* *Veratrum* recently been added to the list.

But as the disease, when uncomplicated, may terminate so late in recovery, and may sometimes subside with marvellous rapidity under every variety of remedy, it is obvious that no sound inference can be drawn as to the success of any particular method of treatment, unless such treatment has been largely adopted in cases of similar severity—as estimated by the correlation of pulse, respiration, and temperature (measured by thermometer)—and attended with tolerably uniform results. Each and every proposed treatment which has therefore been hitherto proposed is regarded by the profession as unsatisfactory. (See account of a discussion upon a paper read by Mr. Dickinson to the Royal Med.-Chir. Soc. of London, and reported in the Medical papers of 21st of June, 1863.) If in one person's hands any particular remedial course has proved efficient, it has signally failed in those of another. If at one time a remedy has proved efficacious, it has been found inert or injurious at another, under different circumstances of age, sex, constitution, and the like. These facts ought not to appear strange to those who consider the true nature of the disorder, and the variety of circumstances under which the physician may be called upon to minister to his patient's relief. Bleeding, which in the young, plethoric, and robust, may be necessary to allay excessive vascular action and cause free secretion, may in the weakly induce irritability of heart and a consequent attack of cardiac inflammation. Opium, which in one person may prove of the greatest service in promoting free perspiration and in allaying the general irritability of the system, may in another check the biliary, "lock up" other secretions, and thus prevent the elimination of effete or deleterious matters from the system. The continued use of calomel, and constant purging by its means, which may be beneficial to one patient, by moving large quantities of unhealthy secretions, may unnecessarily exhaust the strength of another, and tend very greatly to impede recovery. And so in regard to every remedy which has been proposed: what is useful at one time may prove useless, or positively injurious, at another.

[Of 12 cases of acute rheumatism, without cardiac complications, treated by Dr. Austin Flint, at the Bellevue Hospital, New York, in which "palliative measures solely were employed," the mean duration of the disease was 26 days—the maximum being 56, and the minimum 12 days.\* Of 25 unselected cases, treated without curative drugs (except a little opium when the pain was very severe, and a purgative when the bowels were costive), by Dr. T. K. Chambers, at St. Mary's Hospital, London, the mean stay in hospital was 27.7 days.†

In the years 1863, '64, and '65, 41 cases of acute rheumatism

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\* [A Contribution towards the Natural History of Articular Rheumatism. Jour. of Med. Sciences, 1863.]

† [Lectures: Chiefly Clinical. Am. Ed., p. 176.]

treated by Drs. W. W. Gull, and G. Owen Rees, pretty much on the expectant plan. In Dr. Gull's thirty-seven cases, in several an occasional dose of Dover's powders, or half a grain of opium, and two or three ounces of brandy a day, were given, but the main treatment was Julep. menthæ c. taraxaci. One of the main objects he had in view was to endeavor to gain a more accurate knowledge of the natural course of the disease. No selection was made in the cases; they were all well-marked. The four cases of Dr. Rees were treated with mint-water only. They were all under 30 years of age, except 4, whose ages were respectively, 33, 34, 42, and 43. 22 were males, and 19 females.

	Males.	Females.
The average number of days from the admission into hospital to the cessation of pain, skin cool and no relapse, .	18.1	15.5
The average number of days from the admission to complete convalescence, out of bed, and no relapse, . . .	16.6	21.4
The average number of days in hospital, . . . . .	27.6	26.8

The condition of the heart in these cases was as follows :

Evidence of pericarditis, . . . . .	in 6
A bruit mostly heard at the apex, . . . . .	17
Rhythm of the heart irregular, but no bruit, . . . . .	2
First sound prolonged, but no actual bruit, . . . . .	2
Suspected myocarditis, . . . . .	1
Not stated, . . . . .	2
No abnormal sounds, . . . . .	11
	<hr/> 41

The average duration of the disease in 7 cases, in which there was no evidence of the heart being involved, was 8.5 days; while in 6 cases, in which the heart was affected, it was 23.6 days. In the cases with little or no cardiac disease, the shortest duration of the acute symptoms was 6 days, the longest 16 days. Where the cardiac disease was great, the shortest duration of acute symptoms was 7 days, the longest 38 days. On the heart-complication, the effect of not using curative drugs could not be settled, as in nearly two-thirds of the cases the signs of cardiac disease were present on admission into hospital, but Dr. Gull's impression is that the cases so treated were not more liable. Dr. Rees remarks: "The great point here illustrated is, that when treating acute rheumatism we are dealing with a disease which seems to go on to cure quite unassisted, and that this happens even when it appears in its acutest form."\*

Dr. Garrod says: "For several years I have treated some of my rheumatic patients in hospital on a purely expectant plan, and although the cases have not yet been tabulated, some of the results have been so decided as not to be mistaken. I am quite certain that many cases of even severe rheumatic fever get rapidly well without the administration of drugs, and on simply colored or camphor-water the improvement is often so quick and satisfactory, that had not the nature of the treatment been known, great virtue would surely have been ascribed to it; on the other hand, in many cases the disease runs a lengthened course with many partial relapses; such tardiness is often found under other plans of treatment." (Reynold's *System of Medicine*, vol. i.)]

To learn how to adapt our present remedies to the exigencies of

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\* [Guy's Hospital Reports, 8d series, vol. xi, 1865.]



Each particular case is what is now most of all required. Rheumatism is manifestly a highly inflammatory disease, of peculiar constitutional origin, as has been fully illustrated. Blood drawn presents a more copious layer of "buff" than other diseases. The proportion of fibrine being greatly increased, we can hardly feel surprised that bleeding has been largely resorted to. But although bleeding has been extensively employed, the profession has hitherto been much divided as to the advantage derived from the practice. A careful analysis of conflicting evidence leads to the conclusion that general bloodletting is not recommended, and on pathological grounds it is not justifiable. No advantage is gained as to time; the loss of a large quantity of blood is worse than the disease, for it would be felt by most persons as to their lives; but temporary, if any, abatement of sufferings is obtained by its use; lastly, this mode of treatment appears to have caused in the practice of Bouillaud an unusually large number of pericarditis—a larger number, indeed, than has been witnessed in any other person in the profession. A predisposition to cardiac inflammation is doubtless engendered by copious and repeated bleeding: one-half the cases became so complicated after trepan bleeding (DICKINSON). It undoubtedly increases the irritation of the heart, and consequently favors the production of other complications. Repeated observation has led Dr. Fuller and others to believe that general bloodletting is only beneficial in first attacks occurring in young, robust, and otherwise healthy persons, particularly in those cases which are marked by unusual severity of symptoms, or are unaccompanied by free perspiration. It must at all times be cautiously employed, and carried to a small quantity—viz., from *eight to twelve* ounces, according to the age and strength of the patient, the object being to *favor the action of remedies, and to promote free secretion*, by its use, rather than to arrest or cut short the disease (LATHAM, FULLER). If the perspiration is free, and remedies are absorbed, bloodletting is not to be thought of. In mild cases it is unnecessary; in the debilitated and those of weakly constitutions, it is inadmissible; and in well-expressed rheumatic diathesis, or when the disease is discovered to be hereditary, it utterly fails in modifying the morbid action, is ill-borne by the system, and therefore should not be practised under the extremely urgent and specific conditions mentioned (WILLAN, FORDYCE, ALISON, TODD, WATSON, and DICKINSON).

If circumstances, therefore, ever warrant its employment, it ought necessarily to take the lead of all other measures, and be used in the young, plethoric, and robust, in whom secretion is insufficient, whose pulse is full and bounding, and whose skin is dry, and hot, and burning. A single bleeding is sufficient to relieve the excessive congestion, on which the want of secretion, the great measure depends, and which forms an obstacle to the action of those remedies on which we rely for effecting a cure. Purgation is also to be avoided. It is injurious for three reasons—*First*, Because it is not necessary to the cure of the patient.

like bleeding, tends greatly to reduce his strength and protract recovery. *Second*, Because, from the nature of the complaint, the patient is quite incapable of moving, and his sufferings are aggravated, his irritability is increased, and his heart's action accelerated, by the repeated shifting of his position, which is rendered necessary by the calls of nature. And, *Third*, Because it necessarily gives rise to more or less exposure, which must be prejudicial to a person bathed in perspiration (FULLER).

The aim of purgation in rheumatism should be to obtain merely a single free evacuation every morning, after having obtained one full dejection by the following combination :

**R.** Calomelanos, gr. v; Pulv. Jalap Co., ℥ij-℥j; Pulv. Zingiberis, gr. iij-gr. v; *misc.* This powder, taken in a little milk, will secure an abundant secretion from the glandular follicles of the intestinal mucous membrane.

The daily evacuation of the bowels is then to be maintained by mild saline purgatives, such as the *potassio-tartrate of soda*, *sulphate and carbonate of magnesia*, taken as a draught in the morning ; and preceded every second night by a Plummer's pill at bedtime, combined or not, according to circumstances, with a full dose of opium ; or a grain of the watery extract of Barbadoes aloes may be found sufficient on alternate days. *Opium* may be given with the greatest advantage in the early and most painful stage of the disease occurring in adults, to the extent of *six or eight grains* of the powder in *twenty-four hours*, or two grains of the extract of opium may be given every night : to children, *half-grain* doses of the powder, or less, according to age, may be given every three or four hours. The bowels require to be kept open during its use ; and *its influence* requires to be *closely watched*. If the urine increases in quantity, if the motions become more healthy in appearance, and the coated tongue cleaner and less red, the influence of the opium is beneficial. It encourages sweating.

*Colchicum* administered alone is a dangerous agent ; but its virtue as a remedy may be obtained in small doses in combination with other medicines. It may be given with small doses of *ippecacuanha*, *alkalies*, and *opium*. It promotes evacuation by the kidneys, and appears to exercise some unknown influence over the process of assimilation, whereby the formation of the rheumatic poison is checked. It is only in some cases, however, that it appears to be of service. It is far less efficacious in the weak and nervous than in the more robust and less easily depressed, and of less value in purely *fibrous rheumatism* than in cases where the *synovial inflammation* predominates ; and in the hands of Dr. Fuller it has proved less advantageous in proportion as the fever has exceeded the articular swelling, and as the urine has been less highly charged with lithates. The operation of this remedy must under all circumstances be most carefully watched ; and a daily evacuation from the bowels must be secured during its use. If the lithates disappear from the urine, if the pulse becomes weak, if faintness, nausea,

or purging supervene, the *colchicum* must be at once discontinued; but till some one of these symptoms occur, a grain or a half of the *acetous extract* or the *inspissated juice*, or from ten to twenty minims of the *wine of colchicum*, may be safely to advantage administered two or three times a day.

*Veratria* is to be used in small doses. The ordinary form recommended is:

*Veratria* and extract of opium, one grain each, to be divided into pills, of which two pills are to be given the first day, three the second, four the third, five the fourth, and so on, increasing one pill until the condition of the pulse or the irritation of the mucous membrane compels a diminution.

The beneficial effects of *guaiacum* are obtained in those cases in which are unaccompanied by perspiration; and in which the torpid organs are gently excited by the action of the remedy. When the patient is perspiring freely, and when it neither promotes nor causes diuresis, very little benefit is obtained from it. Combined with *bitartrate of potass*, *sulphur*, and *rhubarb*, it constitutes the chief ingredient in a celebrated empirical formula known to Army medical officers under the name of "*Cheltenham*," which has obtained a high reputation amongst old soldiers as a remedy for the "pains," or chronic rheumatism.

In some large hospitals, both civil and military, this remedy is in common use in the treatment of old chronic cases. Its administration is as follows:

R. Pulv. Guaiaci, ℥j; Pulv. Rhei, ℥ij; Bitartr. Potassæ, Sublim., āā ℥j; Pulveris Nucis Moschatæ, ℥ij; Mel., lbj; mi. Of this compound two large spoonfuls may be taken night and morning.

The general treatment originally recommended by Brodie (1764), and followed successfully by Macbride (1772), Ives, Fuller, Furnival, Garrod, and now by many other physicians, consists in the use of *alkalies* and the *neutral salts*, with *colchicum*, a little *opium* being sometimes added, with the occasional aid of *calomel*, used only as a purgative, to promote evacuation from the bowels, and to soothe the intestinal mucous membrane. Baths are never to be employed if the skin is acting freely; but if it is hot, dry, and burnt, the cutaneous action may be successfully stimulated by means of the *hot-air bath*.

If the bowels act once a day, a laxative dose of *calomel* and *opium* may now and again be prescribed, if necessary, with the view of modifying the character of the intestinal secretions. Dark-colored, offensive stools indicate the necessity for the dose, followed by a draught of *infusion of senna*, together with half an ounce of *potassio-tartrate of soda* and twenty minims of the *vinum colchicum*; these should be repeated every evening and morning till healthy evacuations are obtained—i. e., till the motions are light-colored, more bilious, and less offensive.

Mercury has been recommended with the view of warding



diac symptoms ; but when it is given so as to affect the constitution before the commencement of cardiac inflammation, it not only has no influence in preventing the disease, but, by the irritability and general depression which it occasions, appears to modify the course of the disease in a manner by no means conducive to recovery. It destroys the red blood-corpuscles, and adds to the anæmia. About one in four cases treated with mercury are liable to cardiac complication (DICKINSON).

*Alkalies*, or the neutral salts, may be given in combination with *colchicum*, full doses of *opium*, and a little *antimony*. They aid the disintegration, and increase the elimination of sulphuric acid, by augmenting the alkaline condition of the blood (PARKES). The *alkalies* ought to be given largely. The patients lose their pains under their influence, and proceed rapidly to convalescence. The pulse is generally tranquillized within *forty-eight* hours from the commencement of treatment, and if in twenty-four hours the pain is lulled and the local inflammation greatly subdued, the constitution is evidently coming under the influence of the remedy.

The form in which the remedy is to be given is that of a simple *saline* or *nitre draught*, to which from two to three drachms of the *potassio-tartrate of soda* may be added, with ten to fifteen minims of the *vinum colchici*, from fifteen to twenty minims of the *vinum antimonii*, and from ten to fifteen minims of the *tincture of opium*, or of Battley's sedative solution, to prevent the salt running off by the bowels. This draught is repeated for the first twelve or twenty-four hours, at intervals of three or four hours, according to the strength of the patient and the severity of the attack ; and if pain is excessive, a pill containing half a grain to a grain and a half of opium, or an equivalent dose of Dover's powder, may be given once or twice daily. In the use of these remedies, constipation and narcotism are to be avoided on the one hand, and diarrhœa to be guarded against on the other (FULLER). If the saline treatment is to be used alone, then a solution of *nitrate*, *acetate*, and *bicarbonate of potash* should be given in such doses that *ten* or *twelve* drachms of the two latter salts together are taken in the twenty-four hours. Half a drachm of the *acetate*, with a drachm or a drachm and a half of the *bicarbonate*, and *ten grains of nitre*, dissolved in an ounce and a half of water and sugar, or lemonade, and given every two hours, night and day, until the joint affection and pains have begun to yield in severity, are sufficient for this purpose. Or the following: *Nitrate of potash*, one drachm ; *acetate of potash*, three drachms ; *water*, eight ounces. Of this mixture one ounce for a dose ought to be repeated every two, three, or four hours, according to the urgency of the symptoms ; or from twenty to sixty grains of the *bicarbonate of soda*, or of *potash*, may be given every three or four hours in half a bottle of soda or seltzer water ; or in an effervescent *citrate of ammonia*, or *potash draught* (TANNER). This is the treatment which most of all seems efficient in warding off the cardiac complications ; and Dr. J. J. Furnival was the first (after Prout) to direct the attention of the profession to the use of alkaline remedies (*carbonas potassæ*) in the treatment of *Rheumatism*, and especially as

a preventive of the cardiac complication (*Lancet*, 1841, p. 100). The good effects of such treatment as a preventive of the cardiac affections have been since fully demonstrated by Fuller, G. Basham, Goodfellow, and others. To be effective, the remedy must be carried out with energy and perseverance, till the articular febrile disturbance is lessened, and till an alkaline condition of the urine is established as soon as possible. *Liquor potassæ* in ʒss. may also be given to the extent of ʒiij to ʒvj in twenty-four hours (PARKES). Brocklesby is the earliest authority for the use of doses of *nitre* in the treatment of acute rheumatism. He enjoined for a diluting drink, water gruel boiled smooth, in each quart of which he dissolved two drachms of *nitre*, with or without a little sugar. He often thus prevailed on soldiers to take *ten drachms* or more of *nitre* in *twenty-four hours*.

[Of 26 cases treated by Chambers with *nitre*, the mean stay in hospital was 40 days. Of 141 treated with scruple doses of bicarbonate of potash twice daily, the mean stay in hospital was 34.3 days. Of the 33 treated with less quantities of the potash, it was 40 days.]

The *alkaline treatment* of rheumatism as the basis of operative treatment seems by far the most rational; for it has now been abundantly demonstrated that in cases of acute rheumatism there is an absolute deficiency of the saline ingredients of the blood; and while there is also a liability to fibrinous exudation, the tendency of saline remedies is to suspend the separation of fibrine (STEVENS). The treatment which has saline remedies for its basis thus contributes to restore the balance of the saline ingredients in the blood, and controls the tendency to fibrinous deposition. The progress of inflammation being thus retarded, time is gained for other remedies to effect the diminution of the excess of fibrine present in the blood, as well as the destruction of the rheumatic element, whatever that may be. *One, two, or even three ounces of nitre* may be taken in the two or four hours, but it must be very freely diluted in lemonade or water gruel. The urine passed under its influence acquires a high specific gravity, 1.026, 1.040.

In the experience of Dr. Chambers the *nitre* treatment is not a preservative against cardiac complications as the treatment with *bicarbonate of potash*; but his observations are not conclusive, because those treated with the latter remedy were also enveloped in blankets and flannel, so that no linen was allowed to touch the patient—a most important curative management of rheumatic cases, which cannot be over-valued. “*Bedding in blankets reduces by a good third the risk of inflammation of the heart run by patients in rheumatic fever, diminishes the intensity of the inflammation when it occurs, and diminishes still further the danger of death by that or other lesion; and at the same time it does not protract the convalescence*” (Chambers’s *Lectures*, chiefly Clinical [Am. Ed. p. 178]).

[The patient’s bed is made in a peculiar fashion. A muslin sheet may be allowed; but if the patients have underclothing only of a prohibited sort, they are better naked. Sheets are removed, and the

is carefully wrapped up in blankets, the newest and fluffiest that can be got, which are so arranged as to shut off all accidental drafts from the head. The bed-clothes being put so, are kept so, and the attendants and patients are warned, that on no account are they to be thrown off. "It is impossible," says Dr. Chambers, "to make too much of the value of absolute rest, and an evenly high temperature to the skin in rheumatic fever. They are worth all the other means of relief put together. Since I have succeeded in getting our nurses to adopt them as a universal rule in every case of rheumatic fever, I have had hardly any patients to treat for inflammation of the heart" (*loc. cit.*, Am. Ed., p. 158).

In the chapter on Rheumatism in Reynold's *System of Medicine*, Dr. Garrod speaks thus of the *quino-alkaline* treatment, of which, he states, he has made very extensive use during the past ten years, and from which, he thinks, he has obtained more valuable results than from any other: "Sulphate of quinine is ordered to be rubbed up with a solution of bicarbonate of potash, to which a little mucilage and some aromatic, as tincture of cardamoms or spirit of chloroform, is added: each ounce-and-a-half dose contains five grains of the quinine and thirty grains of the potash salt, the quinine being reduced to the state of carbonate. To the adult, the above dose is given each four hours, and persevered in until the joint affection and febrile disturbance have completely abated. It neither increases the thirst nor the furred state of the tongue, and its influence upon the heart is to lower its pulsations, but not to weaken them, and hence, when endocarditis and pericarditis are present, its employment is not contraindicated" (p. 914). The treatment by large doses of cinchona was long ago strongly recommended by Fothergill, Morton, Haygarth, and others, but the bulk and nauseousness of the remedy caused it to fall into disfavor. It was a few years ago revived in France by Monneret and Briquet, and the sulphate of quinine substituted for the bark: as much as one and a half drachms were given by them in some instances. The action of the alkaloid in rheumatism is powerfully sedative and analgesic, but its good effects can be had by much smaller doses,—five to ten grains, two or three times a day.

*Lemon-juice* has been recommended by Drs. Perkins, Ciraud, and G. Owen Rees, the latter relying much upon it as assisting to diminish general excitement, and to lower the pulse. He believes the citric acid to undergo changes in the stomach, supplying oxygen to such elements as tend to produce uric acid, and favoring the formation of urea and carbonic acid. It is given in doses of from four to six ounces daily. The stomach often illy tolerates it, and it sometimes causes very depressing effects. An advocate of the transformation theory, Dr. James F. Duncan, of Dublin, recommends the *permanganate of potash*, in consideration of its chemical constitution, containing, as it does, oxygen in large quantity, and holding it in very loose affinity. The form in which he gives it, is one part of Condry's disinfecting fluid (9.26 grains of the salt to the fluid ounce of water), and seven parts of distilled water; one-half ounce of the mixture every second hour (*Med. Press and Circular*, vol. i, 1866).]

There is some difficulty in fixing exactly the date at which the disease may be said to have yielded, or to have disappeared. Cessation from pain has sometimes been taken as the sole indication. But a morbid state of the system in rheumatism may exist after the joints are free from active pain, and even without the presence of any cardiac affection, which requires but the smallest exciting cause to bring back the articular or heart affection in all its pristine

intensity. Patients in such a state, though free from pain time, cannot be considered rid of the disease. Dr. Garrod looks the following conditions, therefore, as indicating real freedom from the disease: (1.) Absence of pain and any acute tenderness of joints; (2.) Freedom from any active cardiac affection; (3.) Absence of marked thirst, with returning appetite; (4.) No rapidity or hardness of the pulse; and I would add, a normal temperature.

External topical applications are also efficient aids in treatment. Warm fomentations are always employed with advantage; in all applications a mixed alkaline and opiate solution, applied by fomentation, is the most powerful in allaying the pain of rheumatic inflammation. The solution so highly recommended by Dr. Lush for this purpose is composed of an ounce of the *carbonate of potash* dissolved in a pint of the decoction of poppies or of rose-water, to which six drachms of Battley's solution is added, or common tincture of opium, which is less expensive and equally efficient for external application. The efficiency of salines as a local application is probably due to their influence in correcting the disordered state of the blood at the part, by preventing or destroying the aggregation of the corpuscles, and consequently their tendency to separate from the fibrine and to accumulate in the minute vessels. On this principle the application was theoretically indicated many years ago by Mr. Gulliver as useful in inflammation. *Spongipiline* is admirably adapted for the purpose of applying locally saline remedies. Dr. Basham recommends its application in the following manner: "If the hands, elbows, knees, or feet are the seat of inflammation, gloves or caps are made of the *spongipiline* to cover these parts; if a greater extent of surface be involved, a portion of the *spongipiline* is cut large enough to envelope the entire surface. The spongy surface of this epithem is first moistened freely with water, and any superabundant fluid squeezed from it, so that the linen or bed of the patient may not be unnecessarily wetted. The *carbonate of potash*, or the salt to be employed, in powder, is then finely and plentifully sprinkled over the moistened surface, or rubbed in to secure its solution and the thorough impregnation of the surface; it is then applied to the inflamed part, and lightly secured by a roller. Nothing further is required than once in about twelve hours to re-moisten the spongy surface; fresh addition of nitrate is never required if a sufficient quantity has been used in the first instance. The salt in powder is hence better than a saturated solution, which nurses seldom succeed in making" (Basham, *Med. Trans.*, vol. xxxii, p. 10).

[Alkaline baths, in the acute, and particularly in the subacute or chronic form of the disorder are, according to the writer's experience, apparently of great service. Two pounds of the bicarbonate of potash and one pound of the nitrate of potash are dissolved in water of the temperature of 98° Fahr. The patient should not remain in the bath longer than ten minutes. Dr. Flint says: "Extension of the limbs by means of an apparatus for that purpose, or by simply a cord, pulley, and weight"

in order to relieve the pressure of the articulated surfaces, has been tried to some extent at Bellevue Hospital, and found to afford in some cases marked relief" (*Practice of Medicine*, p. 830).]

In contradistinction to the principle of cure now laid down, and which has been called the method of *neutralization*, Dr. Herbert Davies, physician to the London Hospital, has recently advocated a method by *elimination* as a safe and rapid mode of combating rheumatic fever. For this purpose his treatment is *absolutely* and *entirely local*, and consists of *free blistering* only. Believing [the morbid matter is not in the blood, but] that the *virus* localizes itself for a time in the inflamed joints, and that the intensity of the local inflammation is a measure of the amount of *virus* collected, he orders blisters, varying in width, but of considerable size, according to the locality, to be applied round each limb, and in close proximity to the parts inflamed. Thus he affords, through the serous discharge from the blistered surface, a ready means of exit for the *virus*. The blisters are to be applied *near to, but not upon every joint* inflamed, at the very height of the inflammatory stage, even when the local pains are the most severe and the constitutional disturbance the greatest.

The success of the plan is said to depend entirely upon the blisters being applied and allowed to remain until they have thoroughly acted, [from six to eight hours.] Linseed-meal poultices, [or cotton-battings,] subsequently applied will be found highly serviceable in promoting a sufficient flow of serum. The blisters should be put entirely round the affected limb, and when the knees are the joints which suffer, the blisters should be cut at least three inches wide.

If this method of treatment be adopted, no medicine ought to be given beyond an occasional purge, and no advantage results from combining the *alkaline* or neutralization system of treatment with that by *blisters*, or the method by local elimination. If the two methods are combined, the period of convalescence is protracted. That the poison is really thrown out by the blister method of treatment is deduced as well from the rapid and permanent relief resulting from the local treatment, as from *the neutral and even alkaline condition of the urine*, which is a usual and early result of the treatment, as well as a rapid diminution in the force and frequency of the pulse (*London Hospital Clinical Reports*).

[The treatment by free blistering the affected joints in the acute stage of rheumatism was, it is believed, first practised by Dr. Dechilly, of Vaucouleurs, France. In 14 cases, treated in this way, recorded in his memoir presented to the Academy of Medicine in 1850, the cure took place, in several of them, in from five to six days, and in all the febrile symptoms were lessened, and the pain and swelling of the joints diminished in from twenty-four to forty-eight hours. Dr. Dechilly did not regard the plan as antiphlogistic or local, but as acting on the rheumatic element, "the morbid cause which exists in the economy, and of which the articular phlegmasiæ are but the symptomatic expression" (*Bul. de l'Académie de Méd.*, t. xv, 1850). Dr. Martin-Solon made a favorable



report upon the communication, in which he related three cases treated by Dr. Dechilly's method with like results. After a debate by the Academy upon the nature and treatment of rheumatism generally, the report was not adopted, the number of records being in its opinion insufficient to warrant any conclusion as to the efficiency of the proposed treatment, and the author of the memoir recommended to continue his observations. The treatment attracted little attention, or had not at least any advocates until recently revived by Herbert Davies. Dr. Dechilly enveloped the whole joint in a large blister, and followed the local troubles when they cropped out in instalments. Dr. Davies puts the blisters in bands near the affected joints. Dr. Davies claims that the tendency to cardiac disease is greatly lessened by blistering treatment. Of 50 cases treated at the London Hospital, 23 had already hearts damaged by recent or old mischief, and 23 were free from heart-disease. The result of the blister treatment in these 50 showed that 25, when discharged from hospital, were totally free from any endo- or peri-cardiac disorder; every heart was sound which was previously sound, and 2 recent cases of endocarditis were apparently cured. Davies states that those cases answered best to the treatment when a large number of joints are simultaneously affected. Dr. Jeffreason, of St. Bartholomew's Hospital, and Dr. Greenalgh, of the Middlesex Hospital, report favorably upon the plan of treatment, the former stating that in those who came into the hospital with the heart unaffected, no cardiac disorder was subsequently developed. It has been tried also by Dr. Lasègue, in the Necker Hospital at Paris, in three classes of cases: (1) recent acute rheumatism; (2) recent subacute rheumatism, with a febrile action; (3) recurring rheumatism, with imperfect remissions and sharp febrile phenomena during the paroxysms; and in all with like results (*Archives Gén. de Méd.*, Nov., 1865).]

The diet of the patient, in acute rheumatism, should be strictly limited to slops, such as arrowroot, beef tea (Liebig's *Extra Flesh*), milk and lime-water [or milk and Vichy water], or made warm, to which a little *carbonate of soda*, *nitrate of potash*, or *bicarbonate of potash* is added, light puddings, to which sherry or brandy may be added, if depression exist; and even in many cases it is desirable to limit the diet to puddings and white fish.

To obviate the great drain upon the system, it is necessary, however, that the rheumatic patient be better fed than in cases of febrile inflammation. Beef tea and jellies may be given, and stout coffee might also be administered frequently, on the same principle that it has been given in typhus fever by Dr. Parkes, following the indications obtained from the physiological action of that beverage as expounded by Lehmann. (See p. 410, vol. i, and p. 39, vol. ii.)

The nature and treatment of the local complication, such as *pericarditis*, will be considered subsequently; but in addition to the general line remedies, which have an undoubted influence in shortening the duration of the illness, the bedding in blankets, referred to at p. 61, is a most important measure for preventing the cardiac affection.

In the forms of muscular rheumatism the local appliances which secure rest and warmth are those which afford most relief. In lumbago nothing is so instantaneously beneficial as strapping

back from the level of the "seat" upwards, in imbricated layers, by straps of adhesive sticking-plaster, or warm-plaster (TURNELL).

## [GONORRHOÆAL RHEUMATISM.]

(DR. CLYMER.)

This affection was first mentioned by Sir Astley Cooper; its symptoms and nature were described by Sir Benjamin Brodie in his work on *Diseases of the Joints*; and it has recently been treated of by Bardwell, T. K. Chambers, Brodhurst, Fournier, Pidour, &c. It is an acute or subacute inflammation of the joints, happening in the course of a gonorrhœa, or, sometimes, simple urethritis, having in its external aspect a resemblance sometimes to one, and sometimes to another, form of rheumatism. Though it may occur in all stages of gonorrhœa, it generally appears after the local disease has lasted some time.

**Symptoms.**—From ten days to three weeks after the gonorrhœa is established, one or more joints become stiff, painful, and swollen, generally after the sufferer has been exposed to cold or damp. A good deal of constitutional disturbance commonly accompanies the articular troubles; the skin is hot and dry, the tongue furred, and there is more or less fever. Usually there is no change in the amount or nature of the urethral discharge, though sometimes it is slightly lessened. The fever and local inflammation are, generally, more severe in the robust and plethoric than in the weak and anæmic. "In the young and plethoric the inflammation is of an acute character, and lymph is for the most part deposited on the synovial membranes, giving rise to false ankylosis; whereas, in the debilitated, serum alone will be effused. In both cases the joints are liable to be destroyed; in the former through the deposit of lymph and the production of false ankylosis, and in the latter through the destruction of the cartilage. The joint having become inflamed, a large effusion of serum takes place in the synovial cavity; but although there may be great tension, suppuration never occurs. Absorption of the effused serum takes place, and the joint may resume its healthy action. The limb remains in a semi-flexed position during the period of effusion into the joint; for in the flexed position of the limb, the surrounding structures are somewhat relaxed, and consequently they yield to the bulging membrane with its contained fluid. When, however, the hip is inflamed, the limb remains much more extended than in ordinary hip-joint disease." (BRODHURST, *Reynold's System of Medicine*, vol. i, p. 921.) The knee is more frequently affected than any other joint. One joint after another is very apt to be involved. The synovial membrane is sometimes left thickened, and the movements of the joint permanently interfered with. In some cases the cartilages become diseased, and the joint is permanently rigid. Although the effusion into a joint may be very considerable, dislocation never occurs, and in this respect the disease differs from ordinary rheumatism, where the tendency is for the articular surfaces to become more or less displaced; but in this disease ankylosis, rather than dislocation, is a result. (BRODHURST.)

The disorder may last for weeks, or for months, or even years. The first attack may leave no trace behind, though slight stiffness and a crackling sensation may remain for several weeks; but subsequent ones are apt to cripple the patient for life. There is a remarkable disposition to relapse, and each attack is more severe than the preceding one; and though the first attack is nearly always the result of a specific urethral disorder,

subsequent ones happen from any urethral discharge, or irritation case mentioned by Brodie, where there were four attacks, he says inflammation of the urethra in all of them was the first symptom; of the attacks the discharge from the urethra was attributed to the existence of gonorrhœa, and in the two others to the use of the bougie.

The female seldom suffers from the disease; but Brodhurst says, wherever he has observed an instance, it has always terminated in suppuration.

Sir Astley Cooper and Sir Benjamin Brodie both pointed out that the form of ophthalmia may precede or accompany the joint affection. The conjunctiva, sclerotic, and iris, may all be affected. This form of ophthalmia is not usually severe, and yields readily to treatment.

**Nature.**—Sir Benjamin Brodie says: "The disease is generally described under the name of Gonorrhœal Rheumatism, though it is distinguished from the course of its symptoms and from the effect of remedies, differs from ordinary rheumatism in many respects" (*Pathological Observations on the Diseases of the Joints*). Mr. Barwell regards it as a slower form than ordinary of purulent infection, produced by inflammation of the prostatic veins," but admits that he has "no cases of it to prove this position." (*On Diseases of the Joints*.) Dr. Chambers observes: "Gonorrhœal rheumatism is a convenient conventional term, which we continue to employ, really for want of a better. But it has not the slightest other relation, either pathological or therapeutical, besides external similarity, to rheumatism. It is really due to poison absorbed into the blood from a mucous membrane affected by purulent gonorrhœa, and thus has more claim to be classed by the name of pyæmia, than in the position it is now placed. Like pyæmia, it has a strong tendency to disorganization of the affected part; pus and fibrine are formed, and the tissues are destroyed in bad cases. Like pyæmia, too, it does not exhaust itself by the inflammations which it produces; it is not an acute disease, in the sense of tending to a recovery, but a chronic disease, getting worse and worse if not arrested. It is an attendant of gonorrhœa, not an essential part of it, just as pyæmia is an attendant of surgical operations or wounds. . . . I suspect the cause of the disease to be a virus especially fatal to the vital functions of the non-vascular tissues, which is carried to them from the urethra by the blood. The partial loss of vitality in these white tissues causes congestion and inflammation in the neighboring capillaries, with pains and extra-vascular accumulations of serum.") CHAMBERS, *loc. cit.*, pp. 2-9.) The pyæmic theory of the pathogeny of gonorrhœal rheumatism is supported by the fact of its occasional occurrence during simple gonorrhœa, produced by catheterism or other causes.

**Treatment.**—Moderate bloodletting, general and local, if the patient is robust, in a first attack, with purgatives, opium, and blisters, and fomentations to the affected joints, is the treatment recommended by those who claim to be successful in its management. "The reason for its adoption is experience of its good effects, the little effect which other treatment has, and the certainty that the tendency of the disease is to get worse and worse, if let alone," says Chambers. According to Mr. Brodhurst, "when gonorrhœal rheumatism is treated in the beginning of the attack with vigor, the joints may become affected in a slight degree only. Many of the sufferers, however, with this disorder are debilitated and anæmic from the life they have led, and active measures are inadmissible, and it too often proves intractable. Blisters to, and around, the joints, and the continued administration of the iodide of potassium and ammonia,



or the muriate of ammonia, with an occasional opiate, and the use of the Turkish bath, when not contraindicated by existing organic disease, will prove the safest and surest treatment in a large majority of cases. Mr. Brodhurst says, that he has "known the pain to cease entirely in the bath. Some time since I saw a gentleman who suffered very acutely from pain and inflammation consequent on this form of disease of the joints; the tension from the effusion was also excessive. He was lodged in a house attached to a Turkish bath, and each day was carried down into the bath. When profuse perspiration was obtained, the pain left him, and absorption of the fluid, within the synovial capsules, was certainly promoted by submitting the limbs to the high temperature of the bath." The writer's experience agrees with that of Mr. Brodhurst, provided the rules laid down for the use of the bath, vol. i, p. 736, are observed. In the chronic form, iodide of potassium may be largely used. Though in an acute attack abstinence from flesh-meat, as well as from fermented and distilled liquors, may be necessary, the rigid diet recommended by many is harmful. Dr. Chambers remarks on this point: "As to the starving, I do not know what to say—perhaps it does good by promoting absorption—perhaps it is not so requisite as we suppose." The patient should be well nourished, and quinine, iron, and arsenic are often required. On the subsidence of the acute articular symptoms, gentle friction of the joints must be used several times a day, and subsequently passive motion; sometimes it may be necessary to administer an anæsthetic, and resort to some force to overcome rigidity. By these means mobility may be entirely restored.]

### ACUTE GOUT.

**LATIN EQ.,** *Podagra acuta*; **FRENCH EQ.,** *Goutte aiguë*; **GERMAN EQ.,** *Acute Gicht*; **ITALIAN EQ.,** *Gotta o Podagra acuta*.

**Definition.**—*Febrile excitement of a specific kind, attended with a specific form of non-suppurative inflammation, with considerable redness of certain joints, chiefly of the hands and feet, favored by congenital or hereditary constitution, associated with disorder of the digestive and other internal organs, characterized especially by an affection of the joints, and especially on the first joint of the great toe—by nocturnal exacerbations and morning remissions—by vascular plethora and the presence of lithic acid, and probably also of phosphoric acid in the blood. The constitutional affection tends to culminate in a paroxysm, or "fit of the gout," at longer or shorter intervals, when various joints, textures, or parts of the body, are apt to become affected.*

**Pathology.**—The constitutional origin of gout may be explained in a similar manner to that of rheumatism; but the nature of the *gouty poison* appears to have been more definitely ascertained. About 1787–1793, Mr. Murray Forbes, remarking the close connection between gout and gravel, and the tendency of the disease to form concretions, ascribed gout to the presence of *Lithisiac*, or what has since been named *uric* or *lithic acid* in the blood. These concretions are liable to periodical deposition, through the medium of inflammation, in organs whose vessels are of the smallest order—for example, tendons and ligaments (*Treatise on Gravel and Gout*,

pp. 78–80). About the same time, Berthollet had advanced opinion not dissimilar with regard to the presence of *phosphoric acid*. From numerous observations this chemist concluded *phosphoric acid* is more sparing in the urine of the gouty and rheumatic than in that of healthy persons, and that during an oxysm it became more abundant, and equalled the proportion in the urine of the most healthy persons. He therefore ascribed gout to the retention and accumulation of this acid, and its excretion through the system (*Journal de Médecine*, Juin, 1786, p. 178). The reverse of this doctrine was afterwards maintained by Brand, who observes that, on the approach of gouty fits, the phosphoric ingredients of the urine diminish, and seem to be converted to the joints, to produce the arthritic concretions (CRAIGIE).

In 1848, Dr. Garrod, of London, published a paper in *The Chir. Transactions* (and he has since that time constantly directed his attention to the subject), in which he proves experimentally the statements of Forbes, and shows that the blood, in cases of gout, contains *lithic acid* in the form of *lithate of soda*, and that in cases of chronic gout with chalky deposits round the joints, *lithic acid* is always present in the blood and deficient in the urine, both absolutely and relatively to the other organic matters. He believes that the *acid* is always in excess in the system during gout, and constitutes a very important and almost a pathognomonic sign of the gouty disease with affections of the joints. The uric acid is found to exist not only in the blood-serum, but also in the fluid effused by blisters, and in the abdominal and pericardial fluids. The separation of a very small amount of blood is required to detect the uric acid if it is present in quantity. From one to ten fluid drachms of the serum being taken, it is to be put into a broad flat glass dish (not watch-glasses), about three inches in diameter and about a third of an inch deep. Add acetic acid, of the strength of the London pharmacopœia, in the proportion of about six minims to each fluid drachm of the serum; a few bubbles of gas are generally evolved at first. When the fluids are well mixed, a very fine thread is introduced, consisting of from one to three ultimate fibres, from a piece of unwashed linen fabric, about an inch in length, which should be depressed by means of a glass rod. After resting for from eighteen to forty-eight hours, depending on the warmth (temperature at or below 70°) and dryness of the atmosphere, the uric acid will crystallize upon the thread. To observe this, a microscope must be used with a linear magnifying power not below 60.

When gout has become fully developed, and has assumed a specific inflammatory character, it produces all the forms of articular inflammation which have been described in rheumatism, and these inflammations attack nearly the same parts, as the bones, cartilages, synovial membranes, bursæ, ligaments, muscles, tendons and aponeuroses. These inflammations have little to distinguish them from rheumatism, except the singular pathological phenomenon of a tendency to the deposition of the urate of soda—a discovery we owe to the late Dr. Wollaston.

Occasionally the urate of soda appears to be nearly the sole secretion from the affected part, nothing being seen on the poultice or fomentation-cloth applied to the part but this salt, in a more or less fluid state. It is secreted from the joints of the toes or fingers, and probably from all their different tissues. Portal gives a case in which the articulations of both hands presented deposits of urate of soda, both within the capsules of the joints and externally among the ligaments, while the tendons of the extensor muscles of the fingers were almost destroyed. In the Hunterian Museum of Glasgow there is a finger from a gouty hand, with a joint opened and bent upon itself, showing not only a deposition of the salt, but an erosion of the cartilages; also another, in which the joint is full of this peculiar secretion; and a third, in which the joint is everywhere invested with it. In the Museum of St. Thomas's Hospital there is a specimen in which the femoral cartilage of the knee-joint is coated with it, as if smeared over with plaster of Paris; and another in which it is deposited on the ligaments of the extensors of the hand. Guibert gives a case in which the metatarsal articulation of the great toe was surrounded by urate of soda of a rose tint, and on the inside of the foot, in the cellular tissue, was an abscess containing urate of soda, making its way to the surface. On opening the joint the same substance was found, and, on cutting through the tendons, pieces of urate of soda were distinctly seen between the fibres. Simon gives an account of a gouty skeleton, of which the bones were completely ankylosed. In the preparations of diseased joints in the Museum of the Army Medical School at Netley (and described by Mr. Gulliver, in *The Edin. Med. and Surgical Journal*, vol. xlviii), the material is said to be deposited outside the joint in the cellular tissue, exterior to the periosteum and articulation-capsules. Similar dissections are described by Watson, Moore, and Parry. The bones affected in cases of gout have been found swollen, and sometimes so soft as to have been easily cut by the scalpel.

The urate of soda is deposited first in a white fluid state, like a mixture of chalk and water; after a time this fluid portion becomes absorbed, leaving concretions, consisting of little more than bundles of crystals of urate of soda, and often in such quantities that a poultice, though applied several times a day, has been covered with them, and that for several days together. The concretions afterwards harden, and form what, from their color and appearance, have been termed *chalk-stones*, *tophi*, or *tophaceous* deposits, often superficial and of considerable size, so that, when the skin has ulcerated, a patient has been said, in one instance, to have scored his game of cribbage with his knuckle, and in another to have written on the table with the *chalk* penetrating through the ulcerated tips of his fingers. Not unfrequently, after a time, deposits of *phosphate* and *carbonate of lime* ensue in and around these semifluid concretions; but it is not believed that such deposits ever replace the *urate of soda* in gouty subjects.

Although these specific exudations and morbid elements in the blood are but indications of the gouty condition, they nevertheless

confirm the belief, again gaining ground, which teaches the *humoral doctrine*, that the phenomena of gout are induced by peccant matter (probably uric acid) which, through mal-assimilation of food, or of food and drinks of particular kinds, combined sometimes with excessive labor of body as well as of mind, becomes bred in the constitution, and which it is the business of the physician of gout to eliminate.

[The views of Dr. James F. Duncan, of Dublin, upon the pathology of gout,\* leading to new principles of treatment, are worthy of consideration. Assuming, as a matter of fact, that the blood in gout is charged with acid to an extent far beyond the natural standard in health, he attempts to show that its production depends upon imperfect oxidation of the blood, and more or less impaired nervous energy as the cause of that imperfect oxidation. Dr. Garrod looking upon the kidney as an organ of excretion alone, he considers the urea and uric acid already in the blood in the condition in which they come to be eliminated, and only require to be separated from it by a sort of elective affinity. He looks upon these two products as having no necessary relation to each other, and that the power of excretion of the one may be maintained in full energy, while that of excreting the other may be diminished or arrested. In gout, he regards the excretion of uric acid as impaired, just as the secretion of urea is impaired in Bright's disease, the consequence being, if its formation in the blood continues to take place, it must accumulate there, and, acting as poison, gives rise to those symptoms which characterize a paroxysm of gout. "Gout," writes Dr. Garrod, "would thus appear partly to depend on loss of power (temporary or permanent) of the acid-excreting function of the kidneys; premonitory symptoms, and those also which constitute the paroxysm arising from an excess of this acid in the blood, and from the effort to expel the *materies morbi* from the system. Any undue formation of uric acid compound would favor the occurrence of the disease, and hence the connection between gout and uric acid, gravel, and calculi; and also the influence of high living, wine, porter, and want of exercise, etc., in inducing it" (*The Nature and Treatment of Gout*, 2d ed., p. 339). Before mentioning Dr. Duncan's objection to this theory it may be well to state here, that the recent experiments of Oppler, Perls, and Zalesky have shown that both urea and uric acid are actually produced in the kidney, and that when found in the blood it is due to reabsorption (see *Chronic Bright's Disease*, in this volume). It is really then the primary product of tissue metamorphosis (creatine, creatinine, &c.), which accumulates in the blood, and, in a later stage of histolysis, undergo transformation into urea and uric acid. Dr. Duncan argues that there are naturally two sources of uric acid in the system; one, which may be considered almost constant in its amount, arising from the metamorphosis of tissue undergoing disintegration, and another, variable in quantity, arising from a greater or less amount of nitrogenized food consumed by each individual. Now, gouty patients are met with chiefly among that class of persons who feed well, and consume animal food. If much exercise is taken in the open air, or some laborious occupation followed, there may be an adjustment between the functions and no harm follow. But if the per-

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\* [Dublin *Quarterly Journal of Medical Science*, May, 1865. London *Medical Press and Circular*, May, 1866.]

be of sedentary habits, little or no exercise taken in the open air, and the function of respiration only imperfectly kept up, it is plain that the oxidation in the lung being insufficient to convert all the uric acid which is formed into urea, an accumulation must take place, and prove a source of disease. Another cause of the retention of uric acid in the system is to be found in the imperfect manner the function of respiration is performed, owing to the disturbed state of the digestive system. The pneumogastric nerve being interested in the healthy performance of both functions, it is not unreasonable to suppose that whatever tends to concentrate the energy of this nerve in one of the two functions, must interfere with the discharge of its functions in the other. If the stomach is too much taxed, and the process of digestion hindered and protracted, the function of respiration must suffer. Now the function of digestion is much impaired in every case of gout. Sometimes this disturbance seems to be merely the result of lessened nervous energy, and persons of abstemious habits may be martyrs to a disease which is commonly caused by indulging in the pleasures of the table, but which in their cases owns no such origin. But most frequently the fault is not in the organs which suffer, but in their bad usage, by more work being thrown on them, in the quantity and quality of the food taken, than they are capable of performing. If proof of this position were needed, we have it in the foul breath, the loaded tongue, the nausea and sense of weight in the stomach, and the flatulence that most gouty patients are subject to, and still more in the common interval observed between the time when the error of diet was committed and the development of the actual paroxysm. A disordered digestion is then the *primum mobile* of the whole train of morbid phenomena. This leads to imperfect performance of the function of respiration, this, again, to imperfect oxidation of the protein compounds in the blood, and the consequent accumulation of uric acid in the blood. This accumulation again leads to the special symptoms of the gouty paroxysm, general disturbance of the entire system, diminution or suspension of the function of the kidney, and further augmentation of the *materies morbi* in the blood by non-elimination.

"The gouty diathesis," says Dr. Bence Jones, "consists in an excess of urate of soda not only in the serum of the blood, but in the fluid that diffuses from it into all the vascular and non-vascular textures of the body. An attack of gout is a chemical process of oxidation set up in the parts where the urates are most able or liable to accumulate. By the oxidizing action the urates are wholly or partly changed into urea and carbonates, which can more readily pass from the textures into the blood, and be excreted by the kidneys, skin, and lungs. The oxidation even in the bloodless textures causes increased flow of blood and mechanical pressure in the vessels nearest to the inflamed part, and hence pain and redness, and then swelling and œdema, proceed. Though the gouty diathesis is a disease of the textures as well as of the blood, yet in its origin and situation, an attack of gout is even more a disease of the tissues than a disease of the blood. The urate of soda bears the same relation to gout that sugar does to diabetes; and as the want of oxidation of sugar is the cause of the diabetic diathesis, so the want of oxidation of the urates, and their consequent accumulation in the textures and blood, is the cause of the gouty diathesis. There are at least two ways in which an excess of uric acid may occur in the blood and textures: 1st, from an excess of animal or vegetable albuminous food entering the system,—i.e., from excessive production; and 2d, from an arrest of oxidation,—i.e., from want of destruction." (*Lectures on Some of the Applications of Chemistry and Mechanics to Pathology and Therapeutics.*) ]



The natural history of the disease shows—(1.) That there is a disposition or tendency in the morphological relations between solids and fluids of the whole system to develop some specific constitutional poison, which betrays itself by certain constant effects at periodical intervals, although these may be irregular. (2.) That these local effects are prone to develop themselves in the joints of the extremities, or to express themselves by symptoms of a peculiar kind in the internal organs, and in various textures of the body.

Dr. Graves has given the most interesting illustration of these propositions in his clinical lectures; and especially of the development of peculiar local affections in connection with gout. For example, the occurrence of—(1.) “Fugitive pains,” or twitches which suddenly attack persons of gouty habit. The pain is, no doubt, due to congestion. Sometimes the congestion is more lasting, as in the lobes and cartilages of the ear, and where, as Dr. Graves shows, concretions sometimes occur. (2.) A singular affection of the teeth, which consists in an insuperable desire to grind them, first noticed by Graves. (3.) The occurrence of *tic-douloureux* of the terminal branches of the fifth pair. (4.) Daily paroxysms of intense heat of the nose, which continues for three or four hours, the nose becoming first of a bright and then of a purplish red color, spreading over the upper portion of the cheek. (5.) The occurrence of *gouty sciatica*, in which the specific inflammation affects the trunk of the sciatic nerve, and which, extending by the neurilemma to the nerve, may in process of time extend to the spinal marrow and its investments, and give rise to derangements of the latter, terminating in ramollissement and structural degeneration. This complication of gout was first decidedly pointed out by Dr. Graves. These affections are always greatest when the stomach is much deranged.

It is now generally believed that gout is hereditary; and in many instances it is so, whether the intemperate habits of ancestors have been followed out, or whether the mode of living be abstemious. In some families it attacks only alternate generations, following what has been called “the law of *atavism*.” The power of hereditary transmission to induce gout has been illustrated in a most interesting way by Mr. Spencer Wells in his treatise on this disease. He shows that the children of a patient born prior to the development of gout in their parents were free from this affection in after-life, but that those children born subsequently to the development of gout in the same parents became afterwards gouty. The powerful influence of hereditary predisposition in regard to gout is well known to betray itself even amongst some of the laboring classes, and in the upper classes in a still greater proportion. Dr. Graves gives instances the case of a gouty gentleman who is able to trace the disease in his family for four hundred years, and in which his eldest son has invariably inherited, *with the estates*, gout of the worst form and developed at an early age. On the other hand, again, it is certain, and consistent with the constitutional origin of this disease, that an unusually large proportion of non-heredit-

cases are met with among the indolent and luxurious inhabitants of large metropolitan towns.

A flattering hallucination has been transmitted traditionally, hereditarily, and historically, which connects the gouty diathesis with high mental development. But the conditions for the constitutional development of this disease must either be more common, or physicians must be getting more acute, for eminent and observant men are now inclined to discard the doctrine which teaches the noble origin of gout, and its necessary association with high mental development. The disease is now certainly common and plebeian, as well as aristocratic. It may have been, in the days of Sydenham, that the gouty patients of a physician were to be found amongst "*magni reges, dynastæ exercituum, classiumque duces, philosophi, aliique his similes.*" Nowadays it is no less certain that the physician, in London at least, must pay his visits and prescribe for gout amongst "the London labor," as well as among the "London poor," and his list will number "coal-heavers, bakers, brewers, draymen, house painters, butchers, innkeepers, publicans, butlers, coachmen, and porters in wealthy families especially" (COPLAND, BUDD, GARROD, TODD). A gouty conformation has been accurately described by Sydenham, Frank, Scudamore, and others; and one of the constant characters of such a conformation is former or existing corpulence; and it is now known as consistent with the constitutional origin of this disease that the disposition to develop gout may be brought about by abnormal habits of existence; and if the hereditary predisposition is present, the conditions for developing the latent diathesis are more easily made efficient. All are agreed as to the influence of full living, with the free and habitual use of wine, and especially now as to the influence of what may be called gross living, great and indiscriminate consumption of animal and vegetable food, with indulgence in beer and malt liquors generally (BUDD and TODD); and it appears, both from the observations of Sydenham, Craigie, Wm. Budd, Todd, and Wood, that it is not so much the particular variety of alcoholic drinks used, as the mode and extent of the use, which tends to develop the gouty state; but it is believed that, of all wines in common use, those of the Rhine vintages are the least productive of gout. They are said to contain less alcohol than any of the Southern wines, and less than those of Portugal, Spain, Sicily, Cyprus, and Madeira. The influence of malt liquors is especially obvious in those examples of gout which occur in the lower classes. "There is a body of men," writes Dr. William Budd, "employed on the Thames, whose occupation is to raise ballast from the bottom of the river. As this can be done only when the tide is ebbing, their hours of labor are regulated by that circumstance, and vary through every period of the *night* and *day*. They work under great exposure to inclemencies of the weather; their occupation requires great bodily exertion, occasioning profuse sweating and much exhaustion. Each man drinks from *two to three gallons of porter daily*, and generally a considerable quantity of spirits besides. *Gout is remarkably frequent among them.*" Such an instance contrasts strongly with the prophylactic influence of

corporal labor displayed amongst other classes of the people *do not labor under the influence of gallons of malt liquor*; and interesting to notice that in the non-beer-drinking countries lower classes escape. Gout is rarely seen in Scotland.

A belief in the prophylactic virtue of labor and moderate living probably instigated the abrupt reply of Abernethy to the question "What is the cure for gout?" when he said, "Live on sixpence a day, and earn it." The disease very rarely occurs before puberty but is seen in both sexes under twenty. Many cases occur between twenty and thirty, but the period of greatest liability is perhaps from thirty to fifty. After this the chances of exemption increase with age, probably from the more temperate habits of advanced life. But at whatever age gout may appear, every attack establishes a greater disposition to another. Women often suffer greatly from gout, but not in an equal degree with men. Gout is never developed without being associated with indigestion, or as a consequence of impaired digestion from some error in diet or drink; of all wines, Port wines and Burgundy are most apt to induce a kind of indigestion as accompanies or precedes an attack of gout. The immediately exciting causes of this disease are very much similar to those of rheumatism. In thus describing the *Pathology* of gout, the cause and constitutional source of the disease have been sufficiently indicated.

The varieties of this disease, in addition to the acute form, are (2.) *Chronic Gout*—a persistent constitutional affection, characterized by stiffness and swelling of various joints, with deposits of lithate of soda; (3.) *Gouty Synovitis*.

**Symptoms.**—These vary according as the disease attacks joints, the stomach, or the intestinal canal, but the proportion of frequency with which these different parts are attacked is not ascertained. It may be acute or chronic, and when the viscera are affected, it has been termed irregular, retrocedent, or misplaced gout. Sydenham was himself a great sufferer from this affection and labored under it for more than thirty-four years. He thus describes an acute attack or fit:

"It comes on a sudden towards the close of January or beginning of February, giving scarce any sign of its approach, except that the patient has been afflicted for some weeks before with a bad digestion, crudities in the stomach, and much flatulency and heaviness, which gradually increase till at length the fit begins. The patient goes to bed, and sleeps quietly till about two in the morning, when he is awakened by a pain, which usually seizes the great toe, but sometimes the heel, the calf of the leg, or the ankle. The pain resembles that of a dislocated bone, and is attended with a sensation as if water just warm were poured upon the member; and these symptoms are immediately succeeded by a chilliness, shivering, and slight fever. The chilliness and shivering abate in proportion as the pain increases, which is mild in the beginning, but gradually becomes more violent every hour, and comes to its height towards evening, adapting itself to the numerous bones of the tarsus and metatarsus, the ligaments whereof it affects so as sometimes to resemble tension or laceration of those ligaments, sometimes the gnawing of a dog."



and sometimes a weight and coarctation or contraction of the membranes of the parts affected, which become so exquisitely painful as not to endure the weight of the clothes, nor the shaking of the room from a person walking quickly therein; and hence the night is not only passed in pain, but likewise with a restless removal of the part affected from one place to another, and a continual change of its posture. Nor does the perpetual restlessness of the whole body, which always accompanies the fit, especially in the beginning, fall short of the agitation of the gouty limb. Hence numberless fruitless endeavors are used to ease the pain by continually changing the situation of the body and the part affected, which notwithstanding abates not till two or three in the morning—that is, till after twenty-four hours from the first approach of the fit. Being now in a sweat, he falls asleep, and upon waking finds the pain much abated, and the part affected to be swelled; whereas before only a remarkable swelling of the veins thereof appeared, as is usual in all gouty fits.

“The next day, or perhaps two or three days afterwards, the part affected will be somewhat pained, and the pain increases towards the evening, and remits towards break of day; and what we call a fit of the gout is made up of a number of these small fits. At length the patient recovers, which, in strong constitutions, and such as seldom have the gout, often happens in fourteen days; and in the aged, and in those who have frequent returns of the disease, in two months; but in such as are more debilitated, either with age or the long duration of the distemper, it does not go off till summer advances.”

In aggravated cases it attacks both feet, the hands, wrist, elbows, knees, and other parts; sometimes bending the fingers crooked and motionless, and at length “forms stony concretions in the ligaments of the joints, which, destroying both the scarf skin and the skin of the joints, stones not unlike chalk or crabs’ eyes come in sight, and may be picked out with a needle. Sometimes the morbid matter is thrown upon the elbows, and occasions a whitish swelling almost as big as an egg.”

During the first fourteen days the urine is high-colored, and after separation lets fall a kind of red gravelly sediment, and not above a third part of the fluids taken is voided by urine during the febrile paroxysm, the bowels also being generally constipated during this time. The fit is accompanied throughout with loss of appetite and chilliness of the whole body towards the evening.

The urea is probably not below the normal standard, its elimination not appearing to be interfered with during the paroxysm (GARROD, PARKES). The elimination of uric acid by the urine is impeded during the paroxysm; but it exists in large amount in the blood. It has also been suggested, as a hint for further inquiry, that the substances which, in a healthy state, would furnish urea, are converted into uric acid in gout (GAIRDNER). The phosphoric acid is also greatly lessened, and its retention is probably as common as that of the uric acid. Albumen is not uncommon in small quantities, but its occurrence is generally temporary; and if it is persistent, there is reason to suspect the chronic disease of the kidney which is produced by gout (TODD); and casts without albumen may be found in the urine sediment. After the paroxysm the water is usually copious; and the uric acid increases as the fit is

passing off, just after the paroxysm. Before the paroxysm of the urine is found to be very deficient in solids, especially in uric acid, extractives, and phosphate of lime. There is, in fact, a diminution of all the chief ingredients before the paroxysm; and during the paroxysm there is insufficient elimination of uric and phosphoric acids, while the urea and sulphuric acid are in sufficient amount; and after the paroxysm the elimination again increases (PARKES, *l. c.*)

When the fit is going off, a violent itching seizes the foot especially between the toes, and the skin peels off.

When the disease has become chronic, or, as Sydenham terms it, inveterate, "after yawning, especially in the morning, the joints of the bones of the metatarsus are violently stretched, seem to be squeezed with great force, as if with a strong hammer. And sometimes, though no yawning has preceded, when the patient has disposed himself to sleep he feels a blow on a sudden, as if the metatarsus were breaking in pieces, by a large stick, so that he wakes crying out with pain. The tendons of the muscles of the tibiae are sometimes seized with so sharp and violent a convulsion or cramp, that if the pain it occasions were to last only a short time it could not be borne with patience."

After many racking pains the succeeding paroxysms become less painful, when "instead of the usual external pain, a certain sickness, a pain in the belly, a spontaneous lassitude, and sometimes a tendency to diarrhoea succeeds." Besides the pain and sickness the patient becomes lame, and almost incapable of motion, and perhaps obliged to be wheeled or carried from room to room; and he is not only reduced to this helpless condition, but to complete his misery, his mind sympathizes with his body. "For every paroxysm may be justly termed a fit of anger, the rational faculty being so enervated by the weakness of the body as to be disordered on every trifling occasion, whence the patient becomes as troublesome to others as he is to himself."

Another form of chronic gout is known as *atonic gout*, when the joints enlarge, and the tissues and ligaments become thickened, and are the seat of various effusion, so as often to distend and even to dislocate the bones; and yet, if the patient be kept quiet, he suffers no pain. The general symptoms, however, are most distressing. The patient suffers from loss of appetite, indigestion, sickness, nausea, flatulence, acid eructations, pains of the stomach, cramps in the legs and in various parts of the body; also great dejection of spirits, vertigo, palpitation, fainting, asthma, and also, perhaps, from stone or gravel. These affections, in some form or other, continue with occasional intervals during the remaining life of the patient, who believes that he has the gout *flying* about him, and that he should be well if he had a regular fit.

In the course of this disease there may be metastasis to the stomach or other part, and the affection is then termed "*retroceded gout*," the pain in the joints being trifling, or having entirely subsided. The term is "*applied to cases of gout in which some internal organ becomes affected on the disappearance of the disease from*

*joints, and should be referred to acute or chronic gout."* When the metastasis is to the stomach or intestines, it may be either of a spasmodic or inflammatory character. The spasmodic is the most frequent. The patient is seized with violent pains in the stomach, with faintness, coldness of the extremities, and a quick, small, and scarcely perceptible pulse, accompanied with much flatulence, acidity, or vomiting. If on the contrary, the attack be of an inflammatory character, the pain is perhaps equally great, but is increased on pressure, and there is more reaction, some fever, a fuller pulse, with vomiting, and perhaps obstinate constipation. The duration of these attacks is short, as the patient must be quickly relieved or quickly perish. Besides metastasis to the stomach and intestines, this retrocedence may take place to other parts, as to the testicle, bladder, rectum, or to the head; and in the latter case the patient may die apoplectic. The transition of the gouty virus is often marked by a pain shooting along the nerve, as sudden and as rapid as a galvanic shock, and so violent as to have been compared to stabbing with a knife. Besides being thus a migratory disease from part to part, gout often alternates with other chronic diseases, such as *asthma* and *rheumatism*, and may coexist with them.

The functions of digestion, and especially the hepatic and urinary secretion, are much deranged in all cases of gouty paroxysms. Besides loss of appetite, flatulence, heartburn, stomach-ache or colicky pains prevail, the tongue is loaded, the bowels are bound, and air, with impacted *fæces*, distends the intestines, especially in the epigastric and umbilical regions. The hypochondriac regions, especially the right, are the seat of painful tension and uneasiness. The first alvine dejections are generally solid and dark-colored, not unfrequently very fetid; and in some instances large quantities of dark-colored excrement are brought away.

The urine, when scanty and of a deep red color, is voided with pain and scalding along the urethra. The sediment already mentioned, and soluble in water, is rose-colored or lateritious, and is deposited during the whole course of the attack, and its appearance is not confined to the close or subsidence of the febrile symptoms, though at that time it is more abundant. It consists of urate of soda, the phosphoric salts, and urea mixed in various proportions. When dyspeptic symptoms are associated with feebleness, a whitish magnesia-like powder, consisting chiefly of the phosphates, is deposited, or alternates with the other deposits.

When the paroxysm of gout has taken place, and terminated in the manner described, the patient appears to enjoy better health than formerly. The appetite is good, the mind more cheerful and active, the body more agile, and the patient is delivered from many feelings of languor and oppression with which he had previously been afflicted. In this state he may remain for two or three seasons without being conscious of any complaint or symptom which would indicate a return of the disorder. In the course of time, however, generally about the same season of the year, he begins to be sensible of the presence of some of his former feelings, and at

length a fit takes place much in the same manner as it did at first occasion. This second paroxysm is, in some instances, milder and less violent; in others it is accompanied with various circumstances which show some deviation from the first attack. Whatever be the mode or duration of this attack, another supervenes after a less lengthened interval; in some cases, the same year—the autumn—in others, next year in the spring or summer. In general the intervals are shorter the younger the patient. The most common, perhaps, is the biennial or triennial attack for the space of eight or ten years; then the attacks are annual, then twice the year; and in some severe cases the attacks are so frequent that the patient can scarcely be said to be free from gout the whole year round. Much in all this depends on the habits and constitution of the individual. The disease tends to acquire a chronic character and rarely quits the patient till it destroys him, either by complete lameness, helplessness, debility, or by attacking the heart or brain (CRAIGIE).

**Diagnosis.**—The diagnosis between gout and rheumatism is exceedingly difficult, so much so that nosologists have described with perfect pathological correctness a hybrid disease, and named it “rheumatic gout.” Mr. Hunter warmly opposed this compound appellation, as many have since done. Hunter’s opinion was founded on the belief that no two distinct diseases, or even distinct diatheses can coexist in the same constitution—a belief which must now be admitted to be incorrect (see vol. i, pp. 139, 207). We must do no less, therefore, recognize such a hybrid disease as Craigie, W. Spencer Wells, and Fuller have described, depending on the influence of the combined cachexia of gout and rheumatism.

Cases of chronic pain, with stiffness and swelling of various joints are thus far cases of chronic rheumatism; but when they are attended with deposits of lithate of soda, such cases are directed to the College of Physicians to be returned as cases of “*chronic gout*” and those in which there is marked distortion, as cases of “*chronic osteo-arthritis*”—a disease which may be defined as follows: “*an affection characterized by pain, stiffness and deformity of one or more of the joints, associated with deposition of new bone around them.*” This disease is also named “*chronic rheumatic arthritis.*”

**Prognosis.**—Every assurance office objects to a gouty person being liable to a disease which tends to shorten life from the wear and tear of the constitution it occasions. The objection is unquestionably well founded; for although a few persons may reach advanced age notwithstanding repeated attacks of gout, yet many die prematurely from this affection, or from asthma, disease of the heart, apoplexy, or from the accidents to which helplessness and debility render the patient liable.

**Treatment.**—Seeing that the disease is clearly of constitutional origin, its treatment resolves itself into—(1.) The selection and administration of those remedies which shall tend to subdue, control, or eradicate the latent disposition, constitutional tendency to gouty diathesis; (2.) The adoption of such means as may be said to be used to modify the severity of, or shorten, the paroxysm.

paroxysm must be interfered with cautiously. It is the means which nature takes to rid the constitution of the *materies morbi*, and which it undoubtedly relieves for a time, if allowed to run its course. The removal of the paroxysm does not necessarily remove the constitutional diathesis.

As to any local treatment during the fit, Sydenham writes, "If outward applications be required to ease the pain of the gout, I know of none, though I have tried abundance both on myself and others, and I have laid aside the use of topical remedies for many years." It is generally admitted that cold is dangerous, while warmth is productive of little relief. In some instances the urate of soda is deposited in such quantity that the skin ulcerates, and the salt is discharged in considerable abundance in a fluid state. It might appear the right practice to apply a poultice, and encourage the discharge, in order that, by its entire removal, the joint might be saved. This, however, is by some considered dangerous practice, for the discharge is so debilitating that patients are said to have sunk under this mode of treatment. It is much safer to wait till the chalk-stone becomes concrete, and then operate, by the smallest possible incision, for its removal. With respect to the use of cold water, the practice is as old as Harvey, and subsequently it has been adopted and abandoned by many practitioners. Dr. Parry had at one time two patients who attempted to cut short the fit by plunging their feet in cold water. The relief was instant, but in a few hours both were dead of apoplexy. The fatal result of this remedy in Sir Francis Burdett's case is another instance in point.

"A gentleman," writes Dr. Wood, "contrary to the advice of his physician, and anxious for speedy relief, ordered a bucket of water to be taken into his chamber at bedtime, with the view of employing it in this way. In the morning he was found dead in bed."

The "bootikins" of Horace Walpole, so strongly recommended and given away by him to all his gouty friends, seems to have been merely a fine bandage applied moist and firmly over the limb, and then a roller of oiled silk over it, giving moist warmth like a poultice.

A few leeches have been recommended to be applied to the part with great caution, if the inflammation is unusually violent; but Dr. Garrod is of opinion that in no case are they likely to prove efficacious, and their use may be attended with injurious consequences. Warm anodyne lotions or fomentations may be used, and the part afterwards lightly covered or incased in flannel or fine wool, while the limb is at the same time kept elevated. Dr. Wood uses a warm mixture of *tincture of camphor* with milk, applied by means of linen compresses, and frequently renewed. *Tincture of acmite* may be similarly applied (℥iiss. to ℥iv of milk). Blisters have been recommended, and are of most advantage in asthenic chronic cases, when the inflammation has a tendency to linger in the articulations, and to cause liquid effusion. In the early fits of



sthenic gout they are unnecessary; and are contraindicated in advanced gout, when the kidneys have become impaired, and in cases of extreme gouty deposits (GARROD). As a general rule, however, the less that is done to the *local affection* the better. If the pain is very intense, relief may be given by the application of a solution of atropine, made by dissolving from two to three grains of the alkaloid in a weak mixture of spirits and water, and applying to the inflamed joint by means of compresses of lint, preventing evaporation by a covering of oil-silk (GARROD).

[Great and long relief from pain may be had by the hypodermic injection in the neighborhood of the affected joint of one of the salts of morphia from one-fourth to one-third of a grain in ten minims of water.]

In the general treatment of gout, bleeding is now generally restricted to two methods, namely—(1.) By leeches to the part where the inflammation rises so high, or is so chronic, as to threaten the patient with the permanent loss of the use of some joint; and in cases of metastasis of the disease to the stomach or other internal organ, when leeches are absolutely necessary; (2.) By general bloodletting.

The following rules are laid down by Dr. Garrod as to the use of bleeding in gouty cases: (1.) Never bleed with the idea of directly subduing gouty inflammation; (2.) Never bleed in advanced gout or when the constitution is much weakened; (3.) If bleeding be required in order to relieve general or local plethora, abstract only as much as will effect that object, remembering that further depletion tends greatly to aggravate the disease and prolong its duration. Bloodletting produces a decided sedative action on the heart and other parts of the circulating system, and it has a most powerful influence upon the progress of gouty inflammation—an influence not merely limited to the removal of gout when it attacks the joint, but also of great efficacy in its marked and irregular forms, in ophthalmia of gouty habits, in gouty bronchitis, and in headache connected with the gouty diathesis (SIR H. HOLLAND, GARROD).

Sydenham, who was as great an enemy to purging as he was to bleeding, says,—“I am abundantly convinced, from much experience, that purging, either with mild or strong cathartics, whether used during the fit or in its declension, or in a perfect intermission or healthy state,” . . . . . “endangers the life of the patient by hurrying on the disease to the viscera, which were quite safe before.” The objection taken by Sydenham to purgatives was quite valid against those in use in his day, which were of the most drastic kind; but it may be laid down as a rule, that neutral salts are not only safe but efficient in relieving gout, though perhaps not to be depended on alone for its cure. The theory on which they are prescribed is, that the alkaline base of the neutral salt is absorbed, and combines with the insoluble urates deposited in the joints, forming a soluble sub-urate, which can readily be absorbed; and, again, the alkali being sent to the kidney, that organ is enabled to remove more uric acid, in a soluble state, from the system than under ordinary circumstances.

nary circumstances. The salts most in use are the *sulphates of magnesia* or of *soda*, and especially the former; and half a drachm to a drachm should be given every eight, six, or four hours, according to the state of the bowels and the acuteness of the symptoms. It is also necessary to afford some relief to the patient from his excessive suffering. With that view an anodyne should be added, such as the *tincture of hyoscyamus*, *syrup of poppies*, or *some preparation of opium*. This method of treatment relieves the patient and shortens the paroxysm; but when the relief is complete, it should be abandoned, for sometimes a paroxysm of gout will return even under its use.

*Colchicum* or meadow-saffron was long ago introduced as a specific in gout, of which the once popular "*eau médicinale*" was supposed to be a preparation. *Colchicum* is still, however, used, and is valuable for its purgative qualities; and in some cases it seems to be almost specific in its effects, and may be given as an extract or tincture, or as a wine, combined with some form of saline draught. Its mode of action is unknown. It very generally promotes secretion from the skin and kidneys; but it is useful even when it does not exercise any such physiological action. The quantity of uric acid and urea in the urine is said by some to be increased during its use (CHELIUS). Dr. Graves makes an opposite statement; and Dr. Garrod proves conclusively that *colchicum* does *not increase* the amount of uric acid. Dr. Laycock suggests that it may have a sedative effect on the vital actions going on in the tissues themselves, and so may arrest the formation of the gout poison.

The wine of the root of *colchicum* is generally the form preferred, *fifteen to thirty minims being given every four, six, or eight hours*, and this remedy ought to be continued for some time in reduced doses, after all symptoms of gout have disappeared (BUDD). The bowels ought to have been freely moved before *colchicum* is given; and a full dose having been given at first, much smaller doses may be continued, as from ten to twenty minims two or three times a day, in Seltzer or Vichy water, or in other alkaline solutions, and combined with *Iodide of Potassium*, carefully watching its effects on the pulse, and never allowing sickness or depression to ensue.

Scudamore's mixture of *colchicum*, *magnesia*, and *sulphate of magnesia*, is said to be an excellent purgative in gout, when a purgative is required. Although Dr. Garrod has shown that purging is not necessary to the action of *colchicum*; yet, as there are many who believe that the action of *colchicum* is promoted by its combination with laxative remedies, Dr. Wood is in the habit of using it in the following formula in a draught:

R. *Magnesiae*, ℥ss.; *Magnes. Sulphat.*, ℥ss.; *Vin. Colchici rad.*, ℥xx; *Aquæ Fluv.*, vel *Aq. Acid. Carbon.*, f℥iss.

Mercury, in the form of "blue pill," or in the form of the "compound calomel pill" (*Pil. Plummeri*), followed by the draught just written, is useful where it is desired to act upon the intestinal secre-



tions. But mercury in any form must not be given if the contains albumen. If mere constipation is to be got rid of, *r pill*, *colocynth and scammony pill*, or *podophyllin*, may be advantageously used. The iodide of potassium has been much recommended by Mr. Spencer Wells. Alkaline remedies are of much value in the treatment of the paroxysms of the joint affection, and may be prescribed in the form of the *bicarbonate* or of the *citrate* or *acetate* of potash. Dr. Garrod prefers the *bicarbonate*, and Dr. Parke has shown that it increases the elimination of uric acid and its solids by the urine (Parkes *On the Urine*, p. 298). Much benefit is also derived from the continuous administration of salines in small doses, repeated two or three times a day, in a *very* dilute form always on an empty or nearly empty stomach, and some little before food (GARROD).

If acute gout should have “retroceded,” as it is called, and the stomach or intestinal canal be inflamed, *leeches* should be applied to the abdomen or epigastrium, followed by a poultice, while the *nitrate* of salts, with the *tincture of hyoscyamus*, should be given at least once in four or six hours. It is very rare that more active medicine is necessary.

In chronic gout the treatment is the same; but in *atonic* cases some light tonic medicine may be added, as *five to ten grains of citrate of iron*. A large number of chronic cases, however, though the general health is improved by this treatment, are often altogether unrelieved as to the local symptoms, and are often quite unable to assist themselves. In these instances the turpentine is apt to be beneficial, as *spruce beer*, the *Canadian balsam*; or one drop of the *oil of turpentine* may be taken in an effervescing draught three or twice a day. Sydenham's method of treatment by *manna* may also be tried.

If the chronic or *atonic* gout should become retrocedent, and the stomach and intestinal canal be the seat of the spasmodic form of the disease, Sydenham strongly recommends that laudanum should be given; but perhaps the following draught is more efficacious, namely: *R. Aquæ Camphoræ, 3x; Sp. Ætheris Sulphurici, Sulphatis Magnesicæ, 3ss.* It will remove from the stomach any undigested matter which may remain as an irritating cause. It should be given every hour till the patient is relieved; and while he is being prepared hot brandy and water should be freely administered, or the *spiritus ammoniæ aromaticus*, in doses of sixty minims, and hot cloths applied to the abdomen, as well as hot bottles to the feet.

Sydenham recommends, from experience in his own case, large doses of *manna* in all cases of what he terms “bloody urine.”

[About eight years since the *salts of lithia*, particularly the carbonate and citrate, were proposed by Dr. Garrod as a remedy for gout. They have since been extensively used, and general testimony is favorable to them. Lithia is a constituent of the human body, of some plants, and can be shown, by means of the spectrum analysis, in the ashes of blood, and even of a cigar; it exists in many of the mineral waters w

have had a therapeutic reputation in the disease, especially in those of Baden-Baden; also in the Carlsbad, Aix-la-Chapelle, Marienbad, and some of the Vichy waters. The salts of lithia are actively diuretic, powerful solvents of lithic acid, and the carbonate of lithia is a great neutralizer of acids. Taken for a long period, in a very dilute form, they would seem to prevent gouty paroxysms in chronic cases, and some evidence has been given of their power of rendering the affected joints more movable, and of causing some solution and absorption of the chalky matter. They certainly act rapidly and actively in hindering the deposition of urates and uric acid in the urine, and in the calculous tendencies of many gouty subjects must necessarily be of value (GARROD). Dr. Flint says: "From a limited trial of this remedy I have been led to form a favorable opinion of its utility in both the acute and chronic form of gout." In several cases the writer has seen decided benefit from its use. The dose of the carbonate of lithia is from five to ten grains dissolved in aerated water; that of the citrate, eight, ten, or more grains; and given three or four times daily. It is a safe remedy, and may be long continued in less frequent doses. Aerated lithia water is largely manufactured in New York by Dr. Hanbury Smith, and Messrs. Schultz and Warker.

The *phosphate of ammonia*, as a remedy in gout, was first brought to the notice of the profession by Dr. T. H. Buckler, of Baltimore (*Am. Jour. of Med. Sciences*, Jan., 1846). In chronic gout it is said to have been used with good results, though the writer's trials with it have been disappointing.

In chronic gout *iodide of potassium* is a remedy of service, especially when the pains are increased at night, and by the heat of the bed, though in the writer's experience the *bromide of potassium* has proved more promptly relieving. Garrod says of the iodine: "It is also useful in removing the recent thickening in the tissues around joints, but proof is still wanting of its possessing any power of causing the absorption of the urate of soda. In gouty inflammation, when fluid has been thrown into the cavities of the joints, and has been slow of absorption, the administration of the iodide of potassium has often appeared to be attended with great advantage." Of *guaiacum* he writes: "Within the last few years I have given this drug extensively, and with great advantage; it is especially useful in the asthenic gout of old subjects, but to young[er?] patients it may also be given with benefit."

Dr. Duncan has proposed, and, he states, used with much success, hydrochloric acid in the treatment of gout. He does not put it forward as a specific, to be used under all circumstances and in every stage of the disease, nor does he exclude other agents as may be obviously demanded to meet special contingencies; but it seems to him to act more directly upon the primary cause of the disease, and to hold out a greater prospect of effecting a cure than any antacid remedy. Though he has prescribed the acid alone, with the effect of immediate relief, his usual formula is:

R. Acidi Hydrochlor. dil. ℥iiss; Spr. Chloroformi, ℥ij; Tinct. Colchici, ℥j; Infusi Cascarillæ, ad ℥viiij. Dose, one fluid ounce every third hour.

In chronic gout, particularly in weakly persons, the writer has seen much benefit from the use of quinine, especially in combination with iron and arsenic in very minute doses, in the form of the chlorides of iron, quinine, and arsenic. Dr. Ranke has stated that quinine diminishes the amount of uric acid in the urine. He gave twenty grains of the sulphate, and found that the excretion of the acid was reduced one-half; its influ-

ence continuing about two days. In some observations made Garrod, he found the average of uric acid in the urine, for two days, 5.89 grains when no quinine was given, and 5.37 grains when eight grains of the salt was taken each day in divided doses. "Assuredly," Dr. Garrod remarks, "that Dr. Ranke's statement is correct, and that the elimination of uric acid is much lessened for two days after the commencement of the treatment. It is a matter of much interest to inquire if the effect is due to a diminution of formation, or defective excretion, from the kidneys. From my observations I was inclined to ascribe the effect to the sudden and powerful impression of the drug upon the nervous system, influencing the excretion of uric acid, and not to any decrease in its formation in the system. I shall, however, be unwilling to offer a strong opinion upon the subject at present, as I consider that further experiments are required. It is advantageous to unite small doses of colchicum to the quinine" (*loc. cit.* 863). The pills of Dr. Debout, and of Dr. Becquerel, are composed of sulphate of quinine, extract of digitalis, and extract of the seeds of colchicum. Dr. Trousseau uses Becquerel's pills, and says he has seen under their use the pain of a paroxysm of gout abate in seven days (*Clinique Med.*, 2ième ed., vol. iii, p. 355). The formula for Becquerel's pills is:

R. Quiniæ Sulph., ℥j; Ext. Digital. Alcohol., gr. iij; Ext. Sem. Colchicid., ℥ss. Divide in pil. no. x. Dose, two or three daily, for four or five days.

Laville's *Anti-gout Liquid and Pills*, a French preparation, are esteemed by many sufferers, who assert that they have used them with excellent effect. According to Dr. Tanner, "From an analysis the liquid contains the active principle of colocynth, quinine, and cinchonine, and unimportant salts of lime. It is used at any period of the attack; a teaspoonful being taken in sweetened water or tea, and repeated in six hours if the pain continue, and the bowels be not moved. Twenty-four hours are to elapse before the next dose, when half the quantity is to be taken daily, two or three times, unless the bowels are irritable. The pills consist of *physalin* [the active principle of the *Physalis alkekengi*, a perennial herbaceous plant growing wild in the south of Europe, and cultivated in our gardens] mixed up with silicate of soda" (*l. c.*, p. 120), and administered with chamædrys. One is taken just before a meal, for several weeks.

In the chronic forms of the disorder, where there is no organic disease of the brain, heart, or lungs, *wet-packing*, and the *Turkish bath*, are cautiously and occasionally used, but not during a paroxysm, under the immediate supervision of the attending physician, would, sometimes, be of service, in lessening stiffness of the joints, and restoring, or improving, the action of the skin. Trousseau speaks well of the friction of the skin, and understands it well employed. Hot salt-water baths are beneficial. In chronic gout, Dr. Goolden has seen the inhalation of oxygen followed by clear urine, and great relief; and in some cases cures have resulted.]

As indiscriminate feeding appears to have a great influence upon the production of gout, so we expect the regulation of diet should have great influence in its removal. During the fit the diet should consist of slops and light puddings, and afterwards white fish, when the paroxysm has terminated. This disease is so distressing that many persons are inclined to diet themselves with great strictness during the interval. Sydenham says that a milk diet, or drink milk as it comes from the cow, or boiled, without adding anything

to it, except, perhaps, a piece of bread once a day, had been much used for twenty years past in his time, and had done much service to gouty patients. But on quitting it, and returning to the mildest and tenderest diet of other persons, gout has immediately revived; and he adds that many cannot bear this regimen. An entirely water regimen he considers hurtful. The most digestible meats, such as mutton, well-kept beef and poultry, with the white kinds of fish, as codfish, sole, and whiting, may be eaten; but salmon, veal, and pork are to be avoided, as well as cheese, salads, highly seasoned dishes, and rich sauces, or other "elaborate preparations on the part of the cook."

[Dr. Garrod very sensibly remarks, that "there should be a due admixture of animal and vegetable food; it is an error to suppose that an animal diet tends more to the formation of uric acid than a vegetable one. The tortoise, feeding on a simple lettuce, excretes a large quantity of urate of ammonia, far more in proportion to the weight of the animal than is excreted by the dog exclusively nourished with meat. Vegetables, as potatoes, greens, and the like, may be partaken of with advantage; the soluble salts they contain are of value in keeping up the activity of the secreting organs. The same remarks hold good with regard to soft fruits when eaten in moderation, as strawberries, grapes, and oranges; also other fruits when stewed or baked, as apples and pears; but these latter, as likewise plums, and stone-fruit in general, should be avoided in a raw state. Extreme moderation should be exercised when saccharine fruits are eaten, as sugar is liable in many subjects to lead to the production of acidity. The same precaution is necessary in reference to the addition of sugar to other articles of diet."]

If alcohol in any form is required, it may be taken in the form of a little weak brandy, gin, or whiskey and water, or pure sherry, like Amontilado, or Manzanilla. Ports, Burgundy, and sweet wines must be avoided; but wines of the Rhine vintages may be taken, if they do not contain a large percentage of alcohol. All of these spirits ought to be taken much diluted with Seltzer or soda water. His recommendations are, that we should be early to bed, keep the mind free from all disquietude, live with the greatest moderation, clothe ourselves warmly, and ride on horseback. When exercise cannot be taken, friction over the surface of the body is exceedingly useful. The patient should be rubbed down with a flesh-brush once or twice a day, just as a horse is groomed.

One other point with regard to the treatment of the patient during the fit is, that if it be necessary to move him, either on account of his restlessness or other cause, this should be done with great care and tenderness by the attendants; for although the pain may be latent while the parts are quiet, yet the least shock often causes the most excruciating agony.

Several mineral springs have obtained reputation in the treatment of gout; and Dr. Garrod lays down the following general rules as a guide to the use of them:

1. Their use should be prohibited when there is much structural disease in any important organ, especially in the heart or kidneys;

and even when organic mischief is slight, the greatest caution in their use is necessary.

2. They are to be avoided when an acute attack is either present or threatening.

3. The particular mineral water must be selected according to the nature of the individual case: for the robust, and those of robust habit, the alkaline saline springs should be chosen; when torpidity of the bowels predominates, the purgative waters should be used; when the skin is inactive, the sulphur springs may be used; when much debility prevails, or an atonic state exists, then more simple thermal springs may be prescribed.

[It is important that the physician should know when to advise when to forbid the use of what Dr. Trousseau styles "a most perilous medication;" and also what waters are adapted to different cases. The waters employed in gouty cases contain either alkaline carbonates, chlorides, or sulphates. Some of the waters are impregnated with sulphuretted hydrogen; and another class owe their powers chiefly to the iron they contain. Many of the springs are of an elevated temperature; some of mean heat; others cold.

All mineral waters rich in saline matters, if taken too freely or long, usually cause febrile disturbance; the system becomes oppressed; there is a feeling of heaviness, languor, or agitation, followed by loss of appetite, thirst, a furred tongue, and heat of skin; sometimes by vomiting and diarrhoea. This is probably due to the blood becoming saturated with the saline matters, from the excreting organs being unequal to the task of eliminating the whole quantity introduced during the treatment.

The several *Vichy* springs are all rich in carbonate or bicarbonate of soda,—about forty grains to the pint. Some have a temperature of 100° Fahr.; others are cold. When taken in moderate doses the urine becomes neutral or alkaline, without its transparency being affected; used as a bath their effects are similar. From the soda they contain they act upon the liver; and from the amount of liquid absorbed, and the temperature of the water of some of the springs, they also influence the function of the skin. They are adapted to the treatment of acute gout in strong persons in whom the function of the liver and digestive organs is disordered, and are contraindicated in very chronic cases, especially if there is a tendency to the rapid formation of chalkstones, or the system is much enfeebled. In Garrod's experience is that, in this latter class of cases, they rather tend to favor the formation of these concretions. The use of the bath is combined with drinking the waters, or is alone employed if they disagree with the stomach.

*Wiesbaden* waters contain a large amount of chloride of sodium, and are of high temperature,—160° Fahr. They are more stimulating to the various functions, and less generally debilitating than those of *Vichy* and consequently better adapted to cases where the circulation is sluggish and the secretions deficient. They are powerless to remove any solid deposition of urate of soda.

The waters of *Aix-la-Chapelle* are slightly saline, high in temperature,—135° Fahr.,—and, in addition to the chloride, contain some carbonate and sulphide of sodium, with free sulphuretted hydrogen. They are stimulating to the secreting organs, particularly to the skin; they are contraindicated in cases where there is cutaneous torpor, and are useful in removing rigidity of the joints.



*Carlsbad* waters are much in vogue for the treatment of gout; they are rich in sulphate of soda, and contain carbonate of soda and chloride of sodium; their temperature is 167° Fahr. They are purgative, diuretic, and diaphoretic, and beneficial where there is congestion of the liver, with constipation; but should be avoided by weakly patients.

The waters of *Baden-Baden* contain chloride of sodium, and a little iron, but are said to be rich in *lithia*. According to Dr. Ruef, they have the power of removing visible deposits of urate of soda.

The waters of *Kissingen*, *Marienbad*, *Homburg*, *Ems*, &c., are saline, and are used in the treatment of gouty conditions.

The waters which possess but little solid matter, and which have a reputation in gout, are those of *Wildbad*, *Töplitz*, *Gastein*, *Buxton*, and *Bath*. They are all of somewhat elevated temperature, are chiefly used in the form of baths, and appear to be peculiarly adapted to the treatment of the disease in the infirm and old, great benefit being often had from their employment\* (GARROD, *Nature and Treatment of Gout*, 2d ed.; Article *Gout*, Reynold's *System of Medicine*, vol. i, p. 871). Trousseau speaks well of the waters of *Aix in Savoy*; and in the asthenic forms, particularly if the patient is anæmic, he recommends the chalybeate waters of *Spa* and *Pyrmont*.]

The irritable state of mind of the patient during the paroxysm has been mentioned; and it is well known that slight moral causes will often produce a fit, while powerful emotions have sometimes cured one. It is quite essential, therefore, that the minds of gouty patients should be kept as tranquil as possible, both for their own sakes as well as for the comfort of others.

[Dr. Garrod holds that acute gout is quite as controllable, and as much under the influence of remedies, as any other inflammatory affection. The duration of the paroxysm and the amount of injury to the joints depend much upon the treatment. The more chronic forms are likewise under the control of the physician, if not for their radical cure, yet for so much relief as will enable the patient to enjoy life, and prevent further increase of the mischief, so liable to ensue, if the disorder is allowed to run its own course, or if recklessly tampered with. The treatment, founded on Cullen's aphorism of trusting to patience and flannel, is to be greatly deprecated. Although a plan may be sketched, applicable to most cases, still each individual case not only exhibits its own peculiarities, but demands, in certain respects, a separate treatment. Owing to the liability of gout to recur, and to acquire a firmer hold on the constitution at each visitation, it is prudent, in the intervals of the attacks, not only to regulate the diet and regimen, but to have recourse to means, scarcely to be called medicinal, by which the blood may be kept free from the impurities which lead to the production of the disorder.]

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\* [For an excellent condensed account of the various mineral waters, see *Tanner's Practice of Medicine*, Am Ed., p. 801; also Durand-Fardel's *Dictionnaire Général des Eaux Minérales et Hydrologie Médicale*. Paris, 1860.]

## ANÆMIA.

LATIN Eq., *Anæmia*; FRENCH Eq., *Anémie*; GERMAN Eq., *Anaemie*—*Sy Blutarmuth, Wassersucht*; ITALIAN Eq., *Anemia, Idropisia generale*.

**Definition.**—A special morbid state in which there is either a *re* diminution of the mass of blood (ANDRAL), with the general composition of the blood differing from the normal standard; or in which the quantity of blood is diminished, and the liquor sanguinis is watery, poor in solids, and containing an excess of salts. These conditions, coexisting with relative deficiency of the red blood-corpuscles (VOGEL), and a diminution of the urine-pigment (PARKES), constitute anæmia.

**Pathology.**—The term *anæmia* literally means absolute deficiency of blood—a condition of existence obviously not possible.\* A diminution in the quantity of blood, with an alteration in its composition, almost never occurs alone, but is generally a morbid state resulting from many exhausting morbid processes, peculiar to the wasting constitutional diseases as tuberculosis and cancer. In many of these diseases the blood-mass is evidently diminished. We have indications of this diminution in the small pulse, in the pale bloodless appearance of the countenance and surface of the body generally, especially seen in the lips and gums, and in the small, collapsed veins, particularly obvious by contrast on the pallid skin. In such cases one would never think of drawing blood to know whether or not its constitution was changed; but in cases where the opportunities for examination have occurred, the blood-corpuscles have almost always been found relatively diminished.

[Analyses of the blood of anæmic patients show constantly diminution in the red globules. In 24 cases of confirmed spontaneous anæmia Andral found the mean of the red globules 64 in 1000 (normal amount 127). In an extreme case of chlorosis, it was reduced to 27. But Dubuisson found the proportion of red globules in anæmia to be 80 and 40; Denis 64 in 1000 parts. Sometimes a change in the physical characters and constitution of the red globules takes place. Andral has found them altered in form, smaller, and broken into fragments; Dr. G. Owen Rees rounder and swollen; and Virchow says the melanin corpuscle of Schulz is met with in anæmic blood. The white corpuscles lessen in the same proportion as the red globules (BECQUEREL). The white corpuscles lessen simultaneously and in the same proportion as the red, their normal relations of quantity being undisturbed (VIRCHOW, DONNE, ROBIN, BECQUEREL, and RODIER). The density of the blood is less (L'HERITIER, DENIS); Bouillaud, after repeated bleedings, saw it fall from 7° to 5.50° and 5.25° of the areometer of Beaumé.

The causes which immediately hinder the due production of the red globules, or hasten their abnormal destruction, are involved in obscure

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\* [Diminution of the red globules relatively to the serum (aglobulia, hypoglobulia, oligocythæmia); diminution of the whole amount of circulating fluid (oligæmia, hypæmia); deficiency in the quality of the blood (spanæmia); augmentation of the absolute amount of serum without diminution of that of the globules (hydræmia, serous plethora).]



An attempt has been made to localize the proximate cause of anæmia, by referring it to functional trouble in the organs supposed to be engaged in the formation of the red globules,—the spleen, glands, &c. Their destruction, as well as their construction, probably, depend on some property of the blood itself.]

The result of this morbid condition of the blood chiefly betrays itself—(1.) Upon the *vascular system* generally; and (2.) Upon the *metamorphoses of tissues*.

The bloodvessels contract in proportion to the diminution of the blood-mass. The *arteries* contract generally; and the pulse, whenever it can be felt, is found to have become small and tense. The *capillaries* also contract; the skin and mucous membranes becoming pallid and comparatively bloodless. The *heart's* action in extreme cases becomes irregular, and the whole circulation generally is disturbed.

As regards the metamorphoses of tissues the muscles and the nervous system appear to suffer first; debility and prostration, both bodily and mental, occur; and in severe cases sensation may be lost, syncope is frequently apt to occur, and even death may result.

The disease seems to go through different changes, according to the causes which bring about the anæmic condition. After mere loss of blood in quantity, for instance, the water and the salts are renewed with most rapidity, the albumen later, then the colorless corpuscles, and last of all the red corpuscles. Hence it is that anæmia is frequently associated with a watery condition of the blood, as well as with a lessening of the number of the blood-cells. It is a matter of the greatest importance in practice to distinguish, if possible, between these conditions, with a view to a rational means of treatment.

Those exhausting diseases which are attended especially with deranged nutrition and sanguification, such as *Bright's disease*, *carcinomatous diseases*, *tuberculosis*, and *suppuration*, lead also to the development of that form of anæmia in which the *liquor sanguinis* is in excess, in which the blood is poor in albumen, containing an excess of salts, and in which the blood-cells ultimately become deficient. A tendency to *general dropsy* or to diarrhœa ensues, nutrition becomes still more disordered, and new formations are apt to become developed. By "*general dropsy*" is understood "*an accumulation of serum in the areolar tissue, with or without effusion into the serous cavities.*"

It is very important to know, in a practical point of view, that every acute disease which occurs in an anæmic individual assumes a peculiar character: a very high degree of debility and prostration ensues, convalescence is protracted, and all severe intercurrent diseases acquire a lingering course.

**Symptoms.**—In combination with an investigation of the blood, the symptoms and signs of *anæmia* are already read in its pathology. It is only by general symptoms, however, that the physician is able circumstantially to conclude that relative diminution of the cells of the blood coexists with deficiency of the mass. Great debility is

a prominent and striking feature. The skin is of a pale, waxy color; [the superficial veins are not prominent, fill slowly on compression, and can often be traced only by bluish lines;] the "whites" of the eyes have a bluish aspect; the mucous membrane of the mouth is colorless; the gums and lips are white; and the tongue pale, large, and flabby; the pulse in general is about 80, but feeble, and easily excited—the least stimulus, the least mental agitation or movement of the body, produces great momentary acceleration of the pulse.

[Andral found the body temperature always natural; though anæmic patients may suffer from cold extremities, the thermometer in the rectum indicates the normal standard.]

Respiration is hurried on the least exertion. The appetite is lost, and thirst prevails, [the mouth being generally dry. Anæmic persons usually perspire but little.] When the disease has existed for some time, œdema of the feet and ankles may supervene; and finally, sweating, in the extreme stage of *anæmia*. It occurs frequently in the last stages of severe and exhausting sickness, of the lingering and prolonged kind, where a high degree of paleness of the skin and mucous membranes exists, with a small weak pulse, collapse of the veins, and a small volume of the heart, spleen, and liver.

[Anæmic persons suffer constantly from mental depression; they are very impressionable, and easily excited; their temper is capricious, irascible, and irritable; and there is intellectual torpor. Their sleep is broken, and disturbed by dreams. They are very liable to headache, neuralgia of the scalp, and to dizziness and ringing in the ears. Vision is not infrequently troubled, and so-called amaurotic symptoms, homophobia, and disorders of accommodation (DESMARRES) happen. There may be temporary analgesia and anæsthesia, general or partial, or hyperæsthesia, particularly spinal.]

In recent days diagnostic characters of *anæmia* have been recognized in various *murmurs* which may be heard in some parts of the vascular system of anæmic patients. Although the seat, the cause, and the signification of these *murmurs* have been very much disputed, yet the following account may be given of them, based on numerous observations and investigations, especially those of Valentin and of Walshe.

There are three kinds of anæmic *murmurs* which may be distinguished, namely—(1.) *Cardiac murmurs*; (2.) *Arterial murmurs*; and (3.) *Venous murmurs*.

The *cardiac anæmic murmur* gives forth what is usually called "bellows sound," sometimes intense, sometimes faint, and it generally accompanies the first ventricular sound of the heart. The position where these *systolic cardiac murmurs* is best heard is of some importance, as indicating anæmia rather than disease of the heart. They are heard towards the base of the heart in anæmia, rather than towards the apex, as in organic disease of the valvular system.

(WALSHE). The *diastolic* murmurs are really of venous origin ; and their intensity is more or less increased by suspension of the respiration.

The *cardiac anæmic murmur* occurs most frequently in true anæmia, especially if cardiac excitement is associated with it; and simultaneously with it we frequently hear *arterial* and even *venous murmurs*. It has been observed by Vogel and others, however, that while the presence of these *cardiac murmurs* may be employed as an auxiliary sign together with others in the diagnosis of anæmia, yet it is not to be concluded from their non-existence that anæmia is not present. Such *murmurs* are not heard in all anæmic patients; and, although present, they are not in every case due to anæmia, because they occur in organic diseases of the heart, such as valvular diseases and endocarditis; and a physician trusting to and acting upon stethoscopic signs *merely*, without a due appreciation of general symptoms, seriously injures the patient, and does an injustice to the science of medicine.

The *arterial anæmic murmurs* are seldom heard. They consist of an intermittent blowing, sometimes soft, sometimes sharp sound, perceived to be synchronous with the beat of the pulse, which gives at the same time a thrill to the finger, so that the *murmur* may be inferred by practice from the nature of the pulse. It is only in the larger arteries in which they are heard, such as the brachial, the subclavian, the femoral, the carotid, and abdominal aorta. They are most frequently heard after great losses of blood; and sometimes also in *chlorosis*. Vogel has heard them during the paroxysms of intermittent fever, while they are absent during the intermissions. They may be heard in typhus fever. No positive conclusion can therefore be formed from them alone regarding anæmia.

The *venous murmurs*, or *hums*, significant of anæmia, are *continuous*, humming, buzzing, occasionally musical, singing murmurs, easily distinguishable from the blowing *intermittent* arterial murmurs. They are most frequently heard on the right side of the neck, at the junction of the external and internal jugular vein; but they may also be heard in the femoral veins in their maximum degree of intensity. They may be heard, also, over the course of the superior longitudinal sinus, and at the maximum intensity over the *torcular Herophili* (WALSHE). They are supposed to be produced by abnormal oscillations of the venous valves, or by sharp collision among the blood-discs, passing from sideward veins into a large vessel. When the venous murmurs are strong, they may not only be *heard*, but also felt as a gentle thrill, by placing the finger on the part.

These venous murmurs are seldom absent in well-marked anæmia. Nevertheless, anæmia is not to be positively inferred from the mere presence of any one of these murmurs.

[The characters of the anæmic murmur are: (1.) It accompanies the first sound of the heart, which it commonly more or less masks; (2.) It is short; (3.) It is usually soft, but not always, for it may be harsh, though not absolutely grating; (4.) It is exceedingly local, the maximum of in-

tensity being generally in the fourth intercostal space, near the sternum, more rarely limited between the fourth and fifth; beyond these points it is never heard; it follows the course of a line drawn from the point of greatest intensity to the inner portion of the right clavicle. Dr. Pean in his article *Anæmia* (*Dict. Encyc. des Sciences Médicales*, t. i. p. 392, Paris, 1866), says, "The site of the cardiac anæmic murmur is at the arterial orifices, and especially in the aortic orifice; no one doubts this." This was the received site of the murmur by all, except Hunter, until quite recently. He, remarking that it cannot be heard below the nipple, and in the axilla, as is the case in aortic murmurs depending on organic disease, or generally on a level with the ascending aorta, the arch, was disposed to place it in the orifice of the pulmonary artery (*Guy's Hospital Reports*, 1851). Quite lately, Dr. Parrot has given excellent reasons for believing the site of the anæmic cardiac murmur to be in the right auriculo-ventricular orifice (*Archives Gén. de Méd.*, 1866); its maximum of intensity, he observes, is in the fourth intercostal space, near the sternum, corresponding with the right auriculo-ventricular orifice; and its line of propagation is in the course of the superior vena cava and not of the aorta; and he further assigns insufficiency of the tricuspid valve as the cause of the murmur.

The mechanism both of the cardiac and venous anæmic murmurs has generally been ascribed, (1.) To increased friction of blood, whose velocity is diminished, against the walls of the vessels; (2.) To increased celerity of the blood, and consequent loss of relation between the contents of the ventricles and the area of the openings leading from them; (3.) The passage of thin watery blood through tense veins (OGIER W.). Dr. Flint has suggested spasm of the papillary muscles, causing insufficiency of the mitral valve, as the cause of the cardiac murmur; and he believes it due to diminished arterial tension, and increased rapidity of the ventricular systole.

Whenever a proper examination can be made of the veins of the neck, particularly at the base, the seat of the two pulsations—and sometimes only one, synchronous with the beat of the radial artery—will be found to be limited to the course of the vein, to be from above downward, to follow a line which makes an acute angle with the artery, and to cease to be heard immediately on the least compression above the point of pulsation; all of which goes to prove that the pulsations are due to a reflux of blood into the right cavities of the heart—in fact a true venous murmur. Now Dr. Parrot has proved (*Archives Gén. de Méd.*, 1865), that the pulsations of the external jugular veins, double and single, provided they are synchronous with the radial pulse, are a sure sign of imperfect closure of the right auriculo-ventricular orifice by the tricuspid valve, at the moment of the ventricular systole. He has subsequently (*loc. cit.*) attempted to establish a relation between the venous murmurs and the cardiac murmur, which happen simultaneously, and he looks upon the latter as caused by the former, "for," he says, "of all the conditions which can give rise to intra-cardiac blowing, no one is more favorable than insufficiency of the auriculo-ventricular valves, since the blood is driven with force and rapidity by the ventricle, passes from a large cavity where it is subjected to great pressure, and through a narrow orifice into the auricle, a thin-walled sac, where the compressing force is feeble."

One constant urinary character attends both anæmia and chlorosis, namely, a diminution in the urine pigment, which is often reduced to one-fourth or one-sixth of its normal amount. Very general

there is also lessening of the free acidity; and urine which is pale and almost neutral during the whole of the twenty-four hours is almost as good an indication for the use of iron as the pallor of the skin itself. The amount of iron in the urine of anæmia is often very small. The quantity of urine may not be decreased, and may be in some cases large. Its specific gravity is low (PARKES, *l. c.*). With regard to other constituents, such as urea and uric acid, the accounts are very contradictory.

[Becquerel found the proportions of uric acid and urea to be much lessened in anæmic patients, whilst the quantity of urine was normal. According to the researches of Führer and Ludwig (*Arch. Gén. de Méd.*, t. vii, 1856) the production of urea is, to a certain extent, proportional to the quantity of red globules in the blood, and it is not diminished under the influence of diet unless these are decreased; they believe, as a demonstrated fact, that urea is a direct product of the transformation of the red corpuscles.]

**Causes.**—[Anæmia is met with at all periods of life. Infants are born anæmic, and during the first infancy it is not uncommon; at puberty it appears as chlorosis in the female; in adult life it is not infrequent in both sexes; and in old age it is very common.]

As an independent constitutional disease, anæmia may be said to owe its origin to three sets of conditions,—(1.) Copious loss of blood, such as by hemorrhage or venesection, or oft-repeated small losses of blood. (2.) From loss of other fluids of the body besides blood, especially of such as contain albuminous, fibrinous, mucinous, or caseinous substances, such as the excessive secretion of milk in protracted suckling, suppuration, profuse blennorrhœa, leucorrhœa, diarrhœa [spermatorrhœa]. (3.) From insufficient and improper nutriment, or from disturbances in the absorption and assimilation of food, and the process of sanguification; or from repeated temporary interruptions to oxygenation of the blood, as by imperfect ventilation of sleeping-rooms or coal mines.

[Inanition alone will not cause diminution of the red globules. Panum (*Virchow's Archiv für Path. u. Phys.*, Bd. xxix, 1864) shows that in animals starved to death, the solid constituents of the blood, and particularly the red globules, do not diminish; and it results from his experiments, that the weight of the blood-mass maintains its relation to the body-weight, so that an animal dying of starvation has relatively to its body-weight as much blood, and that blood as rich in globules, as when it was well fed.]

(4.) It may result from the co-operation of many influences; for example, excessive bodily and mental labor; continued excitement, pain, care, grief, hardships; many acute and chronic diseases, some of which augment the consumption of blood, while others impede its formation—this effect being produced by an acute disease under certain circumstances, when its invasion is intense and its duration prolonged, such as by *tuberculosis*, carcinomatous diseases, *diabetes*; from poisoning by malaria—the *malaria-chlorosis* of Vogel, or *paludal cachexia* of Martin, as in the “used-up” condition of our Bul-



garian troops, described by Dr. H. Mapleton in *Parliamentary* 247, for 1856, p. 253. "It is the most general of all the conditions incident to tropical invalids" (MARTIN). [(LA ROCHE, *Pneumonia* etc., including an *Inquiry into the Existence and Morbid Agency of Malaria*, 1854; HEUSINGER, *Malaria Chlorose*, 1852; POYET, *Chlorose et l'anémie Palustre*, Bull. de Therap., t. lxxv.)]

[Tailors and shoemakers are generally anæmic, as well as bakers, cooks, and firemen of steam-vessels, from long-continued exposure to high temperature. The female operatives in the silk-factories of France are mostly anæmic, for the same reason. Anæmia occurs as an effect of certain toxic substances, as lead, mercury, iodine, aniline, tobacco. It is also met with in rheumatism, exophthalmic goitre, and other diseases.]

**Treatment.**—The energies of the physician must be directed to discover and counteract the cause of the anæmia. Nutritious substances must be supplied for diet, in the shape of easily digested meat and broths. The purely tonic treatment, in the combination of air, exercise, and diet, must be carried out as far as practicable. A change of air is absolutely necessary, and generally also of climate. Iron is one of the best medicinal remedies. The astringent preparations are pre-eminently tonic; and are especially useful when anæmia is associated with or dependent upon inordinate discharges. The *solution of the perchloride of iron*, in the form of *tinctura ferri perchloridi*, in doses of ten to thirty minims in water, or *infusio quassia* or of *calumba* has properties in common with the numerous salts of iron, and is one of the most reliable preparations. If anæmia is associated with *diarrhæa*, or *menorrhagia*, or *leucorrhœa*, the *solution of the perntrate of iron* in similar doses is attended with benefit. A preparation which is new to the pharmacopœia—the *syrup of the phosphate of iron*—possesses the general properties of ferruginous compounds, and is of great service when the anæmia is associated with certain forms of *dyspepsia*, or with *amenorrhœa*. It invigorates and increases the powers of digestion, and may be given to the extent of one to three drachms for a dose in water. Another phosphatic preparation of very great value is that which has been devised by the late Dr. Easton, Professor of Materia Medica at the University of Glasgow. Although it is not in the pharmacopœia, and although its mode of preparation had not been published, Dr. Easton kindly sent it to me for publication in the previous editions of this work, yet the combination has become very popular throughout the country as a general tonic in anæmia and cachexia generally. As such, it has been largely used by my colleagues, Professors Maclean and Longmore, amongst the used-up cachectic and anæmic soldiers under treatment at the Royal Victoria Hospital, Netley; and I would add my testimony to its being a most valuable medicine in general practice. The following is the original formula devised by Dr. Easton for the preparation of the *phosphates of iron, quinine, and strychnia*, in the form of a syrup ("*Syrupus ferrici et strychnia phosphatum*"):

R. Ferri Sulph., ʒv; Sodæ Phosph., ʒvj;\* Quiniæ Sulph., grs. cxcii; Acid. Sulph. Dil. q. s.; Aquæ Ammoniæ, q. s.; Strychniæ, grs. vj; Acid. Phosph. Dil., ʒxiv; Sacchar. Alb., ʒxiv.

“Dissolve the sulphate of iron in one oz. boiling water, and the phosphate of soda in two oz. boiling water. Mix the solutions, and wash the precipitated phosphate of iron till the washings are tasteless. With sufficient diluted sulphuric acid, dissolve the sulphate of quinia in two oz. water. Precipitate the quinia with ammonia water, and carefully wash it. Dissolve the phosphate of iron and the quinia thus obtained, as also the strychnia, in the diluted phosphoric acid; then add the sugar, and dissolve the whole, and mix without heat. The above syrup contains about one grain phosphate of iron, one grain phosphate of quinia, and one thirty-second of a grain of phosphate of strychnia in each drachm. The dose might therefore be a teaspoonful three times a day.

“The amount of phosphate of quinia might be increased according to circumstances; and if eight grains of strychnia were employed in place of six, as in the above, the phosphate of strychnia would be in the proportion of the one-twenty-fourth of a grain in every fluid drachm of the syrup. I would scarcely venture on a much larger dose. In cases of delicate children, with pale countenances and deficient appetites, I have given, with great benefit, a combination of equal parts of the above syrup and of that prepared by Mr. Edward Parrish [or Mr. Blair, of Philadelphia], often called chemical food. To children between two and five years of age, the dose of this combination may be a teaspoonful three times daily.”

In some cases the *astringent* preparations of iron are not suitable, and are apt to irritate delicate stomachs, or those in whom any inflammatory local disease exists. For delicate females and children the *saccharated carbonate of iron* is a most valuable preparation, in the form of *mistura ferri composita*, to the extent of one to two ounces for a dose; or in the form of the *pilula ferri carbonatis*, in doses of from five to twenty grains in the twenty-four hours. The *citrate of iron and ammonia* is another remedy which possesses scarcely any astringency, and may often be given in cases of anæmia when the stomach will not bear more astringent preparations. Five to ten grains of this salt may be taken during the twenty-four hours. It is best taken during effervescence, prescribed in *solution of citric acid*, and not in *bicarbonate of potash solution*. If it is put into the latter, carbonic acid will be given off, and probably burst the bottle. *Tincture of orange-peel* is the best flavoring agent; but as the salt will not dissolve in the tincture alone, it is necessary to dissolve the salt in water first, and then add the tincture, otherwise the division into doses is impracticable (SQUIRE).

When it is desirable to continue the use of iron for a long time, as in the *anæmia* of neuralgic affections or *tic-douloureux*, or to give it in large doses, the *magnetic oxide of iron* is the best preparation to administer in doses of five to twenty grains twice or thrice a day in water. The *reduced iron*—the *ferrum reductum* of the British Phar-

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\* A little more phosphate of soda gives a better result—say ʒj.



macopœia—is also a remedy which does not possess the astric properties of the other preparations, and is one of the most powerful remedial agents in cases of anæmia. One to five grains may be given several times a day in powder or in pill. It has no taste. *One grain is equal medicinally to five grains of the citrate of iron and quinine*, either in solution or in the form of a pill, is a new and useful preparation, in doses of five to ten grains three times a day. Such a preparation of iron must be found which will not check the digestion of other food. If loss of appetite, feverishness prevail, the form of the remedy is probably unsuitable and requires to be changed. Chlorine, in the form of warm chloric acid baths, is highly spoken of by Dr. T. K. Chambers, as an agent of great value, and as a directly restorative medicine in anæmia (*l.c.*, p. 332) [and hydrochloric acid may be given internally]. The bowels are to be kept regular by four grains of the pills of iron and myrrh taken at bedtime.

Ferruginous remedies are always of use when we have reason to believe that there is a diminished energy in the formation of red blood cells; and if the urine be pale and almost neutral during the twenty-four hours, it is as good an indication for the use of iron as the pallor of the skin in cases of anæmia (PARKES).

[Dr. Trousseau has long held the opinion that in anæmia complicated with tubercles iron should not be given, as it tends, he thinks, to increase their development. Some observations of Millet would give countenance to this notion. At all events, the subject is worthy of investigation. Quinine and senecio will be found a valuable remedy in anæmia, and the writer has seen the chlorides of arsenic, iron, and quinine, with decided advantage.]

The respiration of pure oxygen has lately been employed by Dr. Boussier (quay) (*Essai de Pneumatologie*, Paris, 1866), particularly where there is obstinate anorexia, a common condition in post-puerperal anæmia. Trousseau, speaking of this method of treatment (*Clinique Médicale*, p. 63, 2ième ed., Paris, 1865), says, "In these cases, where all other means failed, you have seen the success of the inhalations of oxygen. Patients brought back to life by their use." Tobarié and Pravaz recommend the breathing for some time of compressed air: common salt is recommended by Plouvrej; grape-sugar is the usual remedy in Schleswig-Hanover (VON MAAK); and manganese, alone or combined with iron, has its advocates (HANNON, PETREQUIN, BURIN-DUBUISSON).]

#### GREEN SICKNESS—SYN., CHLOROSIS.

LATIN EQ., *Chlorosis*—Idem valet, *Pallor luteus fœminarum*; FRENCH EQ., *Chlorose*; GERMAN EQ., *Chlorose*—Syn., *Bleichsucht*; ITALIAN EQ., *Clorosi*.

**Definition.**—A deficiency of the blood-cells, with redundancy of the plasma part of the blood, occurring in young girls at the age of puberty, sometimes in the young of both sexes before the complete development of the distinctive characters of the sexes towards puberty has been effected.

**Pathology and Symptoms.**—A very indefinite idea is associated with the name and nature of this disease. Many employ the term precisely in the same sense as anæmia; or they limit the term

rosis to those forms of anæmia whose causes are unknown. The original use of the term is expressed in the first part of the definition, as limited to that form of anæmia which occurs in the female at the period of puberty. It is to be classed amongst the "*functional diseases of the female organs of generation in the unimpregnated state.*"

A change in the complexion constitutes the most striking symptom of chlorosis. A marked pallor of the skin prevails, sometimes perfectly pale, at other times yellowish, greenish, or waxen-colored. The lips and mucous membranes are also pale—symptoms which are the result of the deficiency of blood-cells, and so of blood-pigment. Slight dropsical swellings occur, such as œdema of the feet and ankles, the face and the eyelids, and a bluish halo sometimes encircles the orbit.

The temperature of the body is generally diminished—the breath is cool, the lips, nose, ears, hands, and feet are cold; and chilliness of the body prevails, which is morbidly sensitive to external cold, and seeks warmth. As in other forms of anæmia, the patient suffers from great prostration of strength and debility; she is tired by the slightest exertion, and the weakness sometimes becomes so great as to lead to fainting. The patient is languid, listless, sedentary, and indisposed to exertion. She is nervous, low-spirited, and frequently a prey to irregularities of temper.

*Hysteria* may prevail with chlorosis. The patient frequently suffers from vertigo, headache, often recurrent, *tinnitus aurium*, especially of the right ear, sparks in the eyes, tendency to fainting, neuralgia, spinal irritation, and convulsions, or a "heaviness for sleep." The mind is sometimes morbidly impressed with grief, while despondency and frightful dreams prevail; there is the apprehension of nightmare, melancholy thoughts predominate, mania may become confirmed, and the insanity may assume a suicidal tendency.

Disorders of digestion attend the disease. Appetite is diminished or perverted, or even depraved. Acids and highly flavored foods are craved for, and sometimes such substances as chalk, paper, ashes, coal, plaster of Paris, hair, earth, and even excrements, are desired to be eaten. Substances also very difficult of digestion are longed for, and are sometimes digested better than simpler kinds of food. Constipation frequently and obstinately coexists; but afterwards diarrhœa may alternate, and lead to *hæmatemesis* or *melæna*, when the evacuations are usually scanty, dark-colored, and fetid. A singular and peculiar pain of one or both sides, which suffer together or alternately, is a frequent symptom. It is referred generally to the region over the false ribs and the *ilia*. The recurrent nature, the particular situation, and the alternating character of the pain, are peculiar and characteristic. It is not aggravated by a deep inspiration, although it may seem to be so at first.

Other functions are no less disordered—for instance, the respiratory, the generative, and the circulatory.

Respiration is oppressed, or performed with difficulty, and the breath is offensive. Breathlessness prevails, and is experienced

especially on any exertion; sometimes also fits of dyspnoea sometimes a sonorous cough occurs. Menstruation is generally absent or performed imperfectly, irregularly, and with pain; the flow is thin and watery, or leucorrhœal. The condition of the menstrual discharge is generally very easily impaired. The catamenia become irregular in their return, inconstant, or of short duration in their flow, deficient in quantity, and pale in color, terminating in a state of leucorrhœa. In some cases, each of the catamenia is preceded and attended with much pain in the back and in the region of the uterus. Later in the disease there may be complete *amenorrhœa*.

The conditions of the vascular system are variable. The pulse is slow, feeble, and soft, but sometimes frequent, and always accelerated; the heart and great vessels are irritable; palpitation is common; or, more frequently, a sense of fluttering in the præcordia with irregular action of the heart, or imperfect syncope and murmurs, as already described, may prevail. It is to be observed, however, that, apart from mere lassitude and palpitation, patients are sometimes chlorotic for weeks, without any other marked symptoms; and the same complication of intercurrent acute disease is apt to happen as described under anæmia. The condition of the urine is similar to that which prevails in anæmia.

[The nervous system is always more or less compromised in chlorosis. The intellectual faculties, sensation, and muscular motion, both of the mind and organic life, are affected to some degree. Perversion of the senses is not infrequent; the temper is fretful, whimsical, and also irritable as to carry the patient to the verge of insanity; there is cutaneous anæsthesia, more rarely hyperæsthesia; neuralgia, especially facial, is very common, and occasionally there is gastralgia, or hepatic or enteralgia, or hysteralgia, and, more often, pleurodynia. Spasm of the voluntary muscles is not uncommon; cardiac palpitations, and general intestinal, and uterine colics happen. The several secretions are affected, showing nervous troubles in the stomach, liver, and kidneys. Broussais mentions the case of a young girl affected with chorea, but otherwise in good health, who, after a sudden and great fright, was attacked with chlorosis; and also another instance in a girl of eighteen years who suddenly became chlorotic, after violent mental emotion.]

**Causes.**—The conditions which bring about this constitutive affection are similar to those already mentioned. The disease is frequent in females between the ages of *sixteen* and *twenty-five* years, and is often of long duration, lasting for months, and even for years, with a tendency to relapse after cure.

**Diagnosis.**—It is especially necessary that the physician should not confound *chlorosis* with *jaundice* on the one hand, or *dyspepsia* on the other, or with incipient *pulmonary tuberculosis* or inflammation within the chest or abdomen.

**Treatment** is chiefly conducted by change of air and diet, and the use of medicines. All the functions of the body must be carefully observed, and regulated. The residence of the patient must be a reputed healthy spot in the pure air of the country, rather than

town. Dry frictions of the back and limbs are also recommended, with bathing in the sea where it can be borne.

The food must be regulated so as not to be too stimulating nor disgustingly bland;—frequent change is demanded, with a due attention to the proper apportioning of nutritive and respiratory elements. The diet should be generous, and carefully apportioned to the powers of digestion. Any symptoms of dyspeptic oppression, impaired digestion, or offensive stools, demand a careful readjustment of the dietary. Three meals, or perhaps four, may be taken during the day—the intervals between the meals being equal; and, half an hour before each of the meals, two grains of *capsicum*, with one grain of *quinine*, may be given in a pill. The breakfast should consist of biscuit, dry toast, or stale bread, with fresh butter and perhaps an egg, and one small cupful—not more—of hot black tea. Five or six hours after breakfast, and after the pill, a dinner of *well-done* meat, such as mutton-chops, may be eaten with potatoes; porter, beef-tea, or milk being taken for drink, according to taste and the powers of digestion. The evening meal may be similar to the morning. At first there may be a loathing of all kinds of food in the form of regular or “ordinary diets.” In such cases no meals should be prescribed, and no solid food; but a cup of milk, with a third part of lime-water in it, may be given every two hours, prescribed as a medicine, and a pint of beef-tea in divided doses may be taken as a drink during the day. This diet may be gradually added to as the appetite improves.

[All who have had much experience in the treatment of chlorosis, will recognize the truth of the following remarks of Dr. Trousseau, and the wisdom of his practice. “There are some chlorotic girls,” he observes, “who would rather die of inanition, than eat ordinary food. We should not hesitate in such cases to make those therapeutic concessions, which we are so often obliged to submit to in the practice of our art. I allow alimentary substances acknowledged to be very indigestible—caring not for the kind of food so that it is eaten—as radishes, salads, fruits hardly ripe, highly seasoned sausage-meat, old cheese, vegetables, meats prepared with vinegar, acid drinks, &c.; all that I require is, that in this whimsical diet there shall be variety. By these means we often succeed in awakening the digestive functions, bring back the sensation of hunger, and gradually lead the patient to proper food.”]

Change of air and bathing will also greatly aid in restoring health. If the patient lives in a town, she ought to be sent into the country; and, best of all, to the sea-side, where sea-bathing may be prescribed, if the strength of the patient is sufficient. Should the patient have been living in the country, a change of air and scene to a lively town will be no less useful as a remedial agent.

The much-be-quacked use of water, in its varied modes of application to particular cases, will be found a powerful restorative agent, if judiciously used by the patient under medical advice.

Of medicines, the preparations of iron have most reputation. They seem to act by promoting the formation of the red blood-corpuscles; and they operate best in those cases in which the blood is

rich in albumen. They also act as stimulants to digestion. From what has been stated at page 94, it is not altogether material which of the numerous *officinal* preparations of iron be prescribed. Almost every physician has some preparation of iron he fancies better than another; and some of them seem aided in their good effects by combination with *carbonate of iron*, such as the *sulphate of iron*, in doses of *three to five grains*, or *ferrisulphas exsiccata*, in doses of two to five grains, prescribed in pills. The use of ferruginous remedies must be persevered in for months, and the general indications for prescribing the preparations are the same as those given under anæmia, page 94. In pills iron preparations may also be combined with *extract of nuxvomica* or with *strychnia*, in suitable doses; or the syrup of the phosphate of iron, quinine, and strychnia may be given (see page 95); the eliminative action of the colon is to be promoted by four grains of the pill of aloes and myrrh taken every night at bedtime, or a grain of the *watery extract of Barbadoes aloes* with a little ginger as a pill. Simple bitter tonics are useful adjuncts to the chalybeate treatment, such as *gentian*, *calumba*, and the preparations of cinchona. They aid feeble digestion.

Where a high degree of serous plethora exists, and productive of violent excitement of the vascular system, palpitation of the heart, and congestion of the head, venesection may be practised. Phlebotomy only acts as a sedative, but aids the radical cure of the disease inasmuch as it causes the subsequently administered ferruginous preparations to be borne more easily. Both general and local bleedings may be used; but the blood must be taken in small quantities, a couple of ounces at a time being quite sufficient (Voss).

*Tartarated iron* (*Ferrum tartaratum*) is also a useful remedy. It may be prescribed with alkalies, in doses of six to twenty grains, dissolved in water.

#### LEUCOCYTHÆMIA—SYN., WHITE-CELL BLOOD, OR WHITE BLOOD.

LATIN EQ., *Leucocythæmia*; FRENCH EQ., *Leucocythémie*; GERMAN EQ., *Leucocythæmie*;  
—SYN., *Leucocythæmie*; ITALIAN EQ., *Leucocitemia*.

**Definition.**—A disease sui generis, in which the number of white corpuscles in the blood is greatly increased, with a simultaneous diminution of the red. This state is brought about by chronic exhausting diseases, exposure to cold and wet, or serious acute affections—such as typhus, pneumonia, puerperal fever, affections of the lymphatic glands, or spleen, and is attended sometimes by cough or diarrhœa, epistaxis, hæmorrhagic effusions, furunculous or pustulous eruptions.

**Pathology.**—In the present state of our knowledge regarding leucocythæmia, an account of the phenomena which attend its course is all that can be given.

Having conveyed in the previous editions of this work, a concise chronological account of the discovery of this remarkable disease, and of the steps through which the views regarding



pathology, as at present entertained, were successively reached, I am the more anxious now to do justice to those distinguished men whose conjoined investigations have enriched science with the knowledge of leucocythæmia which we now possess.

In the sixty-fourth volume of the *Edinburgh Medical and Surgical Journal* a case of disease of the spleen is described by Dr. Craigie, "in which death took place in consequence of purulent matter in the blood." The case occurred in 1841, and proved fatal on the 1st of April of that year. The late Dr. John Reid examined the case, and, "on examining the blood of the veins of the abdomen and sinuses of the brain by the microscope," found "that it contained globules of purulent matter and lymph." Dr. Craigie inferred "that, by some means or other, purulent matter and lymph had been mixed with the blood, and, circulating with it, had given rise to the peculiar febrile and inflammatory symptoms which occurred during life, and to death in the manner in which it had taken place." He inferred, that the spleen was the only organ from which the purulent matter and lymph could have proceeded, it having been for several weeks in a state of chronic inflammation; that in this form of disease of the spleen the pus-cells are secreted, and, being mixed with the blood, they cause much disorder in the sanguiferous system, and finally destroy the patient. Dr. Craigie thus clearly recognized a connection between the diseased state of the spleen and the changed condition of the blood; and that "it was in some respects new." On this account he made a correct description of it, expecting at some future period that the chief facts might be confirmed. He kept the case unpublished till 1845, and it was only published then in consequence of the occurrence, to another physician in the same hospital, of a case "in many, if not in all, respects similar, which led Dr. Craigie to anticipate similar results, and which went far, as he thought, to confirm his conclusions deduced from the first case" (*Edinburgh Medical and Surgical Journal* for 1845, p. 400, *et seq.*). The details of the second case referred to by Dr. Craigie were published by Dr. Bennett in the same volume of the *Edinburgh Medical and Surgical Journal* immediately after the account of Dr. Craigie's case. Dr. Bennett describes his case as one of "*Hypertrophy of the Spleen and Liver, in which death took place from suppuration of the Blood;*" and although the most evident lesion during life was enlargement of the spleen, Dr. Bennett agrees with Dr. Craigie "in thinking that the immediate cause of death was owing to the presence of purulent matter in the blood, notwithstanding the absence of any recent inflammation or collection of pus in the tissues," and that it produced the febrile symptoms. In these valuable papers Drs. Bennett and Craigie are at issue, however, about the source of the pus in the blood. Dr. Bennett considered his case particularly valuable, because he believed it demonstrated "the existence of *true pus*, formed universally within the vascular system, independent of any local purulent collection from which it could be derived" (*l. c.*, p. 414). He believed the white corpuscles he saw in the blood "were true pus-globules, and he then was of opinion that they were formed in the *liquor sanguinis*

within the vessels, independent of inflammation or of phlebitis, what was then understood by *pyæmia*; that the transformation had taken place throughout the system, and that the whole of blood was affected: and the case appeared to him capital in this respect, of furnishing an important fact which may serve to throw light on the doctrine of *Zymosis*, as applied in Pathology (*l. c.*, p. 423).

In the same year (1845), about a month after the publication of Dr. Bennett's case, Professor Virchow, of Berlin, described a case explained in *Froriep's Journal*, of November, 1845, a disease of the blood as due to an increased development of white blood-cells. No inflammation of veins was observed; and the hypothesis of the spontaneous formation of pus in the blood (*pyæmia*) was contrary to the pathological doctrines of Virchow. To this form of disease he gave the name of "white" or "colorless blood." As to its origin, he distinctly points out as *primary* the condition of the spleen, maintaining the idea of an increased formation of colorless blood through the functional relations of that organ.

In 1845-46 several cases of leucocythæmia during life were recognized in this country. Dr. Fuller, of St. George's Hospital, in December, 1845, was the first to determine this condition of life; and it was also diagnosed by Drs. T. K. Chambers, and W. Wilson in London, and by Dr. Douglas, in Edinburgh, in 1846.

Virchow continued to oppose the view of Dr. Bennett as a secondary affection having its origin in the formation of pus in the blood, maintaining that its formation is in any way of the nature of a *Zymosis*; and, on the contrary, the correctness of his first opinion, that the essence of the disease consisted in an increase of the colorless blood-cells (*Medicinische Zeitung*, August and September, 1846).

In 1847 Virchow collected and described cases from the German authors, and compared them with those observed in England, and those seen by himself; and finding also a further basis in fact for his views in the theory of Hewson and Donné regarding the action of the spleen in the propagation of the blood, he not only became more confirmed in his opinion, that from some alteration of the spleen to the blood-cells might be established the conditions under which "white blood" would be produced.

In the *Archiv. of Path. Anatomy* of the same year a case of great value was described by him as a new form of this "white-blood" disease, in which this form the spleen presented no change whatever, while the lymphatic glands were enormously enlarged. He was now enabled to declare with more confidence that the "white-blood" disease proceeded from a primary affection of the spleen and lymphatic glands, as a direct consequence of which an increased development of colorless blood-cells takes place; and thus he considers that the disease in its turn, throws light on the physiological functions of the spleen and glands as eliminating organs of the blood.

In 1851-52 Dr. Bennett gave a most interesting and systematic view of the whole subject, first in the *Monthly Journal* of 1851, and afterwards in a separate work, entitled *Leucocythæmia, or W*



*Blood*, in which the explanation of the pathology of this disease is no longer referred to the spontaneous development of pus in the blood.

Although Dr. Bennett originally set out upon what has since been believed to be an erroneous track in the interpretation of the phenomena of leucocythæmia, yet he was the first to show that these phenomena were new to science, while the more apparently correct generalization and elaboration of the subject seems, from the chronological evolution of the history of the disease, to be mainly due to Virchow. He was the first to recognize and regard the colorless cells in leucæmia or leucocythæmia to be identical with the colorless globules of the blood—the view at present generally entertained. More recent observation, pointing to the formation of pus from the white corpuscles of the blood, may (if found correct) again change the views now generally entertained. It is greatly to be regretted, however, that so much personality and ill feeling has been imported into the historical records of this interesting discovery; and if the reader desires to read the several sides of the lengthened controversy, let him consult (1.) The original cases of Craigie and Bennett in the sixty-fourth volume of the *Edin. Med. and Surg. Journal*; (2.) A review of Virchow's *Handbook of Pathology*, in the June number of the *Edin. Monthly Med. Journal* for 1854, p. 546; (3.) An able letter by Professor Kölliker, in the October number of the same journal, p. 374; (4.) A Reply by Dr. Bennett to the same, p. 377; (5.) Dr. Bennett's work on *Leucocythæmia*; (6.) Dr. Bennett's more recent Lecture (vi) in the *Lancet* of April 4, 1863.

The morbid state expressed in the definition has been observed by many physicians and pathologists under various complex conditions of the disease; and hence a variety of opinions have been entertained regarding it in Germany, England, and France, by Vogel, Remak, Henle, Nasse, Weber, Rokitansky, Kölliker, Parkes, Jeuner, Gulliver, Piorry, Bichat, Velpeau, and others, who have described cases since Bennett and Virchow first wrote on the subject.

The increase of the colorless corpuscles of the blood, which is the prominent character of this disease, does not seem in any case to have existed or occurred by itself. Other morbid states precede, coexist, or succeed the augmentation of the colorless corpuscles.

The most frequent complication consists in the enlargement of the spleen. This enlargement is so constant that its existence, if not otherwise accounted for, would at once indicate that leucocythæmia prevailed, and would suggest a microscopic examination of the blood. In nineteen cases, Vogel writes that the splenic enlargement was present in sixteen; and in three cases the weight of the spleen was estimated at more than seven pounds.

Constituents foreign to normal blood have been found by Scherer in a qualitative analysis of the blood in a case of leucocythæmia, where the spleen was enlarged. These consisted of lactic, acetic, and formic acids, gelatin, and a peculiar substance (hypoxanthin) to the amount of from .4 to .6 per cent. Scherer finds, also, that this

same substance exists naturally in the pulp of the healthy spleen. This form of the disease has been named *splenæmia* by Virchow. In it the globules (white) predominate which are peculiar to the elements of the spleen.

The *liver* is also frequently enlarged in this disease, but not to a remarkable degree as the spleen. Vogel writes that as often as thirteen times out of nineteen cases it was either enlarged or otherwise morbidly altered.

Affections of the *lymphatic glands* predominate in some cases rather than enlargements of the *liver* or the *spleen*. According to Vogel, such have been observed eleven times out of nineteen cases. Virchow considers that some kind of lymphatic *diathesis* prevails, so that there is a progressive inclination of the lymphatic system to the formation of the lymphatic elements. In some instances observed by him there seems to have been a new formation of glandular tissue, or that the glandular tissue tended to grow beyond the pre-existing boundaries of the glands. He has observed this development of lymphatic gland tissue to take place in the liver in a remarkable case which contained numerous small whitish granules about the size of the natural lobules of the liver, and which exhibited under the microscope nuclear and cellular elements quite similar to those of the lymphatic glands. This infiltration of the liver followed the ramifications of the portal vein. In one of these cases he observed a similar alteration in the kidney.

This constitutes the *lymphatic* form of leuchæmia, or *lymphæmia*, first described by Virchow in 1847.

In such cases the elements of the lymphatic glands prevail in the blood, which is then characterized by innumerable round granulated nuclei, generally provided with nucleoli, of the size of the usual nuclei of the lymphatic glands. Here and there are also to be seen cells consisting of such a nucleus surrounded by a membrane closely attached to it.

There are three possible conditions given by Virchow under which these elementary cells in the blood may originate,—(1.) They multiply in the blood by the subdivision of pre-existing cells. (2.) They may be primarily introduced into the blood through the lymph or chyle, which are conceived to convey the developed as well as the undeveloped globules derived from the lymphatic glands, the spleen and its connecting tissue; (3.) That they are formed on and detached from the walls of the bloodvessels has not been yet proved.

Virchow regards the colorless blood-corpuscles as simple cells without any specific character, whose transformation into red blood corpuscles cannot take place; that they therefore form a relatively superfluous constituent of the blood,—a kind of excess or waste. The transformation of lymph-globules into red globules takes place before passing into the general circulation; and it appears that a certain cell, when passing into the blood, has gone beyond its stage of development, it is ever afterwards unfit to undergo any specific colored metamorphoses. The idea that the cells are of a purulent nature has been now abandoned; and there is no evidence to support the doctrine that they are the result of *pyæmia*. Be-

the spleen and the lymphatic glands, the other blood-glands, such as the *thyroid gland* and *supra-renal capsules*, are occasionally degenerated, as well as Peyer's glands and the mesenteric glands; and leucocythæmia may arise from disease in them as well as from the spleen or lymph-glands.

The statistics regarding the ages at which the disease has been observed are given by Vogel as follow: One case was observed under ten years of age, two between ten and twenty years, three between twenty and thirty years, seven between thirty and forty years, four between forty and fifty years, three between fifty and sixty years, and three between sixty and seventy years.

**Symptoms.**—In the majority of cases there are obvious indications of general ill-health; and the most prominent symptom has been tumefaction of the abdomen, depending upon an enlarged spleen and liver. Ascites and anasarca of the lower half of the body are not unfrequently present; and a tendency to œdema may commonly be observed, the general surface of the body being usually pale. Transitory pains are frequently experienced in the abdomen.

Intestinal disorders are often also present, such as vomiting, constipation, or diarrhœa, and jaundice is not unfrequent; but diarrhœa is one of the most dangerous complications, and the most difficult to arrest or control.

A considerable amount of dyspnœa may prevail, which cannot be accounted for by elevation of the diaphragm merely. Hemorrhage often occurs in the form of epistaxis, or takes place from the gums. A persistent increased secretion of uric acid has also been observed in the urine. The disease generally runs a chronic course, and a high degree of emaciation ordinarily accompanies it. As in the other forms of anæmia, leucocythæmia is usually well established before it is noticed, and before any remarkable disturbances in the general health have occurred. It is not till towards the fatal termination that any fever sets in, which then assumes the hectic type.

**Diagnosis.**—The diagnosis of the disease consists in demonstrating the extreme increase of the colorless blood-cells, which may be done as follows:

1. By microscopic examination of the blood, for which a single drop is sufficient, most conveniently taken from a needle puncture in the finger; and examined under a power of at least 250 diameters. If the disease exists, the colorless corpuscles will be seen to form a sixth, a fourth, or even a half or more of the numbers of the red.

2. If a large quantity of blood is obtainable by venesection or by the cupping-glasses, and freed from fibrine by heating, and placed in a high narrow little glass, so that the corpuscles sink to the bottom, the upper part of the mass looks whitish colored, like milk. The milky character does not vanish on agitation with ether, and is not produced by fat-globules suspended in the blood-serum, but by the prodigious number of the colorless blood-corpuscles.

3. The clot of leucocythæmic blood shows on its surface grayish-white granulations, which, being observed under the microscope,

are seen to consist almost entirely of colorless corpuscles, distinguishing the condition in leucocythæmia from the ordinary leucæmia; and the separated serum being clear, and not turbid, distinguishes the condition from a fatty condition of the blood.

4. In the dead subject there are found in the heart and in the great veins large, soft; semi-fluid grayish-yellow coagula, which, on microscopic examination, are seen to consist almost entirely of colorless corpuscles (VOGEL).

**Causes.**—The causes which bring about leucocythæmia are entirely unknown; but it seems several times to have suggested itself to Virchow that acute inflammatory processes may lay the foundation of the morbid state; and in an interesting review of the writings of Virchow in *The Brit. and For. Med.-Chir. Review* July, 1857, there is related a case of the lymphatic form of the disease, whose origin obviously dates from inflammatory swelling of the lymphatic glands after exposure to cold and wet. In a similar case in Guy's Hospital, in July, 1863. It occurred to a man after exposure to cold and wet, on Epsom Downs, at the time of the races there.

**Prognosis.**—Hitherto no case of cure is known, and the disease may continue for a lengthened period without a fatal result.

**Treatment.**—The most varied remedies have been tried, with a view to checking the increased formation of colorless corpuscles; but it is suggested that if it is possible to discover the glandular or systemic affection early, before the alteration of the blood has made much progress, it is probable that the disease may be averted. Tonics, nutrients, and stimulants are indicated, to support the system. The use of the nitro-muriatic bath ought not to be neglected. The indications given under anæmia and chlorosis may be followed.

#### ADDISON'S DISEASE—SYN., BRONZE SKIN.

**LATIN** Eq., *Morbus Addisonii*—Idem valet, *Cutis ærea*, *Melasma Addisonii*; **FR.** Eq., *Maladie d'Addison*; **GERMAN** Eq., *Addison'sche Krankheit*; **ITALIAN** Eq., *Malattia dell'Addison*.

**Definition.**—A morbid state which establishes itself with extreme insidiousness, whose characteristic features are anæmia, general languor, debility, and extreme prostration, expressed by loss of muscular power, weakness of pulse, remarkable feebleness of the heart's action, breathlessness upon slight exertion, dimness of sight, functional weakness and indigestibility of the stomach, and a peculiar uniform discoloration of the skin which becomes of a brownish olive-green hue, like that of a mulatto, occurring in connection with a certain diseased condition of the supra-renal capsules. The progress of the disease is very slow, extending on an average over a year and a half; but may be prolonged over four or five. The termination to death is by asthenia, the heart becoming utterly powerless, as if the natural stimulus—the blood—had ceased to act.

**Pathology.**—The pathological significance of morbid states of the supra-renal capsules was brought prominently before the profession both in this country and on the Continent, by the original cases of

vations of the late Dr. Addison, then the senior physician of Guy's Hospital. The cases recorded in the medical journals since Dr. Addison wrote, which connect uniform discoloration of the skin (a condition now known by the name of "bronzing") with various morbid states of the *supra-renal* bodies, are now so numerous that, as a clinical fact, the connection cannot be disputed; but the exact relationship and pathological significance of the morbid states thus connected are still open questions, especially as regards the pathology of the constitutional cachexia which exists. Morbid states of the *supra-renal* capsules are not always attended with bronzing of the skin. It appears, indeed, if the cases recorded are carefully analyzed, that symptoms and phenomena of a very important kind have been lost sight of in describing this constitutional disease, while an undue importance has been placed upon the bronzing of the skin. It is to the cachexia that Dr. Addison calls special attention; but his commentators have been carried away by the inquiry regarding the color of the skin and its connection with the capsular disease. These writers have overlooked the more important portion of his observations, and have been induced to consider the *causes* and *nature* of the bronzing of the skin, which, being established, may be received as a most valuable symptom of a prevailing constitutional cachexia, in which the abnormal deposit of pigment is associated with anæmia and intense prostration, with the phenomena stated in the definition. There can be no doubt that the bronzing must appear of very secondary importance compared with the symptoms and pathology of that peculiar cachexia which attends the cases of *supra-renal* capsular disease, as described by Drs. Addison and Wilks. There seems to be no doubt that the cases Dr. Addison describes belong to the class of diseases now under consideration; and that they are similar in many respects to forms of anæmia already noticed, and more especially to leucocythæmia. "In almost all the cases," writes Mr. Hutchinson, "there would seem to have been great deprivation of the colored constituents of the blood, as manifested by the pallor of those parts of the skin not involved in the bronzing, the great flabbiness of the muscles, and the pearly state of the conjunctiva." By a strange oversight, however, it would appear that the blood has been subjected to but few examinations. "In two only" of the cases, writes the same author, "was the blood examined, and in both of them it was found to be loaded with white corpuscles." In all the cases a most remarkable and fatal cachexia prevails, and the value of Dr. Addison's observations consists in showing that a peculiar bronzing of the skin, combined with *asthenia*—of which emaciation is not a necessary accompaniment—attends this cachexia, and indicates organic disease of the *supra-renal* capsules associated with this constitutional state. His observations are at the same time of the greatest value, as showing how well-directed pathological inquiry may advance the science of physiology, for the cases detailed throw some light on the influence, at least, of the *supra-renal* capsules in the maintenance of health.

Although the change of color of the skin would seem to be a



most marked and constant symptom, still it does not appear to have been stated by a reviewer of Dr. Addison's work, that the change "is one of the earliest symptoms of the disease." On the contrary, there are good grounds for believing, both from the history of the cases and from physiological experiment and observation, that the change in the color of the skin which has been termed "*bronzing*" does not come on for a long time—that from its nature as a pigmentary change it is of slow production; that while in almost all the well-marked cases in which it has occurred the symptoms had existed from one to three years, in other cases where it did not exist it has been alleged that time was not afforded between the establishment of lesions in the supra-renal bodies and the fatal issue for the production of the pigmentary deposit. The change of color seems to depend on the chronicity of the disease, so that, if the disease progresses rapidly, no discoloration is observed, the phenomena being simply those of asthenia. One very important point is thus remarkably deficient of illustration—namely, the symptoms of this cachexia independent of *bronzing of the skin*. Whatever has been shown relative to the disease, it would appear that when *bronzing* of the skin has been established, a sign of diagnosis has been discovered when it is too late to be of any service, for the cases appear to have terminated fatally in which this state was unequivocally established.

In malarious, malignant, and cachectic diseases it is not unusual, but rather the rule, for the serum of the blood to assume a yellow and dirty hue, and that ultimately the cutaneous surface comes closely to approach the color of jaundice, differing from it only in being more lurid and dusky; and it is believed that the hue of the skin, which becomes of so dark a tinge in some malignant or cachectic diseases, is due to the admixture of morbid matters absorbed at the seat of local mischief, and which so tinges the serum of the blood that the *rete mucosum* is rendered dark. If, then, it is true that in this cachexia the supra-renal capsules are always diseased, and if it be true, as M. Vulpian has found (*Med. Times*, October 1856), that the *supra-renal capsules* differ from all other organs of the body, in the presence of a substance which has two peculiar reactions, one with perchloride of iron and the other with tincture of iodine, the first of which gives a dark-blue tint, it is not probable that the coloration of the skin in *supra-renal* diseases may result partly from—(1.) The cachectic state; (2.) Organic lesion of the *supra-renal capsules*; and (3.) The reaction of the peculiar substance in these bodies upon the iron of the blood, which the morbid organic changes in them have allowed to mix with the circulating fluid? It is, unfortunately, the *supra-renal capsules* only which have been carefully examined in the cases recorded; and in connection with the cachexia and the "*bronzing*" the lesions they exhibit have been described as of a very varied character, consisting of (a.) Acute or recent inflammation ending in abscess; (b.) Atrophy with concretions; (c.) The conversion of the organs into indurated fibroid enlargements; (d.) Tubercular deposition in various stages; (e.) Carcinomatous deposition.



[According to the experiments of Dr. Brown-Séquard: (1.) Sensibility is highly developed in the supra-renal capsules; (2.) Contrary to the generally received opinion that, fully developed in the foetus, there is gradual atrophy after birth, they really gain in weight and volume from birth to adult age, so that they must not be regarded as the vestiges of an organ of embryonic life; (3.) After the extirpation of both supra-renal capsules animals die as certainly, and even more rapidly, than after extirpation of both kidneys. In 60 animals the mean time of death after extirpation of the capsules was 11½ hours; (4.) When only one capsule is removed the animal never lives beyond 17 hours; (5.) On the extirpation of both capsules a nearly constant series of phenomena happens—great debility; the respiration at first quickened becomes slackened, broken, irregular; the heart's beats are accelerated; and as death nears, nervous symptoms, as dizziness, convulsions, coma, occur; (6.) One capsule only being removed, the same symptoms take place, only more slowly, and after a time when the animal seems to have gotten over the effects of the operation. Dr. Brown-Séquard believes, as the results of his observations, that the supra-renal capsules are organs essential to the well-being of the economy, and that their degeneration, destruction, or removal is prejudicial to life itself.]

A definite lesion, however, has been recently pointed out by Dr. Wilks, which will be referred to presently.

Sometimes the lesion appears to have been secondary to morbid conditions, apparently of a similar nature, in other parts, all of which must therefore be interpreted as several local expressions of the constitutional disease which prevails; and occasionally the capsules seem to have been the only structures in which lesions were detected; while the degree of "bronzing" of the skin appears to have been proportionate to the length of time which the *supra-renal capsules* are presumed to have been diseased; but neither the time nor the number of cases in which such a proportion can be traced is accurately made out. The general symptoms are those of a person constitutionally diseased, *not* always proportionate in their severity either to the nature or to the extent of the disease in the *supra-renal capsules*. The post-mortem appearances seem to have been in some instances associated with the tuberculous diathesis; but in the best-marked cases there has been no appearance of tubercle on any part of the body (WILKS); and in no case of general tuberculosis has Dr. Wilks ever met with an instance where the *supra-renal capsules* were affected in the manner of Addison's disease.\* In some cases the spleen was much enlarged, the kidneys pale, and in the last stage of fatty degeneration (TAYLOR). Out of 500 cases of post-mortem examinations of all kinds of cases, made at Guy's Hospital by Dr. Wilks, only two instances were observed in which, the *supra-renal capsules* being morbid, the skin did not betray the lesion by "bronzing."† In some cases the mesenteric glands are

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\* [Dr. Greenhow believes that there is some intimate connection between the disease and the tubercular diathesis; and that the origin of the lesion of the capsules is due to the extension of inflammation to these organs from diseased, or injured, adjacent parts, in persons of a tubercular habit (*On Addison's Disease*, 1857).]

† [In a case of Dr. G. O. Rees, where the autopsy showed the characteristic lesion, there was no pigmentation, except a small patch on the inside of the lower lip (*Med. Times and Gaz.*, Sept. 22, 1866).]

stated to have been enlarged ; and calcareous concretions have noticed in the *medulla oblongata*.

Gastro-intestinal disturbance prevails during life ; and a cond of mucous membrane is found after death which may be assoc in its pathology with this irritation. The stomach is often e mosed, and the glands of the intestines enlarged. In several Brunner's glands in the duodenum, and the solitary glands i lower end of the ileum and in the colon, were very prominent

In one case which, through the kindness of my first teacher late Dr. William Monro, of Dundee, I had an opportunity of fully inspecting after death, in June, 1856, the following l existed : "The body generally was anæmic. The dark colori the skin was most conspicuous in the vicinity of the knees, a the lateral and posterior regions of the neck. The deposits c ment gave to the parts a dirty sordes-like appearance wher mucous membrane of the skin meets the lips, and especially a angles of the mouth. The heart was small and flabby. Tul lous deposits were sparingly disseminated throughout the api both lungs. The spleen, the liver, and the kidneys were sev adherent to the adjacent parts, but their structures appeared nc The supra-renal capsules were morbid, and the sympathetic r from the lesser *splanchnic* were greatly increased in size, as the ganglia of the *solar plexus* towards the side of the organ diseased and in contact with it. The texture of the nervous was of a bright rosy hue, as if under the influence of vascul citement. The mucous membrane of the mouth was thin and bloodless, the labial and buccal glands shining promi through. The stomach and glandular substance of the inte tube were uniformly thin throughout. In the stomach the so gastric glands were remarkably prominent, while the mucous brane generally was wasted and atrophic. Microscopic se from the jejunum and ileum showed the villi remarkably atten and the mucous membrane very readily separated from the cent muscular part of the gut. The tubular glands of the m membrane of various parts examined were almost entirely and their place supplied by granular amorphous material. average specific gravity of the mucous membrane of the inte was 1.040."

There are good grounds for believing that Addison himse tertained the belief that death in such cases may be due t implication eventually of the ganglionic nerves. Some of the toms point to this ; and of the special phenomena associated the cachexia, it may be said that the nervous centres are a impaired.

[Dr. Habershon (*Guy's Hospital Reports*, 3d series, vol. x, 1864 opinion that the more fully the disease is known, the more com will it be traced to the sympathetic nerve, and he adduces some observations to show the intimate connection between the supr capsules and the vaso-motor nerves, especially those derived fro semilunar ganglion. A branch of the pneumogastric nerve may a

traced to the supra-renal capsule, and the irritability of stomach is probably due to this cause. In cases of supra-renal degeneration, the relations and conditions of the nerve-structures should always be examined. Mattei believes that the symptoms are due to an implication of the sympathetic nerve. Dr. Wilks says, in his last very valuable paper upon this disease: "I have always believed that the solution of the question was to be found in the implication of the vaso-motor nerves, for the symptoms correspond with those which prevail when these centres are evidently involved; and, moreover, it is clear, that the symptoms are not due to the disease of the structure of the capsules, seeing that these are totally destroyed for months or years before the death of the patient."

Martineau, in his late work on this affection (*De la Maladie d'Addison*, Paris, 1864), enters very fully into the nervous distribution of the capsules. His chapter is thus epitomized by Dr. Wilks (*loc. cit.*). The supra-renal capsules receive so many nerves, that some authors have considered that these organs are a kind of nervous ganglion. The fact of the existence of the nerve-cells in their interior appeared to be a positive proof of the justice of this view, but the researches of Nagel, Bardeleben, Ecker, and Frey show that these little organs are glands. It is true that they receive many more nerves than any other organ in the mammalia, but in the other classes of the Vertebrata they receive but a small share. Besides, the glandular vesicles found in all classes is sufficient to make them rank amongst vascular glands. The discovery of M. Vulpian has contributed to this view. He has shown that the blood issuing from the supra-renal vein has peculiar properties, giving a characteristic chemical reaction, and a peculiar substance, which is formed in the substance of the organ and is poured into the blood, its reaction with the chloride of iron giving a bluish tint. But at the same time, while they are vascular glands, the capsules contain nerve-cells. Besides, Kölliker thinks that the two substances which compose these organs possess different functions. He ranks the cortical substance in the class of vascular glands, and assigns it a part in the function of secretion, whilst with regard to the great nervous richness of the medullary substance, he considers it as an apparatus forming part of the nervous system. Brown-Séquard has established, after Frey, Ecker, and Kölliker, that the peculiar cells of the gland resemble nerve-cells. According to Pappenheim and Remak, the nerves of the supra-renal capsules are composed only of embryonic fibres. On the contrary, Kölliker maintains that he has seen only true nervous tubes, and no trace of the fibres of Remak. Brown-Séquard has shown that in some animals there exist the fibres of Remak; he has seldom found fibres of double outline; very fine nervous fibres (sympathetic fibres of Bidder and Volkmann) abound in them. This author inquires, if the nerves of the capsules are endowed with sensibility, or if they are solely nerves for centrifugal action (vascular or glandular nerves)? He has shown that in rabbits and cats there is very marked sensibility; in dogs and fowls less; still, in all animals the capsules are incontestably the most sensitive of all the abdominal viscera. The nerves of the capsules proceed for the most part from the semilunar ganglion and solar plexus; but some of their branches arise from the pneumogastric and phrenic nerves.]

It has occurred to me also, that, in cases of this disease, all the viscera ought to be examined as to the reaction of their minute bloodvessels with iodine, as there are some phenomena which seem to indicate the not improbable coexistence of *amyloid* degeneration;

and the lesion in the *supra-renal capsule* ought especially to be investigated in this direction.

The state of the skin, which has been termed "bronzing," strongly resembles the color of a mulatto, or of a bronzed statue from which the gloss has been rubbed off. It has been examined microscopically by Dr. Wilks, Mr. Tuffen West, and M. Robin, of Paris. The sections show a layer of very distinct pigment-granules in the *mucosum*, limited to that structure, and exactly resembling that of a negro. The pigment is deposited in granules, but in some instances colored cells are visible.

Before detailing the symptoms observed in the remarkable cachexia from which these lesions result, it is necessary again to draw attention to our very incomplete knowledge regarding many points in its pathology, and especially regarding the state of the liver and the morbid tendencies of the relatives and progenitors of the patient. While the apparent connection of bronzing of the skin with lesions in the *supra-renal capsules* appears to be evident, the information is of the most deficient kind regarding the chronological sequence of the various symptoms which indicate the establishment of the constitutional disease.

**Morbid Anatomy.**—The special disease of the capsules in Addison's disease is described by Dr. Wilks as being of one form only, namely, that which was seen in the earlier cases described by Addison, and which he simply styled as "scrofulous." Judging from the material alone, it would be difficult to form an opinion as to its nature, seeing that a degenerating inflammatory substance can produce a very similar appearance to a so-called scrofulous one. It is only in exceptional cases that any well-marked tubercular deposit has been found in other viscera. "When the disease is recent, the organ is somewhat enlarged, and changed into a material which is semi-translucent, of a gray color, softish, homogeneous, and when examined microscopically, is found to be without structure or sometimes slightly fibrillated, or containing a few abortive tubercles or cells. This lardaceous kind of material is the first deposit, and resembles what is often seen in the early stages of scrofulous enlargement of the lymphatic glands; subsequently it undergoes decay or degeneration, as in these glands, and changes into an opaque yellowish substance; and thus the two materials are constantly found associated." At a later period, as in a scrofulous gland, this may soften into a putty-like matter, "or it may dry up, leaving the mineral part as a chalky deposit, scattered through the organ." These, then, are the changes—first, the deposition of a translucent, softish, homogeneous substance; subsequently, the degeneration of this into a yellowish-white opaque matter; and afterwards a softening into a so-called abscess, or drying up into a chalky mass. Occasionally, also, some fibrous tissue may be found round the capsule, being the product of an inflammation which has united the capsule to the kidney, liver, and adjacent parts." (WILKS.) Some years are necessary for the production of these changes.

[In his most recent contribution to the subject (*Guy's Hospital*

ports, vol. xi, 1865), Dr. Wilks states: "Now, after some years' attention to the subject, I repeat, with much confidence, that the disease of the capsules in morbus Addisonii is uniform and peculiar; that none other than this has yet been found in connection with the symptoms which Addison described, and none other which has been satisfactorily proved to be the sole cause of death. In all the examples which we have now in our museum, amounting to thirty-three, the disease is of the same nature in all, and this has been the case in all the other instances which have come before my notice at the Pathological Society and elsewhere. Also in the description given in all the cases which I have read in foreign journals, as occurring in various parts of Europe, the same form of disease has been present." This peculiar disease, with its effused material, undergoing subsequent changes and degenerations, has just been described. Though the amorphous yellow, cheesy substance, into which the organ is converted, has been styled "scrofulous" by Addison and others, and "tuberculous" by some, there are really no histological grounds to believe its nature to be so; the previous exudation, of which this material is a transformation or degeneration, is a translucent, tough, fibrous tissue, which, as Dr. Wilks observes, "might be styled, according to our present loose phraseology, a low inflammatory product."

Dr. Greenhow collected 196 reported cases of Addison's disease, and found that in 127 the supra-renal capsules had undergone the characteristic morbid change. In the other 69 cases the capsules were either healthy or had undergone cancerous, or other degeneration; or the change in them was too obscurely described to enable him to determine its nature. With a few explainable exceptions, none of these 69 cases presented either the constitutional symptoms or the peculiar discoloration of Addison's disease. On the contrary, among the 127 genuine cases, 95 presented either well-marked constitutional symptoms or characteristic discoloration of the skin, and 72 of these presented both. In 20 cases some of the constitutional symptoms or some discoloration of skin existed, and in many instances both, leaving only 12 out of 127 cases which presented neither any characteristic symptoms nor any discoloration of skin. These 12 cases were all among those complicated with advanced tuberculosis or with other serious organic disease, which appeared to be the cause of death, rather than the coexisting disease in the supra-renal capsules. On the other hand, only 5 of the 95 presenting characteristic symptoms or discoloration were found among the number complicated with serious non-tubercular diseases, and not one among those complicated with advanced phthisis. Dr. Greenhow's analysis of the 127 genuine cases showed that, while 46 cases were reported as uncomplicated, and 16 complicated with other serious disorders without tubercle, no less than 65, or an absolute majority of the whole number, were complicated with tubercular disease of the lungs or other organs, in all gradations of development, and hence the conclusion of a very intimate relation between Addison's disease and the tubercular diathesis (*Ed. Med. Jour.*, June, 1866).]

**Symptoms.**—As in the forms of anæmia already described, so in this disease, the patient has considerable difficulty in assigning the number of weeks, or even months, that may have elapsed since he first experienced indications of failing health and strength; and the rapidity with which the local lesions seem to develop themselves varies in different cases—a few weeks being sometimes sufficient



to break up the powers of the constitution, or even to destroy it. Dr. Addison believed that this event is the more speedy in proportion to the rapid and extensive destruction of the *supra-renal* gland. The important features of the disease, as set forth by Dr. Addison—are—a progressive feebleness of the patient, without any apparent or known cause (*asthenia*), anæmia, general languor and debility, a remarkable feebleness of the heart's action, irritability and nervousness of the stomach, and a peculiar change of color of the skin. In most of the cases the early sequence of symptoms appears to have been gradual and almost imperceptible indications of failing life and strength, consisting chiefly of languor and weakness, and inability to either bodily or mental exertion, the appetite being impaired or entirely lost, the white of the eyes becoming pearl-colored, the pulse small and feeble, perhaps somewhat large, but excessively soft and compressible. The body wastes, without presenting extreme emaciation, or the dry and shrivelled skin usually associated with a protracted malignant disease. Slight pain or uneasiness is from time to time referred to the region of the stomach, and there is occasionally actual vomiting. With every sign of feeble constitution, anæmia, and general prostration, "neither the most diligent inquiry nor the most careful physical examination throw the slightest gleam of light upon the precise nature of the patient's malady; nor do we succeed in fixing upon any special lesion or cause of this gradual and extraordinary constitutional change."

"With a more or less manifestation of the symptoms already enumerated," writes the same distinguished physician, "we discover a most remarkable, and, as far as I know, characteristic discoloration taking place in the skin." It pervades the whole surface of the body, but is commonly most strongly manifested on the face, neck, superior extremities, penis, and scrotum, in the flexures of the axilla, and round the navel. It presents a dingy or smoky appearance, of various tints or shades of deep amber or chocolate brown; and in one instance the skin was so universally and so darkened as to resemble a mulatto. This distribution of pigment is not confined to the skin, but is also visible in the mucous membranes as well as in some other structures. An interesting case occurred in the practice of Professor Biermer, of Berne (November, 1864), in a weakly girl, eighteen years of age. After continued illness of a tubercular kind, the dark color of the skin commenced, and increased in intensity with the decrease in the weight of the patient till death followed. The eyes of this patient happened to be examined with the ophthalmoscope, although vision was not disturbed. A bluish-black color was everywhere visible through the sclera, and a peculiar pigment existed in the interior of the eye. In the plane of a uniform red-brown color there were bluish-black spots corresponding to the intermediate spaces of the *vasa vorticosæ* from which the retina was distinguished by its delicate white appearance. The disease thus originally described by Addison may be associated, like "*morbis Brightii*," with the expression of several distinct local lesions. The discoloration consists of stains in the lining of the cheeks, and a decidedly blackish tinge of the m



membrane of the lower lips, as if after eating mulberries. Dark areolæ become developed beneath the orbits, much marked towards the middle line of the face. In one case under Dr. Barlow, loss of consciousness and what are termed "fainting fits" were the earliest symptoms noticed (*Med. Times*, January 24th, 1857).

"This singular discoloration usually increases with the advance of the disease; the anæmia, languor, failure of appetite, and feebleness of the heart become aggravated; a darkish streak usually appears upon the commissure of the lips; the body wastes, but without the extreme emaciation and dry harsh condition of the surface so commonly observed in ordinary malignant diseases; the pulse becomes smaller and weaker, and, without any special complaint of pain or uneasiness, the patient at length gradually sinks and expires. In one case, which may be said to have been acute in its development as well as rapid in its course, and in which both capsules were found universally diseased after death, the mottled or checkered discoloration was very manifest, the anæmic condition strongly marked, and the sickness and vomiting urgent; but the pulse, instead of being small and feeble as usual, was large, soft, extremely compressible, and jerking on the slightest exertion or emotion, and the patient speedily died" (ADDISON).

In the volume of *Guy's Hospital Reports* for 1862, Dr. Wilks has given a most able, interesting, and impartial account of the progress of our knowledge regarding this disease, since the time Dr. Addison wrote. His own observations entirely uphold the argument which Dr. Addison attempted to develop; and the cases brought forward by him in that report appear to substantiate in a great measure the original facts on which Dr. Addison's history was based. Nevertheless, Addison's views have by no means received the support of the profession at large. Dr. Wilks thinks that Dr. Addison in some measure contributed to this skepticism, by including among his original cases some which did not present the true features of the disease; and the great merit of Dr. Wilks's paper is, that it more clearly defines, and renders precise, the pathological characters of the disease which Dr. Addison desired to describe. He believed, at the time he published his work, that *any disease* which affected the integrity of the *supra-renal capsules* would be attended by the remarkable phenomena originally described by him. This was an error: for all subsequent observations have shown that no recorded instance of the affection has been connected with cancer, or with any other kind of disease of the organ than that found in the genuine cases of the disease which he first described, and which constitutes the true form of the malady, as Dr. Wilks has demonstrated.

[The disease is insidious in its beginning, essentially chronic, of uncertain duration, and subject to remissions. As regards the influence of age, sex, occupation, it may be said that children under ten years of age are exempt, and it is never met with in persons of very advanced age; males are more liable to be affected with it in a larger proportion than females; and it is mostly limited to those engaged in active manual labor.

There are numerous cases, Dr. Habershon remarks (*loc. cit.*), allied to disease of the supra-renal capsules which recover under proper treatment. The sickness, exhaustion, compressible pulse, and feebleness of the vital functions are not peculiar to this affection, and a discoloration of a very similar kind is found in other maladies, as in exhaustion from over-lactation. Here the cause is removable, and the diseased state therefore remediable; but in extensive deposit in the supra-renal capsules there is a persistent cause of irritation of the vaso-motor nerves connected with those bodies, and constant exhaustion and debility. Dr. Habershon gives a case of syphilitic cachexy, which in hue and symptoms closely resembled a case of Addison's disease—bronzed skin, especially of the face, exhaustion, irritability of stomach, compressible pulse—but where, on the subsequent appearance of periosteal nodules, specific treatment was used, and a rapid cure followed.]

**Treatment.**—If the disease be recognized in its earliest stage, its progress may to some extent be delayed. The asthenia, the depression, the evidence of local irritation about the supra-renal capsules, and the pathology of the disease generally, point to the necessity for tonic treatment and nutritive diet, the avoidance of all causes of depression, and the benefit of rest in bed, and of such medicinal agents as may relieve the vomiting. Glycerine in two drachm doses, combined with fifteen or twenty minims of the spirit of rose, and of the tincture of the sesquichloride of iron, have been of service (E. H. GREENHOW). This may be varied by the substitution of twenty to thirty minims of the syrup of phosphates of quinine, and strychnia, in place of the sesquichloride of iron. The greatest caution is necessary in using purgative remedies, as collapse is apt to follow cathartic medicine.

[Cod-liver oil, when borne, is of decided advantage, as well as a preparation of the phosphates known as Chemical Food. The numbness and irritability of the stomach should be first abated, and for this purpose creasote, bismuth, hydrocyanic acid, iced brandy and water are to be prescribed. For diet, when the stomach will not tolerate solids, and soups, oysters, eggs, cream, milk, and jellies should be given.]

#### BERIBERI, OR THE BAD SICKNESS OF CEYLON.

**Definition.**—*A constitutional disease, expressed in the first instance by general anæmia, culminating in acute œdema, and marked by stiffness of the joints, numbness, and sometimes paralysis of the lower extremities; oppression of breathing (anxietas in paroxysms); a swollen and bloated countenance. The urine is secreted in diminished quantity. The œdema is general, extending only throughout the connective tissue of the muscles, but the connective tissue of solid and visceral organs in every cavity of the body is bathed in fluid. Effusion of serum into the serous cavities very generally precedes death.*

**Pathology and Historical Notice.**—This obscure but very fatal disease is little known to pathologists in this country. Though common in various parts of India, the territorial range of its endemic prevalence seems limited in a peculiar manner. It has been called

met with on the Malabar coast, in Ceylon, and in that tract of country reaching from Madras as far north as Ganjam (HAMILTON). It is principally endemic in that portion of Hindostan called the Northern Circars,—a province lying on the west side of the Bay of Bengal, extending from  $15^{\circ}$  to  $20^{\circ}$  north latitude. Madras, in north latitude  $13^{\circ} 6'$ , appears to be the southern limit of the disease in Hindostan. Towards the north of Madras, in the jails of Guntoor, Nellore, Masulipatam, Rajahmundry, Vizagapatam, Chicacole, Bellary, and Cuddapah, the disease is known to prevail. Towards the south it is never seen. It is said to extend from the coast not farther inland than forty to sixty miles.

It is a disease of a peculiar nature; and it has been extremely frequent and fatal amongst all the troops, both Europeans and natives, at Ceylon (CHRISTIE); and although beriberi is a far more common disease among the natives of India than among the Europeans, yet the rate of mortality is nearly twice as great among Europeans as it is among the natives. Indeed, next to cholera, beriberi must be regarded as the most fatal disease (judging of the proportion of deaths to admissions) to which Europeans in India are liable (WARING); while Mussulmans appear to be more subject to the disease than Hindoos (MALCOMSON).

Among European soldiers in India the ratio of mortality or of deaths to admissions from beriberi is above 26 per cent., and amongst Sepoys it is nearly 14 per cent.; and large as these ratios seem, they are small compared with the ratio of mortality which obtains amongst the convicts in the Indian jails, where the percentage is as high as 36.5 (WARING).

A residence of several months in a district where beriberi prevails is necessary to its development (CHRISTIE, HUNTER, EVEZARD); and the greatest predisposition to the disease exists when troops have been about eight or ten months in a settlement.

The influence of season in promoting the development of beriberi seems to be remarkable. Towards the close of the rainy season the admissions to hospitals are far more numerous than at any other period of the year: damp and moisture, combined with cold, seem very favorable to the production of the disease; and the most severe cases at Trincomalee occur during the change from wet to dry weather (RIDLEY), when a strong and hot land wind prevails, when the atmosphere is extremely dry, and when the night temperature is many degrees lower than that of the day. From August to December, when heavy falls of rain occur, with occasional sultry days, and when the alternations of temperature are at the greatest, and the exhalations the most concentrated, then the admissions to hospital for beriberi are the most frequent (WRIGHT).

The name *Beriberi* was given by the Malabar physicians, and has been handed down to us by writers on Indian diseases as a name for almost every fatal disease of debility: paralysis of various kinds, reflex paraplegia, dropsy, anasarca, cachexia, scurvy, and anæmic rheumatism, with various diseases of the heart and pericardium, have all at some time been included under the common name of *beriberi*. It is therefore necessary to give to the term some

degree of precision, by a definition which will embrace the less phenomena of this remarkable disease.

The majority of the phenomena which characterize the well-recorded cases of this disease are undoubtedly referable to *anæmia*. The debility, the cold extremities, palpitation, dyspnoea or exertion, frequent, small, and quick pulse, the bruit occasionally heard in the neck, the scanty urine, the torpid bowels, the deep pallor of the tongue, all indicate a condition of *anæmia* (EVEZARD). The disease makes its advances in an insidious manner, as all forms of *anæmia* do, without any primary or well-marked train of symptoms; and the indisposition appears to be comparatively slight, which exists as a stage precursory to the visible invasion of the fully expressed disease (WRIGHT, EVEZARD). The approach of the final and characteristic features of the disease appears to be gradually brought about, a constitutional state or diathesis gradually established, and a form of *anæmia* sets in, combined with the cachectic dropsy of Andral—a condition allied to that of *perniciosa* in the female (EVEZARD). Many of the more early observations of the disease concur in regarding the fully expressed phenomenon of beriberi as the result of exhaustion and debility (FARRELL, RIDLEY).

**Morbid Anatomy.**—More or less fluid is found in one or all cavities of the chest; most commonly in the pericardium. The muscular tissue of the heart is in some instances loaded with fluid; hypertrophy of its substance is not unfrequent. The areolar substance of the lungs is loaded with water; likewise the substance of the brain. The ventricles contain an increased amount of fluid, and fluid is effused over the surface of the brain.

The abdominal cavity and the general connective tissue of the body abound in fluid. In general, it may be said that in every case examined after death serous fluid in one or more cavities is found to exist; and the pericardium alone, or in conjunction with some other cavity, was the seat of effusion in above 83 per cent. of the cases—the quantity of fluid varying from two ounces to a pint. The spinal cord is variously altered, either by minute effusion of fluid into its substance, or by congestion of its vessels, thereby accounting for the phenomena of paralysis. The unsteadiness of gait and the paralysis of the lower limbs may be in some measure explained by the mechanism of the spinal canal, combined with the presence of serous effusion in its connective tissue. When it is remembered that the spinal cord more completely fills the vertebral canal in the dorsal region than it does either above or below, it will be understood how symptoms of compression of the spinal cord by serous effusion will most early manifest themselves by paralysis and unsteadiness of gait in the lower limbs. The kidneys have been found enlarged, softened, and *anæmic*; the heart is generally enlarged, pale, flabby, and softened; the lungs are *œdematous*; and serous effusion into minute connective tissue is the only unusual appearance which exists in every case (WARING).

**Symptoms.**—Like rheumatism and gout, the disease expresses itself under several forms, of which three may be noticed, namely,

1. The *acute, severe, or inflammatory form* of this disease, is generally the culmination of the constitutional and local phenomena in a first paroxysm. Numbness, paralysis, and œdema of the extremities are the leading symptoms, followed by dyspnœa, and oppression at the *præcordia*. For a short time previous to any other obvious symptom, the patient, though robust-looking, may not have been able to exert himself in consequence of the partial loss of the use of the lower limbs. This rapidly increases, till he finds that there is inability to walk, accompanied with œdema of the extremities, which very soon passes into general anasarca, affecting the innermost recesses of the textures—if such an expression may be permitted. Febrile symptoms are associated with this acute or arterial anasarca. The skin is hot and dry, the urine is scanty and high-colored, the bowels are costive, and the stomach irritable. There are rapid and full pulsations of the large arteries, while the pulse may be variable at the extremities, accompanied generally with dyspnœa and symptoms of effusion within the chest. In other cases there may be headache, restlessness, and delirium, with a slow and full pulse, indicating serous effusion and pressure on the brain. When the œdema is general, and becomes rapidly developed, the condition of the blood is changed from its anæmic character. It becomes dark and ropy, resembling in some degree the appearance of the blood taken from a patient affected with cholera (WRIGHT).

2. In the *second, the asthenic, or chronic form* of beriberi, the patient is very often more or less worn out by some previous disease; or he may have had a previous acute attack of beriberi, of which there may be a relapse; and it appears that men in whom the disease has once manifested itself are the more subject to future attacks (CHRISTIE), for it is found that one attack predisposes to another (WRIGHT); and then the dropsical symptoms more generally resemble those observable after protracted fevers or other debilitating causes. Abdominal dropsy is most prevalent, accompanied with symptoms of general relaxation,—a small and quick pulse, constipated bowels, scanty urine, loss of appetite, universal œdema, much pitting on pressure, and paralysis of the extremities.

The heart partakes of the general debility. It is flabby, and the venous circulation becomes retarded. Soon, perhaps, it dilates, when a temporary bellows-sound may be heard. After several such attacks and recoveries the heart becomes thickened, and hence we have the post-mortem appearance of either a large and flabby heart, or of one eccentrically hypertrophied (EVEZARD).

3. In the *third and mildest form* of the disease the patients are first attacked with some stiffness or rigidity of the legs and thighs, succeeded by numbness, slight œdema, and sometimes paralysis of the lower limbs. The œdema is in general limited, with slight pitting on pressure. There is no unnatural heat of the skin; the pulse is seldom above the natural standard; the urine is scanty; and the appetite unimpaired. There may be occasional palpitations of the heart, with costiveness, blanched conjunctivæ, flabbiness and



paleness of the tongue, and whiteness underneath the nails. though such patients generally say that they are well, they sometimes acknowledge a slight feeling of numbness and cold of the extremities—symptoms which would readily disappear with appropriate treatment; but after a close night, with either a shower of rain, such a patient would apply for medical aid in the morning, with a scared aspect, sighing breathing, violent palpitation of the heart, sometimes with a diffused impulse, pain in the præcordial region, and a variable, fluttering pulse. In such cases there are also dyspeptic symptoms, with acid eructations and flatulency of the stomach. The scanty and high-colored urine has an acid reaction when voided, shows a specific gravity of from 1025 to 1030, and contains an excess of urea. It is such cases which are apt to become aggravated, and to pass into the acute form of the disease. The œdema then progresses from the lower limbs to the hands, the throat becomes swollen, and the face bloated. A feeling of numbness is experienced round the mouth, and a general feeling of numbness is experienced all over the body, particularly over the extremities, which appear to be unusually weighty and rigid (as when walking, the gait is unsteady). The urine, secreted in diminished quantity, is extremely hot when passing through the urethra, and as the disease progresses, a total suppression of the urine generally takes place. A sense of pain and tightness is felt immediately beneath the inferior edge of the sternum, which sometimes becomes so distressing as to induce the patient to solicit that the part may be cut open, hoping to relieve the tightness by that means. Dyspnoea becomes so urgent as to prevent the patient from lying down; heavy sighing (*suspirous breathing*) occurs, with great anxiety and restlessness. The lips and tongue may now be seen to be cyanosed, and the extremities become cold (RIDLEY).

Universal debility, extreme prostration, anxiety, dyspnoea, cyanosis, œdema, anasarca, and paralysis, are the most characteristic phenomena of the disease. And in some instances, when these phenomena are fully expressed, the advent of the fatal event is so rapid that the patient may die within six, twelve, twenty-four, or thirty-six hours; and in other instances the disease is more frequently prolonged over several weeks. Death is in some cases extremely sudden; and from the anxietas, and the fact that obstructions sometimes occur in the veins (EVEZARD), it is not improbable that death in such instances may be due to *embolism*.

Mr. Ridley especially notices the suddenness of dissolution in most of the cases which he observed among the troops in garrison at Ceylon in 1814. "Very frequently, when speaking to one of the patients, he says, 'I have been called to another, whom I had just left under promising circumstances, and have found him gasping, his eyes protruding, his hands clenched; and in a few minutes he was dead; and it has sometimes happened that the man who was addressing has been taken off in the same manner' (*Dublin Reports*, vol. ii, p. 234).

It is clear, therefore, that the disease cannot be referred to a granular disease of the kidney with *albuminuria*, although albuminuria is sometimes present.



is sometimes present in the urine in a high degree, as Dr. Ranking attempts to show; nor yet with scurvy, as Dr. Morehead teaches. It seems rather to be a constitutional disease *sui generis*.

**Causes.**—The etiology of beriberi is but little known; but there are two points that seem well established in the history of the disease, and which must have a prominent recognition in all investigations relative to the causes which develop the disease. These points are,—*First*, The limited geographical range of *Beriberi*; *Second*, The fact that the morbid train of phenomena is never developed till the patient has resided upwards of eight or twelve months in the settlement where the disease is endemic.

**Malaria**, alternations of climate and temperature, noxious material in the waters of districts, have all been indicated as operative agents in bringing about the disease. But, looking to the fact that all the phenomena of the disease point to anæmia, it may be generally stated, that whatever tends to induce this state will favor the development of beriberi. Accordingly, the disease will owe its origin in one place and in one person to the operation of a series of conditions which may not obtain in another place and with another person.

Mr. Christie found in Ceylon that the aged and debauched were those most liable to the disease. A great proportion of his patients were men accustomed to lead a sedentary and debauched life, such as soldiers, tailors, shoemakers, and the like; who, working at their trade, were often excused military duty, and who, by double earnings, were able to procure a larger quantity of spirits than other men.

With regard to œdema there is a great tendency, in almost every disease in Europeans, to result in serous effusions, after long residence in India. Indeed, Sir Ranald Martin observes, in his admirable introductory chapter to the second part of his great work on the *Diseases of Tropical Climates*, that of all conditions incident to tropical invalids, anæmia is the most common. With regard to some places—such, for example, as Masulipatam (where *beriberi* abounds, in the jails especially)—it has been observed by Mr. Evezard that *œdema* is frequently one of the latest symptoms of *anæmia* outside the jail. He also observes that anæmia appears to be almost the normal state of about a third of the population, particularly the higher castes—a circumstance which he states is partly due to the constitution of the Telugoo, and partly to the eliminating power of the drinking-water, which contains so much of *chlorides* and no *iron*. The natives are well aware of the effects of the brackish water which the majority of them are obliged to drink at the end of the rainy season; and when they find themselves suffering from the anæmia, they procure water from Goodoor, a village about six miles due west of Masulipatam. The water at Masulipatam seems to abound in saline material, such as chlorides, lime, magnesia, and a trace of alumina and iron, in the form of a carbonate (EVEZARD).

The sanitary condition of prisoners in jails is, it is presumed, much worse than the free population outside. The criminals in

the jails of India have just sufficient food to keep them alive, scarcely variety enough. They have not sufficient food to store for fat or warmth, and none of those luxuries which are supposed to check waste of tissue, such as tea, coffee, tobacco, betel, and like; and they have not sufficient clothing, many of the prisoners having only one cloth. Mr. Evezard, however, considers that persistence of the anæmic state is more due to the damp and malarious atmosphere of unventilated cells than to food. The soil is for the most part salt. The water of the tank in the jail compound is brackish and its recession leaves a wide margin of saline substance in the soil, which re-attracts moisture like a cloth that has been wet in sea-water, and which afterwards retains and absorbs the moisture. The *chunam* and mud of the jail walls, and the walls of the cells too, are salt; and in the morning they may be seen dripping with moisture, even in the dry season.

Since improvements have been made in drainage, and since the introduction of a mass of iron, which is allowed to rust and corrode, into each *chatty* of water used by the prisoners for drinking and cooking, not a single case of beriberi has occurred during the wet season. It is lying on the wet ground in wet weather which, Mr. Evezard believes, brings on beriberi among Sepoys who have previously rendered themselves predisposed by voluntary starvation.

Under conditions such as these, it is not difficult to understand how a state of ill-health is insidiously established, during which period some *inbred* morbid material is developed in the constitution, which at last produces the phenomena of beriberi when certain endemic circumstances favor the full expression of the disease.

**Diagnosis.**—The most uniform phenomena in the fully expressed disease is the characteristic numbness of the surface generally, and the paralytic affections of the lower extremities which accompany all the forms of the disease. The disease has been confounded with the *barbiers*—a chronic disease in which paralysis, tremors, spasmodic contractions of the limbs, and emaciation are the most remarkable symptoms. Some, however, are inclined to regard the acute or first form described as the true *beriberi*, while others consider the second form described as the disease known by the name of *barbiers*. It is admitted, however, that the one is often associated with the other, either of them being the primary affection, for cases commencing in the form of *barbiers* often suddenly take on the more fatal and acute form of *beriberi*, while the latter frequently presents the symptoms characteristic of the former (MALCOMSON). The two classes of cases prevail in the same places, seasons, and circumstances, and require the same treatment. It is therefore more consistent to consider the disease under the three forms described in the text.

**The Prognosis** is generally unfavorable, the disease approaching insidiously, the anæmia and constitutional state having often advanced so far as to be beyond the reach of repair. A temporary recovery may be established, but relapses are frequent and convalescence is lingering. A first attack generally leaves unpleasant

phenomena behind. In the case of Mr. Ridley, a surgeon of the Royal Artillery, who wrote an excellent account of the disease as he saw it in Ceylon, and who suffered from it himself, the memory became considerably impaired, and an extraordinary fluttering of the heart, almost producing faintness, occurred at intervals after convalescence was established. Violent palpitation succeeded, which, on subsiding, left extreme lassitude and faintness; and when these phenomena first occurred, a violent beating of the carotids came on; and when this rapid pulsation ceased, he was left so extremely weak and languid as to excite considerable apprehension of a fatal result.

In all cases of *beriberi* a suddenly fatal result may be apprehended at any period after the full expression of the phenomena, probably from *embolism* or *cardiac affection*. The symptoms which denote danger are general œdema, much *anxietas*, and palpitation of the heart, the respiration becoming more quick as the fatal termination approaches. It is labored, quick, and interrupted with sighs and groans, the pulse becoming weak and irregular at the extremities, with palpitations of the heart. If coma or delirium supervene, a fatal event may be expected. Much nausea, obstinate costiveness, a dry and black tongue, are all symptoms of danger. The prognosis is more favorable if the œdema sets in very gradually, and is confined to the lower limbs, when the skin is moist and moderately warm, when the patient enjoys sleep, and when the principal natural functions are unaffected.

**Treatment.**—*Bleeding* has been considered applicable to those cases where there is extreme difficulty of breathing and delirium, when the patient is robust, and when the œdema does not pit much on pressure, where there is rapid and full pulsation of the large arteries, and if the urine shows the existence of albumen (WRIGHT); but, judging from the pathology of this disease, the constitutional influence of stimulants, of generous diet, and of tonics, ought undoubtedly to be the basis of treatment. Should there be irritability of the stomach, the effervescing draught with doses of laudanum and camphor mixture are useful. Saline drinks should be administered, and the extremities should be rubbed with stimulating liniments and rolled in flannel bandages. In the *asthenic* or *chronic* form of the disease the strength must be supported by the most nourishing diet that can be given in small bulk, aided by tonics, and wine if necessary; while doses of equal quantities of *squill* and *digitalis* (ten to fifteen drops of each) may be given twice or thrice daily. In the *third* and mildest form a native remedy called *Treack Farook* is very useful. The ingredients of this medicine are unknown. It is known to have been prepared in Venice, and transmitted to India through Arabia, and was first recommended by Dr. Herklotts, of the Madras Presidency, as a remedy in beriberi. It is a thick extract (in which some terebinthine material largely enters), which is only to be procured from the Moghuls, and in those towns which still keep up some communication with the Arabian Sea. Many observers bear testimony to its good effects in removing the œdema and subduing the pulse (WRIGHT, TRAILL, GEDDES, MALCOMSON). In some

recorded cases the pulse has fallen in four days from 108 to 84 per minute under its use.

The prescription most approved of consists of the following ingredients:

R. *Treack Farook*, ℥ij; Pulv. Rhei, ℥iiss.; Pulv. Cinnam., ℥ij; Caryophylli, gr. xxi.

These ingredients are to be made into a mass or *electuary* honey, of which four scruples and a half are to be taken morning (GEDDES), with more honey, or in divided doses (W) three times daily. The remedy does not seem to be an active cathartic, and can have little power in the *acute form* of the disease. Four or five stools are obtained daily under its use, and its action is not accompanied by any violent purging, increase of pulse, or inflammation to the surface; and after it has been used from one to two weeks the œdema generally disappears, when the numbness and paralysis subside.

The patient should feed on animal food, wheaten cakes, and

In instances where the native remedy has failed to produce a beneficial effect, *nux vomica* has been more successful, commenced with doses of two grains daily, and increasing the dose gradually according to the physiological result. The extract of *nux vomica* in doses of half or a quarter of a grain, combined with iron or quinine in a pill, is the most convenient form.

Local abstractions of blood from the spine have also proved useful; and a blister applied over the loins has given relief in obstinate cases.

No single rule of treatment will apply in all cases. The accompanying condition must be counteracted, on the principles of treatment explained under that disease; and any specially abnormal state, as diseased heart, must be treated accordingly.

When the disease prevailed very generally in the Carnatic in 1782 and 1783, the cases were most successfully treated by a plan containing a quarter of a grain of *extract of elaterium*, combined with *extract of gentian*, given every hour until copious watery evacuations were procured; and this plan was repeated every second or fourth day till a cure was accomplished. At another time this plan of treatment was not so successful; the cases recovered best under large doses of *spirits of nitre*, *antimonial wine*, *friction* with warm camphorated oil, *aperient medicines*, wine, and a nourishing diet.

Mr. Evezard's method of medicinal treatment consists mainly in the administration of *acetate of potash* in gin, in the following formulae:

R. Gin, ℥ss.; Potass. Acet., gr. v; Aquæ, ℥ij; *misce*. To be taken three times a day. If vomiting persists, Hydrocyanic Acid may be useful in relieving it; to the extent of one drop three times a day, in milk.

## BRIGHT'S DISEASE—SYN., ALBUMINURIA.

**LATIN EQ.**, *Morbus Brightii*—Idem valet, *Albuminuria*; **FRENCH EQ.**, *Maladie de Bright*; **GERMAN EQ.**, *Bright'sche Krankheit*—Syn., *Albuminuria*; **ITALIAN EQ.**, *Malattia del Bright*, *Albuminuria*.

**Definition.**—A constitutional affection which culminates in a variety of structural lesions of the kidneys, each of which is accompanied by the persistent separation of more or less albuminous serum from the blood, and by its presence in the urine, the connective tissue, and serous cavities of the body. The urine frequently contains blood, renal structures, exudation material, and desquamative renal products. The kidneys may be either large or small, atrophic or not, with fatty, or albuminous, or amyloid (lardaceous) degeneration, and altered in various ways as regards the vessels, the tubes, and the epithelium. When the constitutional disease is fully expressed there is characteristic anæmia, indicated by pallor of the countenance, a puffy face and œdematous limbs, anasarca or general dropsy, pain in the loins, with more or less albumen in the urine. Associated with these phenomena, the morbid states of the kidney may be various. There is scanty urine and frequent micturition; a sensation of heat and scalding on passing water, dryness, heat of skin, general feverishness and occasional chilliness of the surface, an irritable pulse, generally above the normal standard, emaciation, and progressive debility. Dyspeptic symptoms predominate. There is difficulty of breathing and anæmia-chlorosis. The term is generic, including several forms of acute and chronic disease of the kidney, usually associated with albumen in the urine, and frequently with dropsy, and with various secondary diseases resulting from deterioration of the blood.

**Pathology.**—In Bright's disease, as with all the constitutional diseases, an undue share of attention has been bestowed upon the prominent local lesions in which the existence of the constitutional state has finally culminated. For example;—the anatomical characters of tubercle in phthisis—of cancer growths in the cachexia of carcinoma—of the supra-renal bodies in morbus Addisonii—the serous effusions into the spinal and other visceral cavities in beriberi—of the kidney in Bright's disease and mellituria—of the sediments in the urine in oxaluria, lithiasis, and the like, have been so prominently dwelt upon that a very narrow view is apt to be taken of the important antecedents of these diseases—of the constitutional states under which they respectively become developed, and therefore, also, of the principles which ought to guide the treatment of these respective diseases, and of the means by which their development may be prevented. A vast amount of evidence has been accumulating since Drs. Wilson Philip, Tweedy Todd, Sir James Clark, Bennett, and Ancell demonstrated how prominently the general health was impaired and forms of dyspepsia prevailed long before tuberculosis became fully expressed by the deposition or growth of tubercle; and this kind of evidence tends to show that not only are constitutional phenomena characteristic of tuberculosis long antecedent to the expression of the disease by local lesions, but that other



diseases, having similar constitutional antecedents, ought to demand a more extended study from this point of view than have hitherto obtained. *Bright's disease* and *diabetes mellitus* belong to this group.

Between 1841 and 1846 Rokitansky wrote that the nature of Bright's disease is the more obscure because the question is generally evaded. He considers that the cause of the disease is to be sought for in some anomalous constitution of the blood—to some condition which gives rise to a change in the blood, rather than to any special disease of the kidney. It is this anomalous condition of the blood which must be regarded as the primary affection in Bright's disease, which, from some peculiar relation to the kidney, is followed by the secondary and visible disorganization of the renal tissue. The constitutional origin of the disease is also shown in the fact that both kidneys generally express the local lesion about the same time, or with a brief interval.

It is now nearly nineteen years (1849) since Dr. Walshe, Emeritus Professor of Medicine in University College, published a commentary on a clinical lecture, in which he taught that Bright's disease was not essentially a renal disease, but essentially and primarily a blood disease (*Lancet*, July 14, 1849). Since that time he has maintained the same view regarding the pathology of the disease; and there seems to be now an abundance of facts and arguments to establish its correctness. The conclusions to which Walshe arrived were mainly these: That from the very commencement Bright's disease is a general disease of the blood; that the renal textural changes are not the cause of the phenomena comprehended under the name of Bright's disease; and that the lesions which occur in the kidney do not even furnish any measure of the intensity of the characteristic phenomena of Bright's disease; that the textural changes in the kidney are in reality the local expression of the constitutional state, just as tubercle is of tuberculosis; that the primary constitutional and blood changes are probably due to a morbid condition or direction of the primary and secondary digestion processes; and that Bright's disease is recognized as one of the typical *diathetic* or *constitutional* diseases.

Some of the more eminent pathologists and discriminating physicians of the day take a similar view of the essential nature of Bright's disease. Mr. Simon writes that it "depends on a humoral derangement of the entire system, and (the subacute inflammation of the kidney) commences as functional excitement manifested in an act of over-secretion. The *materies morbi* seek to effect its discharge by means of an increased activity in the secretory functions of the kidney: it stimulates it; and the result of stimulation is not so much an increase of the watery secretion as is an augmented cell-growth in the tubules of the gland. The acceleration of function is incompatible with the maturity of the secreted products; the epithelial cells undergo various arrestations and modifications of development, and become more or less palid and imbued with evidences of inflammation."

Dr. Johnson teaches the same doctrine. He shows that the



bid states of the kidney found in Bright's disease "are often mere local manifestations of a more general constitutional disorder;" and Dr. Watson concurs with him in the belief that "it is the quality of the blood sent to the kidney, and not its quantity, that determines the renal disease."

The very interesting experiments made by Dr. Parkes (*Med. Times and Gazette*, 1854, p. 394) regarding various phenomena in Bright's disease, tend to strengthen the view of its constitutional development, more especially when studied in connection with the observations and experiments of Lehmann, Bernard, and Pavy in relation to the excretion of a substance by the liver, and the formation of sugar in the blood when that substance comes in contact with albumen. Bernard's researches have shown that the blood which leaves the hepatic vein contains a peculiar substance (saccharine?) which does not exist in the blood brought to the organ by the portal vein; and Pavy, of Guy's Hospital, has demonstrated that the liver does not form sugar, *but a substance that becomes sugar almost immediately upon contact with albuminous matters*. It is this material (as Dr. Carpenter writes) which is especially destined as a *pabulum* or fuel for the combustion process, being usually eliminated from the blood in the form of carbonic acid and water, during its passage through the lungs, so as not to pass into the systemic circulation, unless its quantity be either unusually large or its elimination be interfered with by imperfect respiration. This substance appears to be mainly elaborated by the converting power of the liver, either from materials supplied by the food or from the products of the waste of the system (*Animal Physiology*, Bohn's edition, p. 808). Now, both of those conditions obtain in many cases of Bright's disease, if not in all. The condition of the urine before and after food is a point which has been in a great measure neglected till Dr. Parkes showed its importance in cases of this affection.\* In many cases of persistent albuminuria with renal casts and anasarca (not dependent on heart disease) the albumen in the urine is augmented after food.

But apart from the albumen excreted, Dr. Parkes found that the solids excreted were greatly increased. A weak, thin, quiescent man gave off daily 981 grains of non-albuminous solids, whose average in health did not exceed 650 to 750 grains. It has usually been considered that in chronic Bright's disease the non-albuminous solids are diminished (CHRISTISON, FRERICHS, JOHNSON); but, according to Dr. Parkes, this rule has numerous exceptions. To detect the increase of the solids, ALL the urine passed during the twenty-four hours must be collected and examined. It is then found that the urine is of low specific gravity, but so copious that the total amount of solids in it is very great. Such cases are found to be constantly feverish—not so much so as to be detected by the mere heat of skin as felt by the hand; but the thermometer under the

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\* [A modification of albumen is present in all healthy urine in the form of a substance not coagulable by heat or nitric acid, but coagulable by chloroform (GIGON), or to a greater degree by absolute alcohol (HARLEY).—EDITOR.]

axilla will show that the temperature of the body is uniform more than a degree and a half above the healthy standard of Fahr. Thus the thermometer will delicately indicate and express how an increased metamorphosis of tissue is going on in such cases, and observing such an increase among the urinary non-albuminous excreta, it may be concluded that they are partly due to increased disintegration of tissue; and, as Simon writes, "the morbid material which thus stimulates the kidney in its struggle for elimination will sometimes consist of products of faulty digestion (lithates, oxalates); sometimes of matters cast upon the kidney in consequence of suppressed function in other organs (*e. g.*, the lungs, the liver); sometimes the mysterious ferment of a fever poison (typhus, scarlatina, typhoid fever)." This view of the subject and the method of investigating cases of Bright's disease, as well as of other wasting diseases, is not sufficiently practised and studied by the student of medicine; and it will be found, as Dr. Parkes has shown, that many chronic cases of Bright's disease are often really febrile cases, just as tuberculous cases are. In all cases of Bright's disease, and constitutional diseases generally, much may be learned regarding their nature by examinations of the urinary excreta, and microscopic characters of the sediment, in connection with the temperature of the body, the number of respirations, and the force of the pulse. The following formula may be suggested as a method for daily record of the chemical composition of the whole urine passed during twenty-four hours in such cases; and if chemical and microscopical examinations of the urine are made from day to day and carried on continuously, the particular morbid state of the kidney associated with Bright's disease may be probably or approximately ascertained:

DATE.	Quantity in cubic Centimetres.	Specific gravity.	Solids (in grains).	Urea.	Albumen.	Sulphuric Acid.	Phosphoric Acid.	Extractives.

The albuminous character of the blood after meals was long pointed out by Dr. Andrew Buchanan, the Professor of the Institutes of Medicine in Glasgow (*Trans. Phil. Society of Glasgow*, vol. xiv). He found a peculiar state of the blood, amounting to an appearance of leuchæmia, which immediately succeeds digestion in healthy persons, and which seems to contain numerous molecular and puscular elements, resembling chyle or lymph, and consisting of an emulsionized with albumen. To this substance in the blood he gave the name of *pabulin*, and which is still further elaborated in

blood, in the glands, and in the lungs. The earliest notice of milky blood is that in the *Philosophical Transactions*, No. 6, p. 100.

During the processes of digestion in the stomach, albuminous substances are known to undergo extraordinary changes; and during the transit of the blood through the liver, the fibrine and albumen undergo still further modification, so that 30 per cent. of the albumen entering the liver in the portal vein disappears in that organ, and is not to be found in the blood of the hepatic vein (LEHMANN). Thus the liver is known to exert an overwhelming influence on every digested aliment which enters it, and the influence which it exercises over albuminous aliments is not the least important (PARKES). Again, if crude albumen is taken and introduced at once into the circulation, as Bernard has proved by experiment, through the jugular vein, it is incapable of assimilation, and is rapidly excreted in the urine, a temporary albuminuria being thus produced.

[The shifting and elusive nature of albumen, its various forms, and the great difference in the diffusibility of these forms, have been well established by physiologists. Temporary albuminuria has been produced by the injection of a solution of white of eggs into the blood of dogs (C. BERNARD), but not when the serum of the blood was thus injected (SCHIFF, STOKVIS). The experiments of Jaccoud and Stokvis show that if solid cooked albumen be taken into the stomach, even to a large amount, no albuminuria follows; but that it is invariably present when an excess of albumen in solution is so taken; for when the stomach is capable of converting albumen into the absorbable modification, albuminose or peptone, there is no exudation through the capillaries of a healthy kidney; but when, either from functional inability of the stomach, or where its capacity is overtaxed from the introduction of an excess of albumen, the same thing happens, as when white of eggs, milk, and isinglass are directly introduced into the blood, a form of albumen circulates in the blood which should not be there, and which, filtering through the renal vessels, is found in the urine. Transitory albuminuria is met with when there is temporary obstruction to the passage of blood through the kidneys, causing stasis in the intertubular capillaries and Malpighian tufts, whether produced by the irritation of a tangible or a zymotic poison, or from mechanical causes. The toxic action of turpentine, cantharides, alcohol, arsenic, lead, &c., is an example of the first, and a typical instance of the second is the albuminuria of pregnancy.]

In the very interesting and suggestive work lately published by Dr. Basham, there are cases detailed, which illustrate, upon these principles, the constitutional origin of *Bright's disease*; so that, connecting all these observations together,—namely, those of Andrew Buchanan, Walshe, Lehmann, Bernard, Parkes, Pavy, Carpenter, and Basham,—may it not be suggested as a topic for further inquiry (and at all events a direction which inquiry should take), as to whether or not substances usually elaborated by the liver are not arrested in Bright's disease, or that the liver is insufficient to use up the albumen carried into the blood, and which therefore comes to be eliminated by the kidney? The inference at any rate now

presents itself; and, as Dr. Parkes writes, "May it not be possible that, by some failure in preparation, either by the stomach or the liver, albumen enters the blood of the right side of the heart in a crude state, and in a condition similar to that in which it would have been had it been introduced through the jugular vein? If so, thus, being unassimilable, is it not excreted, as in Bernard's experiment, by that ready outlet, the kidney? In support of this supposition," continues Dr. Parkes, "we have the facts, that in many cases of kidney disease seem to be most probably of blood origin, and that among the common antecedents of Bright's disease are circumstances of diet or mode of living which would be likely to impair the processes which should go on in the stomach or liver. In how many cases of Bright's disease have dyspeptic symptoms been present for a long time before renal signs have shown themselves? In how many other cases are signs of liver diseases coincident with the renal signs? In how many post-mortem examinations, although no liver disease has been suspected, do we find evidence that for a length of time the structure of this organ had been seriously diseased? In the history of Bright's disease there are many reasons for believing that the nutrition of tissues is generally and deeply affected."

The renal engorgement is certainly not the first in the order of morbid change: it is secondary to more remote morbid action pervading the system throughout. An altered, defective, or contaminated state of the blood is the source, in all probability, of the morbid phenomena which follow. A careful inquiry into the antecedents of patients suffering from Bright's disease will exhibit many predisposing causes of their failing health; and these will even include such as exercise a marked influence over the quality of the blood rather than such as have any special reference to the kidneys. The most frequent and patent of these predisposing causes are habits of intemperance, inducing an alcoholized state of the blood; the various febrile poisons, particularly the scarlatinal; a scrofulous habit of the body, or the tuberculous constitution (BASHAM). There is here a wide field for future exploration, especially in discriminating the forms of dyspepsia or of ill-health which precede the renal cases, and which are frequently to be seen among the wandering and uncertain crowds who frequent the outdoor or dispensary practice of our large city hospitals. In the morbid anatomy of Bright's disease, and diseases generally of constitutional origin, the microscopic structure of the stomach and intestines, as investigated by Handfield Jones, Schäpfer, Habershon, and Fox, promises to yield important results in connection with the early history of the cases.

**Nomenclature.**—The disease is sometimes classed under "Disease of the Urinary System;" and considered under two forms, namely—(1.) *Acute Bright's disease*, of which the synonyms are, "*albuminuria*," "*acute desquamative nephritis*," "*acute renal dropsy*;" (2.) *Chronic Bright's disease*—Syn., "*chronic albuminuria*," with the following subdivisions: (a.) *Granular kidney*—Syn., "*contaminated granular kidney*," "*chronic desquamative nephritis*"; (b.) *Gouty kidney*.

(c.) *Fatty kidney*; (d.) *Lardaceous kidney*—Syn., *amyloid disease*, *waxy disease*.

The disease has been named after the eminent physician of Guy's Hospital, who, in 1837, first drew the attention of the profession to the connection which he observed to subsist between certain forms of anasarca and lesions in the kidneys. It has of late been most extensively investigated by Christison, Frerichs, Ringer, Walshe, Parkes, Gairdner, Wilks, Goodfellow, Basham, [Bostock, O. Rees, Rayer, Virchow, Rasmussen, Abeille, Jaccoud, G. Johnson, G. Harley, Bence Jones, W. H. Dickinson,] and others. The questions which the subject opens up in pathology are of extreme importance. The disease has been termed "granular degeneration of the kidney" by Christison; but looking to its pathology, and to what is now known regarding the morbid anatomy of the kidney in this disease, perhaps it is better designated by the name of the distinguished physician who first described the more prominent phenomena of its course.

[Much confusion still prevails in the classification of those diseases of the kidney which are, during life, made known by the presence of albumen in the urine. Under the generic term of Bright's disease, there is a series of disorders, each of which, to a certain extent, has a distinct clinical history and anatomical character, and arises from separate causes, but they have many features in common, and, frequently, complicate each other. Virchow was the first to adopt a classification on an anatomical basis. Warranted by clinical observation, it greatly simplifies an intricate subject. This plan has been adopted by the most recent and most satisfying writer upon albuminuria, Dr. W. H. Dickinson.\*

For the purposes of the physician, the kidney may be divided into three parts. 1. A complication of tubes, which, with the Malpighian bodies in connection with them, are the essential elements of the gland and the sources of its secretion. 2. A delicate and pervading web of connective tissue, of the appearance of a fine uniform network, surrounding and supporting each tube and each Malpighian body. 3. Abundant intertubular capillary bloodvessels. We have here three loci of disease. Either of these structures may be primarily the seat of morbid change, and the alteration may remain for a long time, or even to the end, limited to the structure in which it has begun. Hence we have disease of the secreting channels, *tubal nephritis*; disease of the connective tissue, *granular degeneration*; disease of the bloodvessels, *lardaceous* or *amyloid infiltration* (DICKINSON); or, a, *parenchymatous nephritis*; b, *interstitial nephritis*; and, c, *amyloid degeneration* (RASMUSSEN); according as one or other of the three different constituents of the kidney is primarily affected. "Each of these forms affect different periods of life; arise from different causes; give rise to dissimilar symptoms; and are unlike each other in their course, in their tendency to recovery, and in the treatment they require" (DICKINSON, *l. c.*, p. 12).]

**Symptoms.**—It is necessary to inquire into the history of each particular case, so as to ascertain the precise period, if possible,

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\* [On the Pathology and Treatment of Albuminuria, 1868.]



when the general health began to be impaired ; and in order to determine the particular morbid condition in which the kidney exists, it is necessary to make a chemical and microscopical examination of the urine from day to day, determining especially the points which have been enumerated in the preceding section.

The general symptoms which are indicative of the several cases, or states of the kidney, comprised under the general term "Bright's disease" have been recently analyzed and admirably described with great care by Dr. Goodfellow, Lecturer on Medicine at the Middlesex Hospital. They may be comprised under the following heads :

1. Although the anasarca or general dropsy, either or both of which are usually prominent symptoms when a patient with Bright's disease seeks medical advice, yet in nearly every instance have been preceded by other phenomena more or less definite, *e. g.*, febrile excitement, a dry and harsh state of the skin, a quick and hard pulse. It is only in the acute cases that the prominent characteristic phenomena of anasarca come on with great rapidity and commence generally with puffiness of the eyelids, or of the whole face, rapidly extending over the rest of the body. In more chronic cases the œdema generally commences in the lower limbs ; and it is at the same time associated with a pallid coloration of the eyelids, and of the looser connective tissue of the face.

[In *tubal nephritis*, the urine becomes scanty and dark, there is pain in the loins, and œdema begins in the face, extending over the whole body ; then there is effusion into the peritoneum, next into the pleural sacs, and finally into the pericardium. Œdema of the larynx may happen.]

Many cases of renal *granular degeneration* occur without dropsy. In 68 cases, there was no dropsy in 19 (DICKINSON). When present, it at first shows itself in œdema ; where there is no œdema, there is no effusion into the serous cavities. After the tegumentary cellular tissue, the pleural sacs are most frequently affected, then the peritoneal cavity, more rarely, that of the pericardium. Where the renal disorder is complicated with heart disease, which is by no means infrequent, dropsy is a conspicuous symptom.

In *lardaceous disease* of the kidney, œdema is of gradual access, the ankles swelling towards evening ; ascites follows ; the pleura and pericardium generally escaping, except when the seat of intercurrent disease (DICKINSON).]

2. Anæmia is another prominent phenomenon. It is indicated by the pallor of the surface of the body and of the countenance. Its progressive appearance may even suggest a suspicion of Bright's disease before the anasarca sets in, especially in patients above the age of from thirty-five to forty, and whose urine ought therefore to be at once examined.

3. Pain in the loins may or may not be a sign of significant disease. Lumbar pains may be considerable in amount, and yet no disease may be capable of detection at the time. In most cases an unpleasant sensation is felt in the lumbar region till the anasarca becomes considerable.



4. In the early stages of the affection there is always a very considerable diminution in the quantity of the urine passed. But there are many exceptions to the rule, and in some cases the urine is more abundant than usual,—*e. g.*, Parkes has measured more than 100 ounces; Rosenstein (quoted by Parkes) relates a case where an increase of 174 ounces was passed daily during eight days. Various circumstances concur in determining the greater or less amount of water passed. (See under "Lardaceous Degeneration," vol. i, p. 134.)

(a.) The anatomical condition of the kidneys influences the amount, and especially with or without uræmic symptoms. The mean of *six* cases without uræmic symptoms gave 61.5 ounces daily. The mean of *four* cases with uræmic symptoms gave thirty-eight ounces daily, the specific gravity in both sets of cases being very nearly the same (PARKES). There is some reason to believe that, in the highly atrophic kidney, when many vessels are obliterated, the urinary water is on an average lessened; but yet on some particular days a large quantity may be excreted: for example, Dr. Parkes has known as much as *sixty ounces* passed in one day by a kidney under such a condition.

(b.) The coexistence of dropsy with anæmia in an advanced period of the disease appears to be associated with lessened water; while, on the other hand, an improvement in dropsical symptoms is attended by more or less profuse diuresis.

(c.) In a few cases the amount of urine is influenced by the presence or absence of fever. The quantity is very much smaller in amount on fever days as compared with fever-free days.

(d.) Organic lesions of the liver, heart, and lungs may either cause lessened rapidity of flow through the renal vessels, or give rise to great variations in lateral pressure.

(e.) One of the main causes of a variable amount of water is the spontaneous purging or vomiting which sometimes takes place, and which would lessen the amount of urine; while, on the other hand, lessened skin exhalations will increase the flow.

Until the metamorphosis of the nitrogenous tissues in Bright's disease is better understood, the question as to the possible lessening of *formation of urea* and *uric acid* must remain undecided. Hitherto, lessening of these products seems to have been the rule, to which, however, there are numerous exceptions (PARKES); and on the whole it appears probable that the effect of a *febrile* Bright's disease on the ordinary urinary constituents is chiefly owing to the physical condition of the kidney. There is no doubt that urea in the blood is increased, and it seems pretty clear that the urea in Bright's disease is still formed to a considerable amount, and that its lessening in the urine is in part owing to *retention* from simple impediment to diffusion through the diseased renal vessels and tubes; but it is yet undecided whether or not there is at the same time a *lessened formation* of urea (PARKES).

The principal abnormal constituent of the urine in Bright's disease is dissolved albumen, which exists in various conditions,

and gives different reactions with the usual tests, heat and nitric acid. Sometimes it entirely coagulates, and is precipitated by heat and a moderate quantity of nitric acid; at other times, after precipitation by heat from an acid urine, it is entirely resolvable in a moderate amount of nitric acid. A very minute quantity of nitric acid will also sometimes prevent its precipitation by heat, while on the other hand, it is sometimes precipitated by nitric acid, and not by heat (various authorities quoted by PARKES). Its usual condition, however, is that in which it is precipitated by heat from a urine which is naturally sharply acid, or which has been made so by a moderate quantity of nitric or acetic acid.

Of the apparently anomalous phenomena just described, the following explanation is given by Dr. Parkes: "When albumen is experimented with, it can be made to pass through various phases of solubility and insolubility, from the actions of acids, alkalis and salts, without its real nature or composition being in any way altered; and as such influences will act on it more or less in every urine, it by no means follows, when the albumen in the urine of a person with Bright's disease presents modifications in its reactions with reagents, that it is in any very peculiar or unusual condition" (*The Urine*, p. 390).

The amount of albumen varies much, ranging from 5 to 54 grains in the twenty-four hours (PARKES); and in any particular case the amount varies greatly from day to day. It is often increased during the day, from movement or from food; and Dr. Parkes has noticed it very greatly increased in the second and third hours after dinner. The phenomena, however, is not constant, nor does it occur in acute cases of Bright's disease; though, when passed, it is often in a peculiar condition, being less perfectly coagulable by heat, and sometimes approaching in character the albuminose of Mialhe. The albumen does not always increase as the disease advances; on the contrary, while there is a tendency to a larger excretion of albumen in the early stages of the disease than in the later ones, and while sometimes even at the last there is much albumen, yet it is sometimes entirely absent in the later period of the disease. In a certain number of cases the albumen may disappear from the urine; but it is important to notice that the detachment of fibrinous casts goes on even when the albumen has disappeared.

In small quantities fat is very common in the urine, derived directly from the kidneys. It is usually in the form of *oleine*, mixed up with albuminous substances, or contained in the epithelial or other cells, and microscopically visible; and it must be remembered, as Dr. Johnson was the first to determine, that some of the secreting or epithelial cells of the kidney inclose a minute quantity of fat, just as in the secreting cells of a healthy liver, as shown by Bowman.

In the more advanced stage of chronic Bright's disease, instead of the urine being scanty in quantity and having a tolerably high specific gravity, the urine passed in twenty-four hours may amount to from *thirty-five* to *fifty* ounces or more, being equal to, and in

some cases greater than, the average in health. The specific gravity is nearly always below the healthy average, the urine pale, and in very advanced cases almost colorless. Occasionally it may be red, reddish-brown, pale smoke-brown, or "smoky," as it is commonly described. If the urine is highly fatty, it may have a milky appearance.

Besides the albumen and other constituents of the serum of the blood, the urine may contain blood-corpuscles, casts containing blood, fibrinous filmy matters, and casts of tubes (granular, fatty, waxy, or hyaline), or simply of epithelium. A perfect acquaintance with these casts and urinary deposits is essentially necessary; for by the characters of these casts, when taken in connection with the history and symptoms, the physician is able approximatively to discriminate the morbid condition of the kidney in each particular case (BASHAM, GOODFELLOW).

The specific gravity may range from 1020 to 1025, from two causes, namely,—(1.) The presence of serum, which is heavier than urine; (2.) From the small amount of water (relatively) (GOODFELLOW).

[In *tubal nephritis* the urine is scanty, albuminous, and generally, at the beginning, contains enough blood to cause discoloration, giving, when acid is present, a black or smoky tinge; but when the urine is alkaline or neutral, a pink or red color. The specific gravity is nearly that of health. A copious sediment is thrown down, which, in the absence of blood, consists of cells of renal epithelium, natural or fatty; or these may be converted into pus-globules. There will be also casts, which, especially in the early stage, are epithelial, and later, are granular; or they may be simply transparent fibrinous cylinders. When there is hæmaturia, the casts may contain blood-corpuscles, or be tinted brown by hæmatine.

The chemical changes in the urine are: diminution of all the constituents; the water, urea, and the chlorides are lessened to a greater extent than in any other renal disorder, while the phosphoric, sulphuric, and uric acids are also decreased.

In *granular degeneration* the urine is the converse of that in tubal nephritis, being first increased and then diminished, and is usually bright and clear. When scanty, in the later stages, it may become turbid from urate of soda. Early in the disorder there is neither albumen nor casts; a few coarse granular casts first are seen, then a trace of albumen, and afterwards albumen, which increases. The specific gravity is below the standard, varying from 1007 to 1015. As a rule, the acidity is lessened. Blood is less frequently passed than in tubal disease. So long as the disease is uncomplicated by catarrh of the tubes, and without hemorrhage, the microscopic sediment will be composed of coarse, granular, large, opaque casts. When tubal catarrh is a complication, epithelial cells are present as a loose deposit, either in their natural state or with more or less of the character of pus.

The chemical changes are: increase of water, except in the later stages, when there is diminution. The urea, uric, and phosphoric acids are slightly reduced until the urine becomes scanty. The sulphuric acid and the chlorine are diminished, especially towards the last. Albumen is not invariably present, is variable in quantity, often being of small amount.

The first change of the urine in *lardaceous disease* of the kidney is an increase of quantity, ranging in the earlier periods from 50 to 90 c in the twenty-four hours. It is pale and clear, and of low specific gravity 1015 to 1006. The albumen, at first, is in minute quantity, and slowly increases until it forms a bulky coagulum. Later, there is generally a reduction of the quantity of urine, due to a certain degree of catarrh, or from the tubes being stopped up by fibrinous exudation; seldom falls below 20 ounces, but may be lessened to 8 or 10 ounces in the twenty-four hours, and it is then largely albuminous. There is an increase in the acidity. Blood is rarely present. In a series of 48 cases there were only 4 with obvious bloody discoloration. Casts appear in a very early period, when there may be only a trace of albumen, and they increase as the disease goes on. Those which especially belong to the early period are simple cylinders of fibrine, and occasionally a few which give the reaction are found. They may be large or small, and are frequently dotted with oil, as a consequence of a change within themselves. A catarrh of the tubes is intercurrent, characteristic epithelial cells are found. When the secretion is scanty, a deposit of uric acid or uric soda is not infrequent.

The chemical changes are: a reduction of all the constituents of urine except the water, which is increased; in cases of long standing the acid is frequently diminished. Of the mineral constituents, that which is constantly lessened is the phosphoric acid.]

5. There is nearly always a frequent desire to micturate, especially at night, when the patient is in the horizontal position (WATSON), with a sensation of heat or scalding on passing water, accompanied with a discharge of mucus from the urethra, which possesses a more or less puriform character, and appears in the urine in the form of slight thin shreds (GOODFELLOW).

6. Dryness of the skin prevails, and the power of eliminating water by the skin seems impaired. Heat of skin and general redness, with occasional chilliness of the surface, and a pulse generally above the natural standard, are common phenomena throughout the latter stages of the disease, and when emaciation and debility are progressive.

7. *The Dyspeptic symptoms* which prevail at an early period indicate irritation of the gastro-intestinal mucous membrane. Loss of appetite, sometimes amounting to actual loathing of food, capricious and uncertain appetite, are amongst the most prominent phenomena. The food taken rests uneasily in the stomach, gives rise to stomachal and intestinal *pneumatosis* and acid eructation, the explosive force of the gas so generated being sometimes great as to cause partial regurgitation of the food. Gastralgia and pyrosis may prevail; and there is very often nausea, retching, sometimes vomiting, at occasional intervals. Attacks of diarrhoea are frequent, alternating with costiveness.

When such phenomena are discovered to exist, the urine ought to be examined at once.

8. *Symptoms referable to the State of the Blood.*—The blood in Bright's disease being decidedly watery, and the red corpuscles deficient, the stimulus of healthy blood is not experienced by

heart; on the contrary, the morbid condition of the blood impairs the action of the heart, and its circulation through the capillary bloodvessels. Palpitation is caused by very slight exertion, or by any mental emotion, and the heart beats *irritably*—the sounds being preternaturally sharp and abrupt, the rhythm disturbed, and the action irregular and intermittent. With the advance of the constitutional cachexia, the nervous and muscular structures of the heart are ill-nourished, and may become temporarily or permanently damaged; signs of pericardial effusion may occur, or even of pericardial or endocardial inflammation. The condition of the blood varies; but (1.) There is an excess of serum, the clot constituting not more than one-fourth part of the blood. (2.) The density of the serum returns to its normal standard, or even exceeds it; sometimes, however, it remains low, even in the advanced stages. (3.) The urea disappears as the disease advances, but reappears towards the fatal termination of the case, even in a larger amount than before. (4.) The fibrine, increased in the first stage, returns to its normal amount as the disease advances, and only becomes considerable again towards the close, especially if some of the intercurrent attacks of inflammation supervene, so common in Bright's disease. (5.) The most remarkable change the blood undergoes is the great decrease of the red blood-globules. They are frequently reduced to *one-third* of the normal proportions; and this diminution progressively advances with the disease; and, as Dr. Christison observes, "there is no disease of a chronic nature which so closely approaches hemorrhage in its power of impoverishing the red particles of the blood." (6.) There are also present in variable, but always in considerable quantities, highly stimulating, irritating, perhaps toxic matters—pure excrementitious material—which can never be retained in the blood without more or less disastrous effects upon several great vital processes, and leading in some cases rapidly to death (GOODFELLOW). Dr. Watson is of opinion "that the renal malady has a direct tendency, by its effect upon the blood, to generate disease of the heart. It induces anæmia, and thereby debility of the muscular texture of the heart, and leads to dilatation of its cavities; and the weak muscle, becoming irritable also, grows thicker as it labors more."

The experiments of Dr. Hammond, the late [Surgeon]-General of the Medical Department of the [United States] Army, have proved that urea, when retained in the blood, either by disease or extirpation of the kidneys, is sure sooner or later to kill.

[The recent experiments of Oppler, Schottin, Perls, and Zalesky, already referred to, indicate, in a very clear manner, that uræmic manifestations depend mainly and essentially on the accumulation in the blood and tissues of those primary products of tissue-metamorphosis (creatine, creatinine, and other extractions), which, in a later stage of histolysis, are converted into urea and uric acid. But these experiments are still more remarkable for their general bearing on the current views of the functions of the kidneys. It has hitherto been supposed that the office of the kidneys was merely to separate from the channels of the circulation and transmit to the urine, the urea and uric acid which already



existed preformed in the blood ; but it now appears that *urea and uric acid are actually produced in the kidneys*, and that any traces of them found in the blood are due to reabsorption from the urinary channels. So the observations on which these conclusions are based are, that in animals whose ureters had been tied, urea was more abundant in their blood than in the blood of those whose kidneys had been removed. Perls found accumulation in the tissues of rabbits whose kidneys had been ligated; whereas if the ureters had been ligatured, there was rapid accumulation of urea. Zalesky found the same in dogs, and that the accumulation of a large quantity of urea in the blood did not increase the amount of ammonia in the blood ; and that creatine was always considerably increased in the muscles after nephrotomy.]

The blood, therefore, in Bright's disease, being poor, thin, watery, and containing much less albumen, and fewer red corpuscles, than in health, and containing, moreover, extraneous offensive matters in the form of urea and the extractives—more or less urinous elements—is ill adapted to facilitate circulation, but, on the contrary, tends to retard its progress. Ultimately the blood is charged with further impurities. It begins to abound in fatty matter, especially cholesterin, which becomes deposited in several tissues, taking the place of their own proper elements of nutrition, and interfering with their function, as in the heart, the arteries, and the capillaries. "The body is poisoned in detail by the retention of its own elements."


[Dr. Dickinson writes: "Some of the disturbances which have formerly been assigned to uræmia, are now known to depend upon specific changes. The diarrhoea of the depurative [lardaceous] disease depends upon alteration of the intestinal vessels ; the vomiting and dyspepsia upon similar alterations in the vessels of the stomach ; the affection of vision which accompany granular degeneration, upon morbid changes in the retina. But there remain many, and most grave, affections, of which the cause lies in the state of the blood. Of these, the most important are the disorders of the nervous system, the coma and epileptiform attacks by which renal disorders are so often terminated, and the convulsions, drowsiness, headaches, cramps, and convulsive movements, as well as various alterations in the temper and state of mind. The reason to believe that many of the apparently spontaneous inflammatory disorders result directly from the condition of the blood ; the uric acid, when present in the blood, having an irritant effect upon the tissues" (*l. c.*, p. 235).

The following table by Dr. Dickinson shows the condition of the various organs as associated with a definite state of the kidney :



[ANALYSIS OF BLOOD IN ALBUMINURIA. PROPORTIONS IN 1000 PARTS.

Case.	Water.	Corpuscles.	Fibrine.	Corpuscles and fibrine together.	In 1000 parts of Serum.			
					Solid matter.	Albumen.	Salts (inorganic).	Urea.
AVERAGE COMPOSITION OF HEALTHY BLOOD ACCORDING TO—								
Christison, . . . . .	775.7	137.1	3.8	140.9	83.4	—	—	
Owen Rees, . . . . .	792.2	—	—	119.95	87.85	79.5	7.5 (alk.)	
Hassall, . . . . .	787.6	—	—	143.0	—	69.4	—	
IN TUBAL NEPHRITIS—								
1. Case 10, p. 208. After Scarlatina. Dr. Christison, .	857.2	42.7	4.5	47.2	95.6	—	—	
2. Lancet, 1864. Edw. G. With fatty change. Dr. Hassall, .	889.5	—	—	86.8	—	23.7	—	
Frerichs. Scarlatinal dropsy. Youth, aged 18, . . .	908.10	130.7	3.4	131.4	91.9	81.4	9.09	
“ “ “ Girl, aged 21, . . .	938.9	122.0	4.2	126.2	61.1	51.7	—	
IN GRANULAR DEGENERATION—								
3. Dr. O. Rees. Guy's H. R., 1843. Wm. Curtin, . .	853.11	—	—	65.61	81.28	68.5	6.0 (alk.)	.05
4. “ “ “ Chas. Scott, . .	835.85	—	—	81.61	82.52	—	—	
5. Dr. Hassall. Lancet, 1864. Francis S., . .	821.0	—	—	120.0	—	59.0	—	
6. { Dr. Harley. Albra. of pregnancy. Before delivery, .	825.9	103.51	2.30	105.81	72.68	60.69	9.08	
“ “ (patient convalescent). After “ “ .	768.7	141.7	2.85	144.55	94.0	86.5	5.000	
IN DEPURATIVE DISEASE—								
7. Dr. Christison, p. 194. Case 7, . .	885.3	56.4	6.2	62.6	52.1	—	—	]
8. Dr. Owen Rees. James Back. Guy's H. R., 1843, .	828.9	—	—	94.1	76.98	65.15	—	

 *The following Table, on page 139, should have been inserted on page 141, preceding “Morbid Anatomy of the Kidney in Bright’s Disease.”*

[TABLE SHOWING THE PERCENTAGE OF SECONDARY AND OTHER AFFECTIONS ASSOCIATED WITH THE THREE FORMS OF ALBUMINURIA WHICH HAVE BEEN DESCRIBED (DICKINSON).

	Tubal Nephritis.	Granular Degeneration.	Depurative Kidney.
<b>URINE, ETC.—</b>			
Hæmaturia, . . . . .	46.1	15.0	8.3
Frequency of micturition, . . .	10.2	19.7	8.3
Pain in loins, . . . . .	25.6	13.5	10.4
<b>DROPSY, ETC.—</b>			
Œdema, . . . . .	97.4	72.0	68.7
Ascites, . . . . .	51.2	26.4	25.0
Hydrothorax, . . . . .	28.2	33.6	2.0
Fluid in pericardium, . . . . .	2.5	4.3	—
Erysipelatous inflammation, . . .	20.5	4.3	6.2
<b>INFLAMMATORY—</b>			
Pneumonia, . . . . .	25.6	10.2	18.7
Pleurisy, . . . . .	20.5	10.2	10.4
Peritonitis, . . . . .	12.8	4.3	8.3
Pericarditis, . . . . .	2.5	23.5	6.2
Endocarditis, . . . . .	—	5.8	2.0
Bronchitis, . . . . .	20.5	35.4	2.0
Enteritis, . . . . .	—	4.3	2.0
Croup and Diphtheria, . . . . .	10.2	—	—
<b>CIRCULATING SYSTEM, HEMORRHAGE, ETC.—</b>			
Hypertrophy of Heart, . . . . .	—	45.4	2.0
Apoplexy (sanguineous), . . . . .	—	4.3	—
Epistaxis, . . . . .	—	5.8	4.1
Hæmatemesis, . . . . .	—	4.3	—
Purpura, . . . . .	2.5	—	4.1
Affection of retina, . . . . .	—	7.3	—
<b>MUCOUS FLUXES—</b>			
Vomiting, . . . . .	23.0	25.0	22.9
Diarrhœa, . . . . .	12.8	2.9	45.8
<b>URÆMIC AFFECTIONS OF BRAIN—</b>			
Convulsions, . . . . .	25.6	16.1	6.2
Coma, without convulsions, . . .	5.1	20.5	4.1
Other cerebral symptoms, . . .	10.2	19.7	2.0
GOTT, . . . . .	—	23.5	4.1]

9. *Dyspnœa* is one of the earliest and most pathognomonic phenomena of chronic Bright's disease; and this shortness of breath is quite apart from, and independent of, any bronchial catarrh or disturbance in the first instance. The patient observes every now and then how short his breath has become. In an ordinary walk he finds he must stop to recover his breath. Occasionally the attacks occur at night, but chiefly during the day; and there is more or less palpitation during the paroxysms; while some wheezing at the chest may also attract the patient's notice. Weeks are thus apt to pass before the patient considers the symptoms sufficiently grave

to seek medical advice (BASHAM). If the urine be examined at early period, the presence of small quantities of albumen may be detected, and the sediment will contain granular casts, with or less decayed cell-structures. Another cause of dyspnoea is œdema which is apt to set in and pervade the base or more dependent parts of the lungs. It must also be remembered that the lungs, the pericardium, and pleura are organs especially liable to the intercurrent attacks of inflammation in Bright's disease.

10. *Symptoms referable to the Nervous System.*—In the absence of anasarca these symptoms are of great importance when they occur coincidently with such other general symptoms as follow anasarca or puffiness of the face. Most of the phenomena are those present in *chloro-anæmia*—*e. g.*, noise in the ears—a blowing or a sense of ringing noises in the ear, occasional dimness of vision, partial amaurosis, from paralysis of more or fewer of the papillæ of the retina, giving rise to the appearance of motes or small bodies floating before the eyes (*muscæ volitantes*). In some instances there may be flashes of light from irritation of the fibres of the optic nerve.

[Certain nutritional retinal changes would seem to be almost constantly associated with granular degeneration. The retina and the optic nerve are infiltrated with serum. The retina opposite to the attachment of the optic nerve becomes tumid and of a whitish-gray color; the capillaries are dilated and thickened. Minute, and, sometimes, extensive extravasations of blood are apt to take place within the thickness of the retina. The connective tissue, as well as the nerve-fibres, are thickened and altered. Galezowski has seen atrophic changes in the nerves as high as the corpora quadrigemina, and has noted fatty patches in and about the chiasm. Liebreich's Atlas, "Bright's retina," as seen through the ophthalmoscope, is pictured (GALEZOWSKI, BADER, HULKE).]

Throbbing of the temporal and other arteries, a sense of fullness or weight at the back of the head and in the nape of the neck, with a disposition to frequent cramp of the muscles of the neck and sides of the neck; neuralgic pains in the face and head, in other parts of the body; headache in the form of *hemicranial migraines*; frequent attacks of giddiness, drowsiness, disposition to comatose sleep, and in some cases profound coma, alternating with convulsions, are all premonitory symptoms of chronic Bright's disease, and may be also fatal ones. The occurrence of what is called *head symptoms* may be frequent during the course of the disease, and death of the patient is so generally preceded by such symptoms, with or without convulsions, that Dr. Christison considers "death by coma" to be the "natural termination" of Bright's disease in the mode in which it proves fatal when life is not cut short by some other incidental or secondary affection. Dr. Watson is impressed with the idea that the pale and watery condition to which the blood is at last reduced may have something to do with the stupor and coma, as in *spurious hydrocephalus* similar phenomena are seen. The disposition to giddiness is often a remarkable characteristic phenomenon. A disposition to erysipelas has also been noticed. Anasarca is one of the earliest symptoms; in ninety

out of every one hundred cases it is the first intimation which the physician has of the disease; but if he could see the patient regularly before this symptom sets in, there are no doubt other phenomena, perhaps less definite, which he would have no difficulty in setting down as the probable antecedents of Bright's disease.

Pallor and emaciation, if combined with anasarca, are almost pathognomonic of the disease.

**Morbid Anatomy of the Kidney in Bright's Disease.**—There are several different forms of lesion in the kidney associated with the phenomena of chronic Bright's disease. The several forms are distinguished by certain anatomical characters, and by more or less characteristic symptoms throughout the disease. Two at least of these forms may be considered as typical and distinct; and other forms may be recognized as a commingling of these two—modifications of them—or a mixture of these typical states together, but not to be considered as a gradation from the one form into the other. The two forms are essentially different, and never pass by any pathological process from the one to the other (WILKS, GOODFELLOW). Other forms of morbid kidney in this disease are regarded as degenerations, namely,—(1.) The fatty; and (2.) The amyloid kidney. (See page 124, vol. i.)

The late Dr. Bright, the distinguished physician of Guy's Hospital (who first discovered the relation subsisting between these morbid kidneys and certain cases of anasarca), described the disease, which now bears his name, as an affection of the kidney passing through *three* stages. Martin Solon describes *five* stages, and Rayer no fewer than *six*. More recent observers—for example, Frerichs, Reinhardt, and Chambers—recognize three stages. Those who contend for the difference of stages affirm that in the first stage the kidneys are usually large, flabby, loaded with dark venous blood, and hardly in any respect different from what is observed in diffuse inflammation, except that externally the kidney has a granular appearance, caused by the deposition of a dark, reddish-yellow matter, which is an infusion of inflammatory products.

The second supposed stage is marked by the granular matter penetrating still deeper into the cortical substance, and which gradually increases till it invades the whole of the medullary substance of the kidney. This granular substance is of a grayish-red or grayish-yellow color, and has in many cases something of a *cheese-like* appearance. The kidney now may be larger than natural, sometimes of the natural size, and sometimes, though rarely, diminished. Its consistency also varies; for, if enlarged, it is commonly softer than the healthy kidney; but, if diminished, it is for the most part firmer. Its color, viewed externally, is sometimes a pale tint of the natural hue, but more commonly it is of a grayish-yellow or yellowish-red color, and mottled. Its surface is also strongly granulated, and even rough. In this state, if the kidney be injected, the matter of the injection does not, according to Dr. Bright, penetrate the cortical portion. This is the stage in which it is considered that a complete and general metamorphosis of the inflammatory products into fat takes place.

The last stage is marked by the morbid granular deposit, which besides invading the medullary substance, attacks the tubular portions of the kidney, so that the tubuli are often to a very considerable extent obliterated, and perhaps, with the exception of a simple pencil of that structure, is entirely converted into one homogenous degeneration. The kidneys are now, in some instances, of their natural size, but more generally they are contracted, and are smaller than usual. Their surface is lobulated, pale, and granular, resembling the roe of a salmon or the vitellarium of a bird. Their consistency also is sometimes softer and sometimes harder than natural, and Dr. Bright speaks of some instances in which they cut like butter. This is the stage which leads to final atrophy and wasting of the kidney, and is no doubt that form now capable of recognition as amyloid degeneration.

In opposition to these views which hold that *Bright's disease* is always one morbid state passing through successive stages, the observations of Dr. George Johnson, of King's College, of Dr. Wilson of Guy's Hospital, and of Dr. Goodfellow, of the Middlesex, have led to the belief that there are two at least, if not more, great classes of disease of the kidney, developed independently of each other, but associated with the phenomena of Bright's disease.

[A few words on the proper mode of examining the kidneys microscopically, are necessary to the correct understanding of the morbid anatomy of Bright's disease.\*]

**Examination of the Kidney under the Microscope.**—The state of the epithelium is best shown by scraping the cut surface, and placing a minute portion of the *débris* gathered on the knife, with a drop of water under a quarter or eighth object-glass.

By this plan, tubes are often detached also, but these structures are best displayed in a section. To obtain this a double-bladed knife may be used; but the best way is to expose a small piece of the organ—a half inch—to the action of a mixture of ice and salt, by which, in a quarter of an hour, it becomes perfectly hard, so that sections of any degree of tenuity can be cut with a sharp scalpel or razor. Sections made in this manner are adapted to show the effects of reagents, in iodine, &c.

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\* [The morbid anatomy of Bright's disease has hitherto been very contradictory and unsatisfactory; and of the microscopical changes, as Dr. Roberts remarks, "flagrant discrepancies between the descriptions of various high authorities are everywhere to be met with." This has been chiefly due to erroneous notions of the anatomy of the healthy kidney. The recent researches of German histologists into the minute structure of the kidney, made, in many instances, independently of each other, have elucidated several disputed points. For some years past, Dr. W. H. Dickinson has studied the healthy and morbid anatomy of the kidney with great care and perseverance, and has succeeded in bringing the microscopic examination of the organ to a degree of perfection never before reached. He is enabled by his method of treating the organs to preserve them all *in situ*, and thus obtain, by means of large sections, an accurate view of the exact relation that they bore to each other during life. The result of his labors, published in his recent work on Albuminuria, has been pronounced by the most perfect treatise yet written on the anatomy of Bright's disease" (*Brit. and For. Med. Chir. Rev.*, vol. i, 1868). As the latest, and, as the writer believes, the most correct view of the several diseased conditions of the kidney met with in Bright's disease, it has been thought best to substitute a brief statement of these investigations in place of the confused and imperfect views of former observers given by the A. in the text.—EDITOR.]



This plan will also show the fibrous tissue, but it is found that the intertubular structures and the attachment of the capsule are seen with more distinctness in the smoother sections which can be made from portions hardened by boiling or by chromic acid. A small piece of the kidney boiled for a few minutes in water slightly acidulated with acetic acid, and then allowed to dry, affords transparent and beautiful sections. (See *Med.-Chir. Trans.*, vol. xliii, plates 7, 8, and 9.) A level surface may be cut with a razor, a drop of water put upon it, and a section removed which includes the moistened part.

This plan is ready, effective, and suited to those who wish to economize their labor.

Better results are, however, obtained by hardening a piece of the organ in chromic acid, and making transparent sections therefrom after the method introduced by Mr. Lockhart Clarke. A piece of kidney of the requisite size is allowed to remain for two or three weeks in a solution of chromic acid in water, in the proportion of about two grains to one ounce. This makes it hard, and fit for manipulation. With a razor, the blade of which is kept wet with spirit, beautiful sections can be cut from the resistant mass. The sections, however, are too opaque to show well unless they are very thin.

The next step is to make them transparent, and at the same time to impart a little coloring matter. The sections are first left for a few minutes in water, so as to remove all traces of spirit. They are then placed in a weak solution of carmine and left there until they have taken a faint pink tint. They are then put again in water, so as to remove all excess of coloring matter, and are thence transferred to spirit. When they have become permeated by the spirit, they must be removed to turpentine. A small quantity of turpentine should be put in a saucer or evaporating dish, or any vessel with shelving sides. The sections should be ranged around the fluid, so that a corner of each touches its surface; the result will be, that in each section the spirit will evaporate from above, while the turpentine rises from below, and after a time the section will be saturated with the latter fluid, and will assume a remarkable translucency. When the section is thoroughly imbued with turpentine, the object of this apparently complicated process is obtained. The several steps are needed in order to replace the water in the tissue by spirit, which is more volatile, and to replace the spirit by turpentine, with which it is not miscible. With regard to the coloring with carmine, precautions are necessary to avoid the contact of spirit with that pigment. When the sections are perfectly translucent, they may be put up in Canada balsam, and will remain imperishable memorials of the patient from whose body they were obtained.

The beauty and clearness of the sections obtained by this method leave nothing to be desired.

*Chronic Tubal Nephritis.*—The kidney does not drip blood when cut open; it is increased in bulk, the weight being often doubled; it is harder than in health, unless it is fatty; there is change of color, the secreting surface having a peculiar opaque whiteness, or a pale buff shade. When white and ivory-like, the epithelium will be found natural; when with a yellow shade it is fatty. The surface is perfectly smooth and glossy, and the capsule readily slips off. The fine network of capillaries, which belongs to the surface in health, has disappeared, and is replaced by red blotches of vessels disposed in a stellate form, large enough to be followed by the naked eye. On section the pallor of the cortex often contrasts

with the cones, which retain their normal tint, though sometimes latter are paler than natural, owing to the same change that has affected the rest of the organ. The cones are separated from the capsule by increased thickness of cortex, and are sometimes compressed into shape of a wheat-sheaf.

If the gland be picked to pieces, and looked at under the microscope with a high power, numbers of distended convoluted tubes will be seen. They contain the epithelial scales, either natural or fatty, and commonly a proportion of them will be found broken up into a granular debris. Fibrinous exudation is often present. Of 27 kidneys thus affected the epithelium was free from oil in 10, slightly fatty in 4, and generally fatty in 13.

The straight tubes offer more variety. Some, like the convoluted, are generally packed with the products of epithelial growth, while others contain transparent fibrine. In the earlier stages this transparent effusion, which has probably come from the Malpighian body, is contained in a natural epithelial lining; as the disease advances, the cells disappear more or less completely, leaving the tube, which now consists of a naked basement-membrane, filled with large transparent plugs.

If a section has been cut from a portion of a kidney previously frozen, it will be seen that the tubes only are distended, and that the other structures are unaffected, except the Malpighian capsules, which may be dilated by the accumulation of their contents. Sometimes all the tubes are alike affected; sometimes those near the surface are chiefly implicated, or they may be found as little isolated masses in the midst of healthy structure. Sections prepared by boiling, or by hardening in spirit of chromic acid, give like results. There is no thickening around the bloodvessels, nor any intertubular spaces visible under the capsule elsewhere. None of the tubules are constricted from without, as in the normal kidney. The kidney is large and smooth to the last, unless a fatty change is superadded, with gradual loss of bulk, and sometimes sparse depressions of surface. It is by this alteration, until lately little understood, that the "small smooth kidney" is produced.

A *fatty condition* of the renal epithelium when of some standing and extensive, gives rise to a striking peculiarity in the appearance of the organ. The outside still remains smooth to the touch, but is covered and sprinkled with little white sharply defined specks, like bits of bran. The tubes have a broken appearance, and their white color forms a contrast with the yellowish cortex, through the whole of which they are interspersed. This is characteristic of a great amount of fatty change in the affected epithelium. The specks are aggregations of fat within the tubes. Where the epithelium has become fatty to a less degree, it is often difficult to detect the change except by the microscope. The only difference to the naked eye is a somewhat yellowish shade over the cortex, and a coarseness of texture which contrasts with the close fine grain seen in the epithelium is unchanged. The importance of fatty change has hitherto been overstated. It is not a primary change, but the result of an altered state of nutrition of the cells consequent upon inflammation. Though rapidly produced, it is never seen in the earliest stages.

*Granular Degeneration.*—In the *early stage* the kidney is not materially altered in bulk; its texture is perhaps firmer than in health; the color is of a slightly more reddish or congested tone; the capsule is adherent to the surface somewhat granular. On section, there is often no change appreciable to the eye; the grain of the cortex may be a little coarser, and possibly one or more cysts may be detected in it. When examined

the microscope, little fibrous processes, starting inwards from the depressions, are seen, and often in these are embedded shrivelled tubes. The deep parts are still free from any change, and the majority of tubes, as well as the epithelial cells, are natural. In the latter there is now and then a deposit of oil in their outer part,—a change, however, which happens in all varieties of renal disease, and also in health. There is no crumbling or disintegration of the epithelium.

The appearance of the gland in the *advanced stage* is very characteristic. It is reduced in size, a kidney which should weigh five or six ounces weighing only two or three; the more chronic the disorder, the smaller the organ, as a rule, though occasionally it retains its size, or this may be increased. Regularity of shape is lost in consequence of the uneven shrinkage; often the central portion seems to have fallen in more than the ends. The capsule is generally thickened, opaque, and adherent, though it will usually come off with tearing the substance. The exposed surface is studded with prominent hemispherical granulations, about the  $\frac{1}{16}$ th of an inch in diameter, of a light color like that of parched peas, while the depressed spaces between them have, from vascularity, a purplish or faint red tint. The superficial vessels are only seen in these intermediate spaces, where an irregular network often exists, forming a contrast with the prominences, which are always bloodless. There are frequently cysts on the surface. When a section is made through the centre, the cortical part, especially near the surface, seems to have undergone most change. The part lying between the cones and the capsule is greatly thinned, whilst there is much alteration in color and texture. The natural brown color is replaced by a yellowish-gray or buff, often intimately mingled with a red tint, as if two materials, a red and a yellow, were closely intermixed. The grain is closer and firmer than in the natural state. Cysts are often found both in the cones and cortex. The cones are less affected than any other portion of the organ; when altered, their color approaches that of the cortex.

When the renal disorder results from heart disease, the kidney is larger and much redder, both within and without, than when it comes on from other causes. The granulations are smaller and more indefinite, and the distinction of color between the granulations and the depressions less marked. The organ is harder and more brittle, often loaded with blood, which should be soaked out to make the buff color of the cortex apparent.

The minute changes in advanced granular degeneration, as seen by the microscope, show that the essence of this affection consists in a slow irritation of the connective tissue, especially that which immediately surrounds the Malpighian bodies and bloodvessels, followed by a proliferation of that tissue, so that it gradually usurps the place of the tubules, which it contracts and flattens till they become mere threads. The microscopic appearances are as follows: The most conspicuous alterations are at the surface immediately beneath the capsule, at the points of superficial depression, little streamlets of fibrous tissue passing into the organ and embedding the Malpighian bodies and compressing the tubes. As the fibroid material extends inwards, it becomes diffused, and spreads over and between all the tubes in its neighborhood. Or its existence may be chiefly indicated by the contraction it has caused, so that an angular space under the capsule is seen filled by the shrivelled remains of the tubes. In such cases the Malpighian bodies are aggregated, owing to the contraction of the parts between them, while they resist the compressing agency. The new fibrous tissue is sometimes seen in isolated patches of

some extent in the deeper parts of the cortex. In some portions the tubular tubes have been reduced to impermeable microscopic threads, in others they are irregularly dilated. The effusion which leads to the increase of fibrous tissue does not take place uniformly throughout, but at points a little removed from each other. The tubes in the track of the effusion may be involved whilst others escape, and, in the earlier stages of the disease particularly, many tubes may be found perfectly normal. Those which are altered are in one or two conditions, both of which are commonly found in the same kidney: (*a*) they are packed with epithelium, or distended with dark granular matter, probably the result of the breaking up of the cells subsequent to their detachment from the wall of the tube; (*b*) a transparent fibrinous material may take the place of the epithelial lining, and occupy the tubes. This material may be quite firm and glossy, or studded with oil-globules. Sometimes it is broken up into very fine fragments, and passes out with the urine as dark, coarse granular casts, which often look opaque and granular till touched with acetic acid, when they become clear, and show the broken pieces of tubes of which they chiefly consist. The condition of the tubes which form the renal cones is precisely the same.

In all cases, excepting those in which the contraction of the organ is extreme, the epithelium is the same as that found in healthy kidney. In alteration of form alone, the cells becoming somewhat angular, cramped in growing space, is sometimes seen in very advanced cases.

In the later stages there are always cysts, both in the cortex and medulla, generally very minute, though sometimes conspicuous to the naked eye. Frequently these have a linear arrangement, particularly evident in the renal cones, where they are often oval in shape, and lie end to end like a string of sausages. Their walls are of thin membrane, upon which nuclei of epithelial cells are seen, thus closely resembling the neighboring tubules. They are produced by accumulation of fluid in, and distension of part of the tube which remains open between two points of obstruction, or by dropsical distension of the capsules of the Malpighian bodies, the necks have been similarly closed by external pressure. The large cystic kidney, where it is transformed into a collection of large cysts, is probably only an extravagant extension of the process.

**Lardaceous Degeneration of the Kidney.**—Examined at the earliest stage in which the change can be recognized, the kidney is slightly paler than normal, and anæmic. If altered in consistence it is a little firmer. The surface is smooth. Later there is an increase of size, the kidneys, when fatty, weighing 10 or 11 ozs. each; and the capsule becomes adherent, and the cortex is firm, pale, and thicker than natural. When there is fatty degeneration, the cortex is of a pale fawn-color and opaque, like a parsnip. When the size is less, with no fat, the cortex has a pinkish or gray translucent appearance, most observable near the capsule. If the gray effusion is abundant the organ is firm and elastic. Sometimes the two conditions coexist, the specks or lines being separated by gray waxy matter. The capsule is adherent; the surface, still smooth, is marked by a few superficial depressions, and has a general bloodless appearance, variegated with irregular red blotches, or a few stellate vessels. The effusion is tubular, and has the same contractile tendency as in granular degeneration. According to the duration of the disease, and the rate at which the contracting process goes on, the appearance and bulk of the organ vary. Sometimes the surface is covered with large smooth elevations, giving an undulating outline; at times it is uneven in places, and elsewhere shrivelled. Again, in the most advanced stage, the organ is shrunken out of

having lost more in width and thickness than in length, with a lean and attenuated look; and when the capsule is with difficulty removed, the surface has a sandy texture. On section the cortex is found much thinned, and contains numbers of shining dots like specks of glass, which are the enlarged and altered Malpighian bodies. Cysts, usually of very small size, are common.

Early in the disease the iodine solution only dots the Malpighian bodies; sometimes they will stand out like grains of brown sand; later brown lines become visible on the cones, which are affected straight vessels; finally, almost the whole substance, as well as the surface, will give the characteristic color,—the deep brown shade of polished walnut wood, which contrasts with the light yellow hue of the healthy portions.

There are three successive steps in the *minute and essential changes*, as shown by the microscope: *a.* An alteration in the walls of the blood-vessels; *b.* An effusion through them into the tissue and cavities of the gland; *c.* Consequent changes in the tubes, causing their obstruction.

The bloodvessels primarily and chiefly affected are the Malpighian bodies, the vasa afferentia, and the arteriolæ rectæ of the cones. By the time the change has extended to the straight vessels, or often while it is apparently limited to the Malpighian bodies, an effusion of fibrinous matter has taken place into the intertubular tissues, especially near the surface, seldom equally diffused, but poured out in greater bulk at those points where the course of an artery has determined the position of a family of Malpighian bodies. Around these new tissue is often seen\* embedding the tubes which lie in its way, and, by its contraction, reducing them to solid threads, or cutting them up into minute cysts. The same material is poured out into the tubes, forming waxy, fibrinous casts, passed in the urine.

In the earlier stages of the disorder the natural structure of the cortical tubes is displayed with great distinctness; they are fixedly open, and the epithelial cells adhere with much tenacity to their walls: this is due to minute fibrous exudation, which glues the cells to each other and to the tube. Sometimes the tubes become obstructed by catarrh, as in tubal nephritis, and they then become plugged with fatty or natural epithelium. The tubes may be distended by fibrinous plugs, and are often irregularly dilated and constricted by the contractile tissue around them. Cysts, from transformation of the tubes and dilatation of the Malpighian bodies, are found embedded in the new growth.

Fatty degeneration is constantly associated with this disease.

The relation of the morbid changes to the symptoms is evident. The primary and essential alteration in the arterioles, though it thickens their walls, makes them leaky; hence an unnatural outpouring from the Malpighian vessels, and an increase of urine, which at the same time is mixed with serum. The tubes remain freely open, and for a long time nothing hinders the passage of serous or aqueous fluid from the Malpighian bodies. The fibrinous part of the exudation which passes from them forms the casts which are so abundant. Partly by the plugging produced in this way, and partly in consequence of a certain amount of tubal inflammation, which is apt to come on towards the end of the disease, the tubes become obstructed, and the hitherto copious urine becomes scanty,—diminution of urine being in every form of kidney disorder, in direct proportion to the obstruction of the tubes.]

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\* [Dr. Dickinson supposes that the effused deposit becomes converted into fibroid tissue; but, as lardaceous disease of the kidney is often complicated with granular disease, it is a question whether this finely nucleated new tissue is not the result of a proliferation of connective tissue, the result of the granular disease.]



**Causes.**—The exciting causes of Bright's disease may be enumerated under the following heads,—namely, hereditary tendency, constitution of body, age, sex, climate, occupation, habit of intemperance, exposure to cold and moisture, influence of zymotic disease poisons.

[TABLE SHOWING AGE AT WHICH THE THREE FORMS OF RENAL DISEASE TERMINATE FATALLY, CALCULATED FROM CASES UNDER THE OBSERVATION OF DICKINSON.]

PERCENTAGE OF CASES FATAL AT DIFFERENT PERIODS.

Age.		Tubal Nephritis.	Granular Degeneration.	Lardaceous Disease.
From	0 to 10, . . . . .	87	0	5
"	11 " 20, . . . . .	17	0	18
"	21 " 30, . . . . .	23	11	85
"	31 " 40, . . . . .	18	18	16
"	41 " 50, . . . . .	10	80	16
"	51 " 60, . . . . .	0	82	5
"	61 " 70, . . . . .	0	6	5
"	71 " 80, . . . . .	0	8	0]

Intemperance, gout, and disease of the heart are the circumstances which seem to bring about the disease most frequently in advanced life.

[Granular disease of the kidneys is frequently associated with gout. In 69 fatal cases there were 16 in which the disorder was dependent or coincident with, gout. The latter is invariably the primary affection (DICKINSON). Dr. Garrod has shown that the gouty habit is often associated with lead poisoning.]

Granular degeneration is produced by the slow absorption of lead. It is constantly met with amongst painters, plumbers, compositors, and other workers, and is the only form of renal disease caused by chronic poison. Of 42 men who had had to do with lead, and who died from disease or accident, and were examined at St. George's Hospital, 23 showed distinct granular degeneration of the kidney, and in most that died led to the death of the patient (DICKINSON). A man with a blue line on the gums, and having albuminuria, is almost certain to be the subject of granular kidney.]

In youth and childhood the hereditary constitution of body, exposure to cold, or the effects of fever, such as scarlatina, erysipelas, measles, variola, syphilis, gout, rheumatism, tuberculosis; the indiscriminate and excessive indulgence in fermented liquors, such as alcohol, ale, stout; the ingestion of turpentine, ether, naphtha, chloroform, and the like, are the circumstances and agents which tend to that morbid state of the constitution, and that impairment of the state of the digestive functions which lead to Bright's disease.

[A tendency to general fibroid proliferation in many parts of the body in old age, and amongst drinkers of ardent spirits, is well known. (See article on Fibroid Degeneration of the Lung, by the Editor, in this volume.) This general tendency, from whatever cause it arise, may involve the kidney in common with other organs. Dr. Dickinson found in 10 cases of granular disease of the kidney, the liver cirrhotic in 37 (



1 in 7), while there was noticeable thickening in the capsule of the spleen in 47 (about 1 in 5).]

A cold, humid, and variable climate is another powerful predisposing cause; although the disease is also by no means infrequent in tropical climates.

[The geographical distribution of Bright's disease shows its intimate dependence on atmospheric influences. Albuminuria, as a primary disorder, belongs especially to temperate climates. Arctic cold and equatorial heat seem alike inimical to its development. It chiefly abounds where the mean temperature of the year is not far from 50° Fahr. In Melbourne, with a mean temperature of about 57°, the disease is scarcely less prevalent than in London. On the Mediterranean coast it is extremely rare (CHAMBERS); in India it is very infrequent (MORTON); whilst Iceland is almost exempt (HJALTELIN).

There is a remarkable correspondence between the variability of temperature and the existence of Bright's disease; but no direct connection between it and humidity of the air appears. The reader may consult with advantage the excellent chapter of Dr. Dickinson "On Climate in Relation to Renal Disease," in his late work *On Albuminuria*.]

These are causes which act directly upon the blood and nervous system, through the digestive and secretory functions; and which tend to interfere especially with the circulation and secretion of the kidney. The hereditary constitution of the body is of great importance. There may be such inherited weakness of constitution that impaired digestion may at once lead to the development of Bright's disease, rather than to any other constitutional disease.

There are causes, also, of Bright's disease which may be regarded as mechanical causes of irritation, but which so secondarily affect the constitution of a person predisposed to the disease that Bright's disease rather than any other is the result—*e. g.*, the irritation of blows, of cantharides, or other irritants; the presence of calculi in the kidney, the practice of onanism, or excessive venery, the existence of amenorrhœa or uterine irritation. Dr. Goodfellow points out that the influence of spirits and of beer tends each to induce a distinct variety of morbid kidney. From the action of spirits the kidneys do not enlarge, nor vary much from the normal weight; but they become extensively granular, and full of small cysts. The cortical portion is greatly reduced in thickness, and there is much fatty matter in them.

[Dr. Dickinson is at variance with most authorities in his estimate of the effects of alcohol in the production of granular degeneration of the kidney. His conclusions, resting on pathological facts, and large observation with special reference to this point, do not accord with opinions based on theoretical considerations alone, for he believes that, while excess in the use of ardent spirits is a pre-eminent cause of cirrhosis of the liver and of the lungs, it rarely injures the kidney, and that when it does act prejudicially there, it gives rise to tubal nephritis with fatty epithelial degeneration, rather than granular change. He brings forward a large body

of facts in support of his views which are worthy of further investigation. For instance, he shows, by the Reports of the Registrar-General comparing the proportion of deaths from alcohol with those from disease, that the prevalence of the one bears no proportion to the prevalence of the other, but rather the contrary holds good.]

In persons who become the subjects of Bright's disease from excessive consumption of beer, the lesions of the kidney are generally of a mixed nature, something between the large white kidney seen after scarlatina and the true granular kidney, with more or less fatty deposit both in the tubes and in the interstitial tissue.

But while intemperance in drinking is a well-recognized cause of Bright's disease, it is not unknown among children and other persons whose manner of life has been strictly sober. Dr. Watson mentions the example of a young girl, fifteen years old, who had menstruated, and who became affected with Bright's disease; he remarks that the disorder has been known in many instances to follow a sudden check or suppression of the catamenia.

**Diagnosis.**—The specific gravity of the urine, the albumen it contains when persistent, and the amount of the solid constituent are the first indications of the real nature of the disease. To determine the albumen, a small quantity of the urine in a test-tube must be slowly and gently heated to the *boiling-point*, by the flame of a spirit-lamp, when, if albumen is present, it will appear in the form of a whitish cloud, of which the constituent particles multiply and collect, in proportion as the quantity is considerable, into small fragments or flakes. These will gradually subside to the lower part of the tube when permitted to rest, leaving the supernatant liquid clear, and so indicate approximatively the amount of albumen present. A second specimen of the urine should be taken in another tube, and, after it has been thus boiled, an excess of acetic acid may be poured into the tube, when the albumen present will be precipitated in a flaky or pulpy form. This latter method is the best where the urine to be tested is alkaline; but both methods should be employed in every case; and they are sufficient to determine the presence of albumen.

The amount of solid constituents may be determined approximatively at the bedside by the following formula: If  $D$  = the density or specific gravity of the urine, and  $\Delta$  = the difference between 1000 and its density, the quantity of solids in 1000 grains will be  $= \Delta \times 2.33$  for diabetic urine, and by 2 for most cases. Suppose the specific gravity to be 10.20, then  $20 \times 2.33 = 46.60$ , which is the amount of solids in 1000 grains of urine; or simply multiply the last two figures of the specific gravity by 2 for most cases, and by 2.33 for diabetic urine, will give the amount in *grammes* of solids in 1000 cubic centimetres of urine (PARKES).

The formula of Trapp (the error of which, according to VENABLE, cannot exceed one-tenth in healthy and one-fifth in morbid urine) is the best for urine *not diabetic*, and is as follows: If  $\Delta$  represent the excess of the specific gravity of urine above that of water, the amount of the solid constituents of 1000 parts of urine will be represented by  $2 \Delta$ .

A sufficient approximation to the truth for clinical purposes is gained by such formulæ; but they can never supersede the exact processes necessary for scientific investigation (CHRISTISON).

The following table, calculated by Dr. G. Bird, according to Dr. Christison's formula, affords results sufficiently accurate for the guidance of the student or practitioner, and shows at a glance the number of grains of solids, and the weight of a fluid ounce of urine, of every density from 1010 to 1040:

Specific Gravity.	Weight of 1 Fluid Ounce. Grains.	Solids in 1 Fluid Ounce. Grains.	Specific Gravity.	Weight of 1 Fluid Ounce. Grains.	Solids in 1 Fluid Ounce. Grains.
1010	441.8	10.283	1025	448.4	26.119
1011	442.3	11.337	1026	448.8	27.188
1012	442.7	12.377	1027	449.3	28.265
1013	443.1	13.421	1028	449.7	29.338
1014	443.6	14.470	1029	450.1	30.413
1015	444.0	15.517	1030	450.6	31.496
1016	444.5	16.570	1031	451.0	32.575
1017	444.9	17.622	1032	451.5	33.663
1018	445.2	18.671	1033	451.9	35.746
1019	445.8	19.735	1034	452.3	36.831
1020	446.2	20.792	1035	452.8	37.925
1021	446.6	21.852	1036	453.2	38.014
1022	447.1	22.918	1037	453.6	39.104
1023	447.5	23.981	1038	454.1	40.206
1024	448.0	24.051	1039	454.5	41.300

The hydræmia is indicated by the general paleness or anæmic appearance of the surface of the body; and even when no albumen can be discovered in the urine, these phenomena are of great value; and when they are observed in persons above the age of from thirty-five to forty-five the physician should at once examine the patient and his urine closely from day to day, in order that he may detect as early as possible the other symptoms and conditions which are characteristic of Bright's disease. The specific gravity of the urine, the quantity passed daily, the amount of urea and of other solids contained in the daily discharge, should all be closely observed, and whether there be not slight puffiness of the eyelids in the morning, or indications of serum beneath the conjunctiva. The paleness is not a dry waxy paleness, as in chlorosis or anæmia-chlorosis. It is a paleness characteristic of dropsy.

When the disease is established, and its diagnosis confirmed, much information will be obtained as a guide to treatment, and as an aid to the diagnosis of the peculiar morbid condition in which the kidney may be, by a daily microscopic examination of the urinary sediments, as recommended by Dr. Basham in his excellent treatise on *Dropsy connected with Disease of the Kidney*.

The specific cell-characters of the sediments have been found by Dr. Basham to be a more certain guide in prognosis than can be furnished by any other property of the urine, or by any other

symptom exhibited by the patient. The cell-elements associated with albuminous urine undergo marked alteration and change as the renal disease advances. Under all circumstances it is difficult to form an opinion as to the rate of progress or advance of lesion in the kidney; and hitherto the physician has been usually guided in his prognosis by the subsidence of the dropsy, or by diminution of the amount of albumen in the urine. The microscopic examination of the cell-elements is now recognized as giving more certainty to the opinion which may be formed as to the progress of the renal degeneration.

The quantity of albumen present is an important point to be considered, along with the characters of the tubular or organic elements contained in the urine; and in private practice the amount of albumen passed is approximatively judged of by noting the length of the *coagulum* of albumen occupies in the tube after it is allowed to rest. The phraseology recommended by Dr. Christy to express the proportions observed by the eye is as follows:

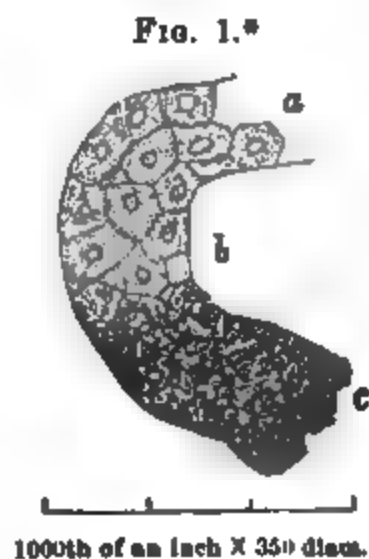
1. *Gelatinous* by heat. 2. *Very strongly coagulable* by heat—nearly the whole tube. 3. *Strongly coagulable*—half the tube. 4. *Moderately coagulable*—one-quarter of the tube. 5. *Slightly coagulable*—one-eighth. 6. *Feebly coagulable*—less than one-eighth. 7. *Hazy* by heat—no visible flakes of albumen.

The epithelial cells thrown off from the renal tubes, as well as the casts which accompany them, suffer very material and obvious alteration as the disease of the kidney advances. These changes consist in palpable deviations from the standard structure of healthy epithelium. The cells lining the straight tubes are the last to exhibit any character of degeneration; and in no case do the cells of the kidney-tubes appear in the urine except under the influence of the disease. Any one single examination of the urine is not sufficient to determine the stage to which the disease has advanced. It is only by comparing the character of the deposit of one period with another, and noting carefully the altered appearance of the casts and cells, and the direction of whatever change is taking place, that an approximation of more or less certainty is made regarding the progress of the disease.

The sediment found in albuminous urine consists principally of cylindrical forms moulded in the uriniferous tubes of the kidney. These present different characters according to the stage of the disease or the intensity of the morbid process. They may be either coarsely granular, finely granular, or partly granular, and transparent, or completely hyaline. Associated with these casts are various cell-structures—blood-cell, epithelium-cell, compound granular cell, and various modifications of cell-growth, as may occur during the advance of acute or chronic disease. When hemorrhage takes place in the uriniferous tubes from rupture of the Malpighian capillaries, the coagulum formed is washed away, and appears in the urine as a cylindrical cast of a granular appearance, containing a number of blood-cells within its mould. These casts are usually stained of a red color by the hæmatin of the effused blood. As the hemorrhagic state subsides, epithelial cells appear in the cast, and

with blood-corpuscles, which gradually become less numerous, and ultimately disappear. In favorable cases the casts become more and more finely granular, and daily more transparent, and the epithelium-cells less and less abundant. In other less favorable cases various modifications of cell-development occur; resplendent granules appear, and free nuclei accompany the cast; compound granule-cells are discharged, abortive epithelial cells appear, with disintegrated granule-cells in the shape of grape-like clusters of nuclei; and other evidence of cell-transformation indicates the nature and direction which the renal disease may be taking. It is the *progressive* and day by day, or week by week, alteration of cell-formation which accompanies the casts of albuminous urine which must be watched to ascertain the progress of the disease.

The first departure from the healthy type of the secreting kidney-cell is evinced by its becoming more granular, the single nucleus being either obscured or accompanied by other nuclear granules. The cells become larger and larger, and present a cloudy appearance; for a larger quantity of material than usual has been taken up by the cells. The entire uriniferous tubules are thereby rendered broader, and even to the naked eye such a tube looks white and opaque. The individual cells are difficult to isolate, because they adhere closely together, in consequence of the alteration of cohesion they have undergone. The few granules normally present in the cells have accumulated and multiplied in greater and greater numbers the greater the energy with which the morbid process is being carried on, so that even the nucleus gradually grows indistinct. This is the condition described by Virchow as that of "*cloudy swelling*" (*trübe Schwellung*), Fig. 1. It is the progressive and constant increase of such compound granule-cells in the urine, or of cells becoming more degenerate and compound, with a proportionate decrease of epithelium, which is the index of advancing disease. The occurrence of such cells is usually preceded by a state of inflammatory congestion; and in proportion to the quantity of these cells, and the degree of degeneration to which they have advanced, so is the degenerative process in the kidney indicated. When groups of grape-like (botryoidal) clusters of resplendent granules appear in the urine, they are generally the nuclei of various cells, and indicate a deteriorated or atrophic state of cell-development, which eventually becomes so powerless that nothing remains but a heap of granules. These accumulating in the tubes and Malpighian bodies, eventually constitute a fatty degeneration of the whole kidney. The occurrence of such clusters in the urine is generally associated with a subacute process of dis-



\* Convoluted urinary tubule from the cortex of a kidney in Bright's disease (after Virchow). (a.) Tolerably normal epithelium; (b.) State of "*cloudy swelling*;" (c.) Commencing fatty degeneration and disintegration.



ease; and when the clusters become daily more numerous, they furnish very unfavorable indications. The large compound cell or inflammation-globule of Gluge never makes its appearance except as a sequel to a state of hyperæmia. There is a period in favorable cases where little or no epithelium is visible, and where transparent hyaline or waxy casts are alone seen. They are more abundant and more frequent in the chronic form of Bright's disease than in the acute; and are then present in the urine of the mildest and most tractable cases equally with the severest and most fatal. Their particular import depends on the cell-structures with which they are associated, and likewise on the relations as to time which other kinds of casts bear towards them. Although they have been named hyaline, yet occasionally they contain traces of granular matter or cell-elements. If they appear in the urine very shortly after hæmaturia, they may be accepted as indicating a state of catarrh of the renal tubes, which in all probability will prove remediable and tractable. In such cases an epithelium-cell is seen here and there in the urine, and sometimes a compound granule-cell; but these are not numerous, and do not increase. In other cases the glairy moulds of the tubes contain numerous abortive cells in almost daily increasing numbers. Compound granule-cells are then always present, clusters of granules (botryoidal or crescentic), and free nuclei are seen. These appearances are characteristic of a chronic subacute process, which progresses slowly, very insidiously, and is too often irremediable.

In a third variety of Bright's disease where these hyaline casts are seen, few or no epithelial cells are present, but the appearances are characteristic of broken-up cell-structures; clusters of granules are seen, having more and more the appearance of fat-granules. Many acquire a large size, and contain oil in abundance. In the more advanced cases the casts seem made up of the fatty and oily materials, and hence they have acquired the name of fatty or oily casts. They indicate the highest state of fatty degeneration.

There is still a fourth variety of the hyaline or transparent cast, and which, from the absence of all structure visible in it, has received the name of waxy cast. This cast may be formed either in the smaller convoluted tubes or in the larger straight tubes of Bellini. They seem to be composed of some viscid material, and become faintly granular on the addition of dilute acetic acid.

The following may be stated as a general summary of the results relative to casts in the urine in Bright's disease:

*The blood-casts* represent more or less active hyperæmia and hemorrhage from the kidney.

*The coarsely granular epithelial cast*, with its compound inflammation-corpuscle, and accompanied by amorphous granular flakes stained with hæmatin, represent the period of inflammatory exudation.

*The finely granular semi-transparent casts*, with scattered epithelium and granule-cells, represent the period of subsidence of the inflammatory process.

*The transparent casts*, with compound cells, or with isolated trans-



parent molecules and grape-like clusters of granules, represent a stage of chronic subacute disease of very grave import; and if these casts become more and more loaded with large and gradually increasing fat-granules and oil-drops, the progress of fatal fatty degeneration is clearly marked (BASHAM).

Deteriorating conditions such as have been here described are not limited to the kidney in Bright's disease. There is reason to believe that the nutrition of most of the textures and organs of the body proportionally fails; and although not so apparent, because not manifested in a manner capable of being demonstrated during life, yet the tissues of every organ become more or less degenerate and inefficient for the purposes of life. The cells of the liver are invariably loaded with an abnormal amount of fat in all fatal cases of Bright's disease; and the heart-fibre and arterial textures exhibit the microscopic characters of atrophy and granular or fatty degeneration. Inflammation of the serous surfaces is a common complication.

The origin of chronic Bright's disease cannot be traced with certainty in all cases. It is one of those diseases, like phthisis, in which the patient rarely applies to the physician till the disease has made considerable advances, often beyond any remedy or means of cure. The commencement of it cannot be recalled or described by any other terms than "a gradual failure of the general health," which is usually designated as "breaking up of the constitution." If the urine be examined at the earliest stage, the presence of small quantities of albumen may be detected, and the sediment shows examples of granular casts, with more or less decayed and broken-up cell-structure. These phenomena often manifest themselves long before any dropsical symptoms point to the existence of renal lesions. There are also exceptional cases in which the urine at this early stage is non-albuminous.

It is important to bear in mind the relation of the hyaline, transparent, and waxy casts to the early and remediable forms of Bright's disease. In favorable cases such cysts become more and more translucent; but if the disease advances, numerous cell-forms occur, and are passed along with it; and when the casts continue to present week after week a great number of free nuclei, associated with hyaline cylinders—numerous compound granular cells, some with, some without, cell-walls—renal degeneration in the most obstinate and intractable form is fatally progressing.

Without a microscopic examination of the urine from day to day it is impossible to distinguish between a case likely to improve under treatment, and one which may be viewed as hopeless; and without the daily use of the microscope the treatment becomes at the best but merely guesswork.

**Treatment.**—It is only the general principles which can be indicated, inasmuch as every case requires a special study, and a line of treatment in detail peculiar to itself. Whatever treatment be adopted, a long time is necessary before any appreciable results are obtained, and therefore it is necessary to persist in one line of treatment steadily from week to week, and even from month to month.

It is obviously of great importance, therefore, to be as accurate as possible in diagnosis as to the probable state of the kidney, so as to define the line of treatment from the first which may seem best adapted for the individual case.

It is a question of very grave importance how far vomiting and diarrhoea ought to be checked. If either of these occurrences are suddenly stopped, the gastric and intestinal membrane acting at the time as an emunctory for the urea and other excreta of the urine, the patient may be suddenly cut off by convulsions, apoplexy or effusion into some of the serous cavities, such as the pericardium or the pleuræ, or the ventricles of the brain. It is necessary, therefore, in the first instance, to determine in all chronic cases the particular organ or tissue which seems in each case to be acting vicariously. The perspirations are often spontaneously profuse; and the skin is by far the safest emunctory for the vicarious elimination of urinary constituents. Therefore it is important to promote the action of the skin if it be deficient, and to encourage it even if it is already considerable. Diaphoretics are always of essential service. The best are Dover's powder, the warm bath, warm clothing, and, for convalescents especially, a moderately warm climate. In Dr. Christison's experience they have always appeared most serviceable when they are so given as to excite a gentle perspiration during a part of the night.

[When the warm bath is used its temperature at the time the patient enters it should be 98° Fahr. and it should be gradually raised to 108° Fahr. After remaining in about twenty minutes, he should be quickly dried, and wrapped in hot blankets (LIEBERMEISTER). Alkaline, hot-air, or vapor baths will promote cutaneous action, and give relief.]

So it is also safe to promote the discharge of secretions from the intestinal canal, with due caution that they do not become excessive, so as to pass into permanent diarrhoea. Urea and other constituents of the urine are found in such discharges in large proportions. When general anasarca prevails, absorption may be promoted by gentle pressure, which at all times must be very cautiously applied, and the effects closely watched, for such effusions afford great temporary relief to important symptoms which indicate the involvement of vital organs. Bandaging to promote absorption is not justifiable so long as the anasarca is increasing. Patients ought to be encouraged to go about as long as they are able, care being taken that they are clothed with flannel and woollen garments, [with an oil-silk suit], and otherwise well protected from chills or draughts of cold air.

The quantity of urea passed by the urine should be determined daily, to ascertain how far the kidneys are capable of secreting and eliminating these excrementitious products. According to the results obtained, the diet must be regulated, and such measures taken as are calculated to reduce the quantity of urea, and other constituents formed daily, to the capacity of the diseased kidneys for the work they are able to do.

In the subacute forms of the disease the action of the skin is especially to be promoted by such saline remedies as the *acetate* or *citrate of ammonia*, to which may be added *one, two, or three drachms of the infusion of digitalis*, and *ten or fifteen minims of antimonial wine*. [Or the *citrate of potash*, made by saturating the fresh lemon-juice with bicarbonate of potash, and of which half an ounce may be given every three or four hours, diluted with water.]

In such cases, also, *ten or fifteen minims of the tincture of the perchloride of iron*, or from *five to ten grains of the citrate of iron and ammonia* may be given *every day* with one of the meals. [Or the *acetate of iron* mixed with *liquor ammoniæ acetatis*, or *acetate of potash*, as the skin or kidney is to be acted on.] *Every second day* a dose of the *compound jalap powder* may be given; and if hyperæmia of the kidneys prevail, it may be necessary to take from four to six ounces of blood from the loins by cupping or by leeches. [Or dry cups, or hot stimulating fomentations may be applied over the loins.] As the urine becomes more free from blood-corpuscles and albumen, the iron medicines may be more frequently given, and the compound jalap powder less frequently.

A very good formula for the administration of salines and iron in the subacute and chronic cases is that given by Drs. Basham and Goodfellow. It is as follows:

*Liquor Ammoniæ Acetatis*, ʒj-ʒij; *Acidi Acetici diluti*, ℞xx-℞xxx-℞xl; *Tinct. Ferri Perchloridi*, ℞x-℞xv; *Aquæ*, ʒj-ʒiss.; *misce, fiat haustus*.

Small doses frequently repeated seem to do better than larger ones given at longer intervals. These remedies tend to lessen the watery state of the blood; and the action of the chalybeate medicines is of no avail till after purgation has been free, and when the hot air or warm baths have caused the skin to act freely. A nutritious diet is then to be given, combined with the chalybeate remedy.

The further treatment of the chronic forms of Bright's disease is influenced by the amount of the anasarca present. If considerable anasarca is present, neither cupping nor leeches can be had recourse to, on account of the bruising and erysipelatous inflammation they are apt to induce. The chalybeate mixture already mentioned is still found of service, taken at least three times daily, and to it may be added, from time to time, *fifteen or twenty minims of the spiritus ætheris nitrici* (GOODFELLOW). The *citrate of iron and ammonia* in from *five to ten grain* doses, with the *sulphuric* or *chloric ether*, are also good and useful remedies; and if the iron medicines are found too stimulating, *sulphate of zinc*, or *tannic* and *gallic acids*, may be used instead. The *sulphate of zinc* is to be given in doses of *one to three grains*, three times a day, in the form of pill, made with *extract of hop*, and with or without a grain of the *extract of nux vomica*, or the same dose of *sulphate of zinc* may be given in a draught combined with *sulphuric* or *chloric ether* (GOODFELLOW). These latter medicines relieve considerably the

flatulence and sensation of coldness in the stomach and bowels, much complained of in such cases.

[When the distension of the skin is very great and threatening, and the means indicated have not lessened or removed it, one or two incisions, three-quarters of an inch in length, and penetrating well into the subcutaneous tissue, should be made lengthwise into the calf of the leg, or one of them on the instep; after the fluid has been pretty well evacuated, the limb should be wrapped in flannel-cloth, wrung out of hot infusion of chamomile flowers, which should be changed every two or three hours while the serum continues to flow in any quantity. (On each change of dressing the limb should be sponged with warm water the cloths should be thoroughly cleansed before being reapplied. This will lessen the risk of erysipelas. Traube recommends that the incision be frequently washed with chlorine water.)]

If the urine is "smoky," or if blood is seen on microscopic examination, the *gallic acid* in *five- or ten-grain* doses may be given with a few drops of *diluted sulphuric acid* and a few drops of the *tincture of hops*, or other aromatic vegetable tincture, in an infusion of the same. [Or the sulphate or muriated tincture of iron may be given.] The objection to these remedies is the constipation they are apt to induce. The bowels should always be kept relaxed, two or three loose evacuations being secured daily. The medicine most generally useful is the *compound jalap powder* of the [U. S.] Pharmacopœia, [with an additional quantity of the salt and a little ginger,] which should be taken in the morning, fasting in half drachm or drachm [or two drachm] doses, in a wine-glass of water. It does not induce prostration; but by repetition it is apt to lose its effect, when *elaterium* may be necessary in small but repeated doses; *two grains of elaterium*, being dissolved in *sulphuric ether*, and *the eighth part of a grain* given every six hours till the desired effect is produced. Very abundant watery purgation is thus obtained, and a sensible impression is made upon the distended dropsical parts (BASHAM).

If *elaterium* may not be deemed advisable, a saline draught of the following composition may prove efficient.

R. Magnes. Sulph. vel Sodæ Sulph., ʒj to ʒij; Ætheris Sulph., ℥x; Acid. Sulph. dil., ℥x; Ferri Sulph., gr. j to gr. ij; Aq. Menthæ Vir., ʒiij to ʒiv.

[Gamboge, in five-grain doses, every second day, is highly recommended by Christison.]

This draught is to be taken the first thing in the morning, once or twice a week. It ought to produce two or three loose and watery evacuations (GOODFELLOW).

[Where there is dropsy with diminished renal secretion, Dr. Dickinson condemns hard purging with jalap and other hydragogue cathartics, and exhausting sweating, for the reason that the water of the blood is diverted from the clogged tubes—the cause of the scanty urine, and concomitant

symptoms, lying in their obstruction. The real principle of treatment is to increase the secretion of water at the kidney itself, and thus flush out the choked-up tubules. This is best done by copious draughts of pure water, with, when necessary, *digitalis* in infusion, as an adjuvant. The repeated use of hydragogue purgatives should be limited to obstinate and hopeless cases, where the failure of other measures has stamped the kidneys as irrecoverable.]

Diuretics are a most certain and speedy remedy when the *drop-sical* effusions are considerable; and they are sometimes advisable when the urine is very scanty, although they are seldom necessary in the treatment of the fundamental disease. Dr. Christison is convinced that the fears entertained by some, of injury being produced by the stimulus of diuretics on the kidneys, are visionary. In his experience, which now extends over more than forty years, the best diuretics are *digitalis*, *squill*, and *bitartrate of potash*, taken simultaneously; and, if these fail, *Hollands* sometimes speedily establishes the diuresis.

When dyspeptic symptoms predominate, and there is considerable flatulence, the following pill is recommended by Dr. Goodfellow to be taken twice or thrice daily:

R. Ferri Sulphatis, gr. j; Ext. Nucis Vomicae, gr.  $\frac{1}{2}$  to gr. j; Mas. Pil. Galb. Co., gr. ij to gr. iij; misce, fiat pilula.

And if there be coexistent bronchitis, a draught of the *acetate of ammonia*, with *ten, fifteen, or twenty* drops of the *spiritus ætheris nitrici*, and half a drachm of the *oxymel of squills*, is to be taken intermediately with the pills. If much nausea prevail, *three minims* of *dilute hydrocyanic acid* may be added to the draught, with the occasional application of mustard to the stomach.

Want of sleep is often complained of, but opiates are inadvisable, because they are apt to check the secretions and to occasion constipation. *Henbane* may be given in place of *opium*.

The patient should select a residence where the soil is sandy or chalky, where the air is mild and dry, so that as much open air exercise may be taken as possible.

[From what has been said (p. 149), the influence of climate in the causation of Bright's disease is evident. In a disorder so little under the control of treatment simply medical, this point becomes, therefore, one of great practical importance. Dr. Dickinson writes: In the impotency of such medicine as is represented by the Pharmacopœia, we may yet be able to take advantage of those universal laws in virtue of which all life and growth are regulated by the physical conditions of the earth. According to our present knowledge, it would seem that a warm and constant climate should be sought. Where the circumstances of the patient will admit, the coast of the Mediterranean, as a winter residence, appears to fulfil the necessary conditions. The Cape of Good Hope would probably answer a similar purpose. It may even be urged that a residence within the tropics, with all its concomitant risks, may be adopted as a preferable alternative to the almost certain progression of the disease in a chilly and changeable climate.]



Diet should be light, but nutritious; no pastry should be eaten and stimulant liquors must be used with caution, although, when exhaustion is great, their use may be unavoidable. The principal meal should be in the middle of the day, and not later than three o'clock; and the last meal should be taken two or three hours before bedtime, retiring early to bed, and rising early. Light but warm woollen clothing is to be always worn, in summer as well as in winter.

The complications of Bright's disease are extremely difficult to manage.

The *diarrhœa* must not be suddenly checked. Thirty to sixty minims in water of the *spiritus ammoniæ aromaticus*, with half a drachm of the *tincture of kino* or of *catechu*, after every loose stool will, in general, be all that is necessary. If there be much griping, the application of a linseed poultice over the abdomen, with two drachms or half an ounce of *tincture of opium* sprinkled over it, will give relief (GOODFELLOW). When it is found necessary to use measures for checking the *diarrhœa*, means should be taken that the skin acts freely, or that the urine flows increased in quantity. Lead and opium pill (*pilula plumbi cum opio*), in doses of five or ten grains twice or even thrice a day, is the best remedy for checking *diarrhœa*. These remedies may be aided by a *suppository* of the *hydrochlorate of morphia* occasionally, each suppository containing a quarter of a grain of the hydrochlorate of morphia (*morphiæ hydrochlor. suppositoriæ*). Vomiting may be controlled to some extent by *bismuth*, but more frequently by *morphia*, *hydrocyanic acid*, *creasote*, *rectified pyroxylic spirit*, *chloroform*, or *chlorodyne*, or little fragments of ice; and if these fail, a blister over the epigastrium has sometimes succeeded (CHRISTISON); [or, a mustard plaster over the lower cervical and upper dorsal vertebræ.]

Intercurrent inflammatory attacks and effusions into cavities are still more difficult complications to manage, and are very dangerous to life. The application of a few leeches, or the abstraction of a few ounces of blood by cupping, may be borne well in the head complications; but the greatest care and caution is required in the employment of similar remedies in the complication of *pericarditis*, on account of the great danger of death by syncope.

*Bronchial complications* are serious, and more or less constantly present; and they are frequently the immediate cause of death in chronic Bright's disease. The least stimulating expectorants may be administered under such circumstances. The following formula is recommended by Dr. Goodfellow:

R. Liquor Ammoniæ Acetatis, ℥ij to ℥iij; Spiritus Ætheris Nitrici, ℥xx to ℥ss.; Oxy mellis Scillæ (*Lond.*), ℥ss.; Aquæ Camphoræ et Aquæ, āā ℥v; misce, fiat haustus. To be taken every four, six, or eight hours.

If the expectoration be viscid, and difficult to discharge, a few

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\* [The use of opium in this disorder is attended with great risk, giving sudden rise to uræmia. It is better to resort to any other suitable means to bring on sleep, induce perspiration, and check *diarrhœa*, than to opium or any of its preparations.—EDITOR ]



drops of antimonial wine may be added to the draught; or if there be much spasm of the bronchial tubes, as indicated by the asthmatic breathing, a few drops of *sulphuric* or *chloric ether* may be given. If, on the other hand, the expectoration be purulent and difficult, a few grains of *carbonate of ammonia* may be given with the *oxymel of squills*.

Every precaution ought to be taken to prevent a fresh attack of bronchitis. So long as the patient is able to bear it, the *habit* of cold sponging and dry rubbing of the surface of the body every morning with a flesh brush or coarse towel is the best preventive, and being clad in warm woollen clothes. Flannel next the skin must be invariably insisted on.

Mercurial preparations are contraindicated in Bright's disease—so great is the tendency to salivation and the otherwise injurious effects they produce. *Podophyllin* (the active principle of the May apple) may be used instead. Its dose is from  $\frac{1}{4}$  to 1 grain; and it ought not to be given in doses larger than one grain. It is best given in the form of a pill combined with soap and hyoscyamus. As an occasional mild aperient pill, the following is also found to answer well:

R. Mas. Pil. Rhei. Comp., gr. ij to gr. iij; vel Ext. Aloes Aquosi, gr. j; Ext. Nucis Vomicae, gr. j; Mas. Pil. Galb. Co., gr. ij; misce, fiat pilula (GOODFELLOW).

[When the symptoms of so-called uræmic intoxication happen, but which are really the result of the combined effects of the retention in the circulation of all the excrementitious products, organic as well as inorganic, whose elimination is the function of the kidney, the vicarious action of the bowels and skin must be promptly and vigorously induced. Freichs, believing that carbonate of ammonia is the real poison in these cases, recommends the neutralization of the free ammonia by chlorine, to be inhaled in the state of gas, or taken internally in water, along with the vegetable acids, the body at the same time to be sponged in vinegar, and vinegar enemata to be given. The spasms are best controlled by chloroform, but when they occur only as breaks in a continuously comatose condition, chloroform affords no prospect of relief. Bloodletting as a rule is inadmissible; its employment under the circumstances is irrational, and nearly always hastens the fatal end. Dr. Roberts on this point says: "I can only conceive of two contingencies in which withdrawal of blood is justifiable. . . . One is, where coma comes on rapidly in a person whose constitution is not, as yet, seriously deteriorated, and whose prospects of life (abstracting the uræmia) may extend to many months or some years; the other is, when there is a necessity for temporary restoration of the faculties paramount to the general chance of prolonging life" (p. 374 Am. Ed.).]

## DIABETES.

LATIN Eq., *Diabetes*; FRENCH Eq., *Diabète*; GERMAN Eq., *Diabetes*—Syn., *Harnruhr*; ITALIAN Eq., *Diabete*.

**Definition.**—*A constitutional disease obviously produced through error in the processes of assimilation, either in the stomach, in solid organs, in the blood, and characterized especially by an excessive discharge of urine more or less constantly saccharine, excessive thirst, and associated with progressive emaciation of the body.*

**Pathology.**—From the time of Charles II of England, when Dr. Thomas Willis first observed the saccharine character of diabetic urine, no disease has had its nature more inquisitively examined, and with more interesting and instructive results. The abnormal state of the urine naturally at first led the inquiry towards the kidneys. They have been industriously examined, both as to their structural and functional relations, but without elucidating the nature of the change in the urine. Dr. Matthew Doison, of Liverpool, in 1779, first established by experiment the fact that the sweetness was due to the presence of sugar. The next step in the inquiry was the detection of sugar in the blood of the diabetic patient. Ambrosiani, of Milan, in 1835, and Dr. Charles Maitland, in 1836, obtained crystals of pure sugar from the serum of the blood, and a large portion of fermentable crystallizable syrup. The late Dr. Robert Macgregor, of Glasgow, in 1837, confirmed these observations, by experiments, followed by those of Dr. G. O. Rees, of London, and Dr. Christison, of Edinburgh. Thus inquiry regarding the essential character of the disease was removed from the urine and the kidney to the blood; and research took a new direction, so as to ascertain, if possible, the source of the sugar in the blood and in the urine.

Although this disease has hitherto found a nosological place under disease of the kidneys, the researches of Bernard, Parkes, Pavy, Ringer, and others, very clearly show that amongst them it is misplaced. If the disease is to be regarded as a local one, it should rather come under *hepatic* than *nephritic diseases*; but the weight of evidence clearly shows that *diabetes mellitus* belongs to the *constitutional* class of diseases.

The unusual discharge of urine in this malady was originally ascribed by Mead to a morbid state of the liver and bile; but subsequently nutritive and assimilative functions connected with the digestive canal were considered by Cullen, Home, and Dobson to give rise to the morbid state. That the process of digestion and assimilation in the stomach was the source of the evil has been hitherto the prevailing theory regarding the nature of this disease. The belief received confirmation especially by the ingenious experiments of the late Dr. Robert Macgregor, of Glasgow, who ascertained that sugar was found in the stomach of diabetic patients during the process of chymification, even when no saccharine matter had been swallowed. Sugar has now also been detected in the

saliva, the sweat, and in the stools. To the ingenious M. Claude Bernard, of Paris, the science of medicine is indebted for the elucidation of the nature of the normal generation of sugar in the animal economy (glycogenesis); and thus we have been provided with data for the comparison and study of its morbid generation in *melituria*. He has shown us that one of the natural functions of the liver is to generate sugar, and thus the sagacious speculations of Mead, which referred the phenomena of diabetes to a morbid state of the liver and bile, are now not only curious, but interesting and instructive. While our knowledge regarding the formation of sugar by the liver, and its physiological relations to the animal economy in health and in disease, have been especially illustrated by Bernard, in France, it is to be observed that Dr. George Harley, of University College, and Dr. Pavy of Guy's Hospital, London, have confirmed many points, and especially elucidated the interesting doctrines of Bernard. Much is still unsettled as to the significance of the phenomena; but the following statements will convey a summary of the present position of the inquiry regarding *glycogenesis*, as set forth in the *Medico-Chirurgical Review* for January, 1857.

From the experiments just referred to, the fact was considered as an established one, that the food, and more especially its amylaceous and saccharine elements, had the power of forming sugar, which then passed into the blood and urine from the alimentary canal. The experiments of Bernard and others, however, have demonstrated that the animal organism has the power of forming sugar altogether irrespective of the nature of the food; and that sugar exists in a certain part of the circulation—namely, from the hepatic veins to the pulmonary capillaries—both in carnivorous and herbivorous animals. The liver is found to be this sugar-producing organ; and it is the only organ of the body which, in the normal state, is found to be impregnated with sugar—a fact established by Bernard, by observations on a large number of animals in almost every department of the zoological series. In man he examined especially the livers of five executed criminals, also that of a man who was killed by a gunshot wound, and that of a diabetic patient who died suddenly from pulmonary apoplexy. In four of these he determined the absolute and the relative amount of sugar in the liver. The total weight of the three healthy livers was 4205 grammes, which yielded of sugar a total of 66.074 grammes; so that the average weight of each liver being 1401.2 grammes, the weight of sugar yielded was 22.037 grammes, to compare with the liver of the diabetic case, which weighed 2500 grammes and yielded 57.50 grammes of sugar. He shows that the relative quantity of sugar varies little when the system is in a normal condition, very seldom exceeding 4 per cent. He especially demonstrated that sugar was secreted in the liver, and entered the blood from that organ; for by a comparative analysis of the blood of the portal vein as it enters, and the blood of the hepatic veins as they emerge from the liver, he found sugar in the latter, but not in the former. Sugar is not only found in the liver of adult animals, but it has

been found in the livers of the human foetus, the foetal calf, and unhatched chick, thus proving that it does not merely accumulate there as a product from the digestive canal.

Further, it has been determined that the sugar is undergoing perpetual destruction and renovation. It can be observed to disappear, and its further formation may be prevented under the following conditions: (1.) By causing an animal to die slowly by starvation, or by dividing the pneumogastric nerve; (2.) When the function of the liver is disturbed by severe, and especially by acute diseases.

The following is the course described by Bernard as that taken by the sugar secreted in health by the hepatic cells:

“It passes with the blood of the capillaries into the hepatic veins, thence into the *vena cava ascendens*. It is at the point of discharge from the last-named vessel that the blood is the most strongly saccharine, then becomes mixed with the blood from the lower parts of the liver, and passes up to the right auricle, where the sugar undergoes a dilution from its admixture with the blood of the *vena cava descendens*. From the right auricle it passes into the right ventricle, and then into the lung. In the whole of the route from the liver to the lung the blood is constantly saccharine, but the amount of sugar varies extremely, being least at the greatest distance from the liver. In the lung the sugar is being brought into contact with the air, and mixing with the whole of the blood, sometimes completely disappears.

“These two organs, then,—the liver and the lung,—stand in an intimate relation to one another, in so far as the saccharine matter is concerned. In a fasting animal, for example, the blood which arrives at the liver contains no trace of sugar, while that which leaves it is distinctly saccharine. Inversely, the blood which arrives at the lung contains sugar, while that which leaves it contains no traces of this constituent. The sugar in this physiological state remains hidden between the liver and the lung, and this is the reason why its existence and formation within the animal were not earlier discovered. The analysis of blood drawn from superficial veins would fail to detect it under these conditions” (*Medical Review*, p. 32, January, 1857).

But under some conditions even in health, and therefore called “physiological conditions,” sugar may be found in the blood before it reaches the lungs. The activity of the glycogenic function being increased with an augmented flow of blood to the liver, such as that which takes place after a meal, it is found that *four* or *five* hours after the commencement of intestinal digestion the production of sugar by the liver attains its maximum. For three or four hours at this time the production of sugar exceeds its destruction, so that during this period of digestion we find sugar in all the vessels of the liver, in both arteries and veins; and we find it in the renal arteries, but in too small a quantity to pass into the urine. This active and increased flow of blood through the liver thus displaces the sugar previously found there, and projects it into the circulation, and at the same time acts as a stimulus to the liver, which is still further excited by the nervous system under the influence of digestion.

increase of sugar is thus altogether independent of the nature of the food."

The cases of so-called "intermittent diabetes" are partly explainable by this interesting observation, in which the urine of digestion is saccharine, while no sugar can be detected at other periods, as in old persons observed by Bence Jones (*Med.-Chir. Trans.*, vol. xxxvi, p. 401).

The influence of diet is thus far remarkable, that the formation of sugar diminishes when fatty kinds of food are used—a fact supposed to be explained by the circumstance that the fats are absorbed directly by the lacteals, and thus do not affect the portal blood. It is also observed that in health the ingestion of amylaceous or saccharine matter does not augment the quantity of sugar in the liver, nor in the animal economy generally, although in cases of diabetes the use of these saccharine substances commonly causes a great and immediate augmentation of the sugar in the urine.

It does not yet appear clear how the sugar is destroyed in the blood in health. The destruction has been ascribed—(1.) To the oxidation or combustion of sugar in the lungs; (2.) To its combustion effected through the agency of an alkali; (3.) To the influence of extreme division in the blood, through which it may be converted into lactic acid by a simple molecular change.

Connected with the formation of sugar in the liver, Bernard shows that two substances are concerned,—(1.) One soluble in water; (2.) A substance slightly soluble in water, and which remains fixed in the hepatic tissue after all blood and sugar have been removed by prolonged washing. While, therefore, Bernard's observations showed that the blood which left the liver by the hepatic vein contains a peculiar substance of a saccharine nature, which does not exist in the blood brought to the organ in a state of health by the portal vein, the more recent researches of Dr. Pavy, of Guy's Hospital, have shown that the liver does not actually form sugar, but a substance that becomes sugar almost immediately on coming in contact with albuminous matters. "It is especially destined as a *pabulum* or fuel for the combustion process, being usually eliminated from the blood in the form of carbonic acid and water during its passage through the lungs, so as not to pass into the systemic circulation unless either its quantity be unusually great or its elimination interfered with by imperfect respiration. It appears to be elaborated by the converting power of the liver, either from materials supplied by the food or from the products of the waste of the system" (Carpenter, p. 308, *Animal Physiology*, Bohn's edition).

Connecting all these observations together—namely, Parkes, Lehmann, Bernard, Pavy, Basham—may it not be suggested, as a topic of investigation, whether or not the elaboration of this material by the liver is not arrested in Bright's disease and increased in diabetes, in the active stage of which disease it ultimately comes to be eliminated by the kidney as sugar in the urine?

The ultimate result of Bernard's investigations shows that the increased formation of sugar by the liver, and its presence in the blood, is the result of some exciting cause, which, acting by reflex



action, conveys the stimulus to the *medulla oblongata*, whence it is propagated by the spinal cord and filaments of the great sympathetic nerve to the liver, and so excites its *glycogenic* function. This doctrine is supported by the following experiment of Bernard:

“When we prick the mesial line of the floor of the fourth ventricle at the exact centre of the space between the origins of the auditory and pneumogastric nerves, we at the same time produce an exaggeration of the hepatic (saccharine) and of the renal secretions; if the puncture be effected a little higher, we very often only produce an augmentation of the quantity of the urine, which then frequently becomes charged with albuminous matters; while, if the puncture be below the indicated point, the discharge of sugar alone is observed, and the urine remains turbid and scanty. Hence it appears that we may distinguish two points, the inferior corresponds to the secretion of the liver, and the superior to that of the kidneys. As, however, these two points are very near to one another, it often happens that, if the instrument enters obliquely, they are simultaneously wounded, and the animal's urine not only becomes superabundant, but at the same time saccharine” (*Me Chir. Review*, l. c., p. 42).

He found also that in cutting the pneumogastric nerves the secretion of sugar was stopped, but that it still took place when the floor of the fourth ventricle was irritated, after the division of the pneumogastric. Any irritation conveyed through this nerve may thus induce the diabetic state. The following are the general circumstances under which the phenomena of *melituria* become developed: Any agents or conditions which cause a suspension of the functions of animal life, while the purely nutritive or organic functions remain intact, may bring about *melituria*. Thus the Indian *worari* poison acts; so does apoplexy produced by a blow on the skull. Local irritation of the liver itself, as Dr. Harley has shown, may also induce the condition. By the injection of alcohol and ether into the *vena portæ*, Harley was able to induce diabetes. The internal use of arsenic and quinine has also been said to have induced saccharine urine; and thus it is not improbable that irritant substances absorbed from the bowels by the mesenteric veins may sometimes bring about the morbid state.

[Of the *morbid anatomy* of diabetes we literally know nothing. There is no characteristic lesion. No constant appreciable changes in the liver have been found. Microscopical investigations have been equally unsatisfactory. Of the bile, Dr. Pavy says, in nearly all the cases where he examined it, its appearance was striking, resembling a rhubarb mixture, and depositing a copious sediment, consisting of columnar epithelium, and yellow, amorphous, granular-looking matter. The floor of the fourth ventricle has been examined, but without constant results, it sometimes being diseased and sometimes healthy.

Of the secondary lesions, those of the lungs are most frequent: of 64 fatal cases collected by Griesinger, there was tubercle in 31, or nearly one-half. Pavy and Wilks do not believe the pulmonary lesion to be genuine tuberculization, but a sort of chronic inflammation, by which the lung-tissue is broken down and cavities formed; but more evidence is wanted to confirm their views. Pulmonic congestion and consolidation are not infrequent; and gangrene of the lungs is sometimes met with.



In the 64 post-mortem examinations recorded by Griesinger, the kidneys were diseased in 32; the lesions were various. The mucous membrane of the stomach is said to be generally thickened and softened.]

**Symptoms.**—The early symptoms of *diabetes mellitus* are obscure, [the onset being insidious and unobserved, a sense of general discomfort, some emaciation, constant thirst, and increased micturition, being the only noticeable symptoms]. Dr. Prout believed that there is a stage which precedes the formation of sugar, and which is marked by a superabundant and highly dense urine, loaded with an excess of urea. But much uncertainty prevails on this point, and nothing is assured except that the constitution is not greatly affected till the saccharine matter forms, and is eliminated by urine. In some very few instances the quantity of urine passed is hardly greater than in health, but more commonly it is in great excess, amounting to eight, ten, sixteen, thirty, and even more pints during the twenty-four hours, so that the patient is incessantly disturbed in the night, and loses his sleep, while the urethra and its orifice become inflamed and sore. [The urine is of a pale straw-color, with a peculiar bright look, and its density varies from 1035 to 1040 or more.]

At this period the general health begins to give way; thirst is intense, and the patient often drinks many quarts, or even gallons, in the course of the day [without relief to the tormenting thirst]. The quantity of water drunk has been believed to be less than the quantity of urine passed,—in some instances only as one to four; but this statement, however, is not now believed to be correct. From the careful experiments of Dr. Parkes and others to determine this point, it appears that the quantity of urine is actually less than the quantity of water taken in liquid and solid food; and this is more especially seen to be the case if long periods (ten or twelve days) are taken for examination. Water is often retained for some time in the body of diabetics; and if an observation be made in a single day, it may happen that some of the urine retained from the previous day is then poured out. It is such retention of water which may account for the apparent excess of urine over drink (Parkes *On the Urine*, p. 339). [The mental faculties are sluggish]; the appetite is capricious [though generally excessive and voracious]; the skin harsh and dry [and scurfy; the mouth is parched and clammy; the tongue glazed and cracked], and the patient becomes greatly emaciated, and loses sexual desire and sexual power.

[Irritation of the urethra in the male, and pruritus vulvæ in the female, are not infrequent. Emaciation is progressive, and not limited to the fatty tissues, but the muscles become atrophied, and even the heart is said to suffer. Some persons retain a fair amount of flesh, until the disease has made considerable progress. The persistent dryness of the skin, so constant and so much complained of, is, on the appearance of fever, apt to give way to copious perspirations. The digestive organs, after a while, become feeble; the inordinate appetite diminishes, and is replaced by loathing of food; there is frequent nausea; sinking at the pit of the

stomach is felt; the bowels are constipated, and the fæces pale. A dry cough torments the patient, and physical examination shows tubercles in the lungs; colliquative diarrhœa, and irritative fever set in: the extremities become œdematous; the general health fails rapidly, and the patient sinks suddenly, from exhaustion, or coma; or convulsions supervene, or some acute intercurrent visceral disorder destroys life.]

Almost all the water drunk passes off by the kidneys, and insensible perspiration is diminished both by skin and lungs. The intestinal excretion of water is also greatly lessened; hence the bowels are costive and the fæces dry and hard. The water is passed off by the kidneys so soon as in health. If a diabetic patient drink water in the morning, the urine may not be increased till midday; but if grape-sugar be added to breakfast, the urine is passed as rapidly as in health. In advanced cases the drain on the constitution is so great that the alveolar processes are absorbed and the teeth, loosened in their sockets, are apt to fall out. The symptoms are much relieved by medicine, and life much prolonged; but often, when the case appears most favorable, tuberculosis comes apparent, and the patient sinks under this disease.

[Diabetes is chronic in its progress, commonly lasting from one to ten years, though it may run on for six, eight, or ten years. Becquerel mentions the case of a boy, nine years old, who died in six days, and Rollet a child of three years, who died in three weeks. Of 100 fatal cases collected by Griesinger, the duration was:

Under 8 months,	.	.	.	.	.	.	.	.	.	1
Between 8 and 6 months,	.	.	.	.	.	.	.	.	.	2
“ 6 “ 12 “	.	.	.	.	.	.	.	.	.	13
“ 1 “ 2 years,	.	.	.	.	.	.	.	.	.	39
“ 2 “ 3 “	.	.	.	.	.	.	.	.	.	20
“ 3 “ 4 “	.	.	.	.	.	.	.	.	.	7
“ 4 “ 5 “	.	.	.	.	.	.	.	.	.	2
“ 5 “ 6 “	.	.	.	.	.	.	.	.	.	1
“ 6 “ 7 “	.	.	.	.	.	.	.	.	.	2
“ 7 “ 8 “	.	.	.	.	.	.	.	.	.	1
Undetermined,	.	.	.	.	.	.	.	.	.	12

The most common *complication* is tuberculosis, it occurring in one of the cases of diabetes protracted to the first year (ROBERTS). It is often quite acute. “A low and fatal type of inflammation of the pleura, or peritoneum, is not infrequent, after the disease has lasted some time.” In every tissue of the body there exists a tendency to pyothenic inflammation, apt to run into abscess, diffuse suppuration, sloughing, phagedenic ulceration or gangrene. Boils, in successive crops, or even carbuncle, may appear in the course of the disease. In one of Roberts’s cases, boils were an initial symptom. Spontaneous gangrene of the lower extremities, with obstruction of one or more arteries of a limb, is not infrequent in diabetes (MARCHAL DE CALVI). Deafness or blindness is met with in a certain number of diabetic patients. Cataract occurs in some long-standing cases; its frequency is variously stated. In 225 cases collected by Griesinger, there was cataract in 20, but in his own 7 cases, it was present in 3; von Gräfe states the proportion at 1 in 5; Bouchardat, 1 in 38; of 45 cases treated by Roberts, there was cataract in only 1; and Garrod says that in the large number of cases

diabetes he has treated, he has never once seen it. Diabetic cataract is nearly always soft, though examples of the hard kind have been reported by Wilde, von Gräfe, and Guersaut. Roberts thus describes it: "It comes on generally after the diabetic state has lasted eighteen months or two years; but it has been known to appear in six months. Its course is rapid; the two eyes may become completely cataractous in a few days; sometimes it is developed more slowly." It begins in one eye,—generally the right,—but soon involves the two. Drs. S. Weir Mitchell, of Philadelphia, and B. W. Richardson, of London, have endeavored to show that it was due to physical imbibition by the lens of the saccharine matter of the aqueous humor; but there is no proof of this. Hepp and Fischer both failed to find sugar in cataractous lens removed from diabetic subjects, and there are other reasons against the reception of this theory, particularly the infrequency of its occurrence, the long delay of its appearance, and its being occasionally unilocular.

Dimness of sight, from supposed functional disorder of some of the internal structures of the eye (amblyopia) happens, according to Boucharlat, in about one-fifth of the cases of diabetes. Fauconneau Dufresne found the sight more or less affected in 20 out of 162 cases. Generally it is slight, temporary, and often recurrent. It is rarely a permanent affection, and when so, ends in total blindness, and betokens a speedy fatal termination. In such cases the retina is said to be atrophied.\*]

[The Urine.]—When *diuresis* is considerable, the urine should invariably be examined, and its constituents determined. A faint sweetish odor may be perceptible in diabetic urine, comparable to fresh hay or milk. The chamber-vessel should be examined for crystals of sugar which may have been formed (Fig. 2). The best evidence, however, is that derived from chemical tests.

A portion of urine, which is usually of a light-straw color, should be taken, and its specific gravity determined; and if greater than 1.020, it should be evaporated, and if sugar be present, we shall have a dark-brown residue, something like treacle. This extract, like the natural sugars, consists of crystallizable matter and of an uncrystallizable syrup; and to separate them, Dr. Christison recommends that the extract be agitated with rectified spirit, and the residue boiled in another portion of the same fluid, when, on cooling, the crystallizable sugar will separate in light-grayish grains like grape-sugar. Again, if sugar should be suspected to exist, even in minute quantity, a small portion of yeast should be added to a small quantity of the urine, when, if sugar be present, fermentation will ensue, and each

FIG. 2†



\* [De l'Amblyopie Diabétique, Gaz. Heb., Nov., 1861.]

† Crystals of diabetic sugar from diabetic urine (after BEALE, p. 160).

square inch of carbonic acid given off corresponds nearly to a grain of sugar. This test is so delicate that one part of diabetic urine, according to Dr. Christison, may be detected in 1000 parts of urine of the density of 1.030. The most certain and approved test is that known as "*Trommer's test*," based on the reaction of the salts of copper. A portion of urine in a test-tube is to be treated with *one or two* drops of a solution of *sulphate of copper*, and afterwards a considerable excess of *potash* is to be added. The dark blue solution which results is then to be held over the spirit-lamp and boiled for a moment, when a yellowish-brown *precipitate* of the suboxide of copper is produced. Dr. Roberts expresses a want of certainty in the results of most of those tests. He recommends Fehling's copper solution as the best test. It consists of Sulphate of Copper, gr. viij; Tartrate of Potash, 3ss.; Liquor Potassæ, ℥. Some of this test solution is to be boiled, and some drops of the suspected urine added to it. If sugar be abundant, a thick yellowish opacity and deposit of yellow suboxide are produced, which changes to a brick-red at once if the blue color of the test remain. If no such reaction ensues, more urine is to be added, until a quantity equal to the bulk of the test employed has been poured in. The whole must be again heated to the boiling-point; and if no change occurs, it is to be set aside without further boiling. As the mixture of the urine and test cools, if no milkiness is produced, the urine may be confidently pronounced free from sugar. No quantity *above a fortieth of a grain* can escape such a test, and any quantity below that does not appear to be of clinical importance. It is important to bear in mind that Fehling's test solution must always be freshly prepared. For the application of further tests see Dr. Beale's Tables, p. 21.

The density of diabetic urine, however, is one of the best indications. This fluid varies in density from 1.30 to 1.074 (BECQUEREL); but on an average, and tolerably constantly, it is 1.040; and when the urinometer stands above 1.030, we may suspect that sugar is present. The quantity of sugar present has been calculated by Dr. Henry, in urine of density 1.020, to be 402 grains in every pint, while at 1.050 it contains 958 grains of sugar—the increment being, as he conceives, one scruple, or nearly so, for every degree of specific gravity between the extremes that have been mentioned. If these data be correct, a person passing sixteen pints of urine daily, of specific gravity 1.050, actually passes nearly two pounds avoirdupois of sugar.

[In two cases reported by Dr. Austin Flint, in which quantitative analyses of the urine, with reference to the amount of sugar, were made, the results were—(1.) Quantity of urine voided in 24 hours, 155 ounces, 6 drachms; sp. gr. 1.035; quantity of sugar, 12 ounces, 413.07 grains; in one ounce, 39.82 grains; quantity of urea, 343.627 grains. (2.) Quantity of urine in 24 hours, 89 ounces; sp. gr. 1.037; quantity of sugar, 6 ounces, 356.16 grains; in one ounce, 36.363 grains (*Practice of Medicine*, 1867).]

But the amount varies greatly, amounting sometimes to *one* pound, or *two* pounds, or even two and a half pounds, in twenty-

four hours. In a few months patients will pass their own weight in sugar (PARKES, *l. c.*). Its amount is mainly influenced, in the first instance, by the saccharine and amylaceous nature of the food, which always augments the amount of sugar. The augmentation is quite perceptible about two hours after food, and continues for four or six hours, if the amount of starch taken has been considerable. It is probable, though it is not absolutely proven, that all the starch eaten is converted into sugar; and in cases accurately observed by Mr. Graham and Dr. Parkes for a considerable length of time, the quantity of sugar excreted by the urine never exceeded the amount of starch; and almost all the starchy food was accounted for by the diabetic sugar (WALSHE, PARKES). It has been supposed by some that when the starch only is converted into sugar, it is the earliest stage of the diabetic state; and it is certain that during the progress of some cases sugar begins to be formed from other sources and in other ways, and is no longer derived solely from the starch.

In some cases the sugar may recognize no other origin than the starchy food for many years. Such seems to have been the case of the late Mr. Camplin, of Finsbury Square, who kept his disease at bay for ten or twelve years (*Med.-Chir. Trans.*, vol. xxxviii, p. 69; PARKES, *l. c.*) Complete fasting or abstinence from saccharine and starchy food for eight or twelve hours reduces the quantity of urine to the normal amount, and brings down the specific gravity to the usual figure. Sugar can then neither be detected in the urine nor in the blood, as was determined by Dr. Parkes after numerous analyses of blood under such circumstances (*On the Urine*, p. 348).

In the majority of advanced cases of diabetes, sugar is not only produced from starch, but also from *nitrogenous* foods, especially *gluten* and *animal food*. In such cases complete abstinence for a time from food lessens, but does not entirely remove, the sugar from the urine. Perhaps in all such cases the formation of sugar from albuminous food indicates a more advanced or active stage of the disease than when the sugar is formed from starchy compounds only; and it is probable that the amount of sugar from this source increases as the disease advances. Dr. Parkes refers to a case related by Schultze, in which the amount of sugar in the urine when the patient was on mixed diet was *one-third* more than could have been furnished by the starch food alone; and the amount can always be ascertained by keeping the patient strictly on a meat diet, and feeding him with starch from time to time as an experiment (*l. c.*, p. 348).

Thus three conditions affecting food would seem to be present in cases of diabetes:

The earliest and fundamental one continues to act throughout, and is distinguished by complete arrest to the normal metamorphic changes of starch and sugar—an arrest probably associated with some substance in the digestive canal which absorbs the sugar. At first it affects the sugar derived from starch food, then it affects the sugar derived from nitrogenous food. Dr. Bence Jones suggests



that in some cases the changes may pass one step beyond the conversion of starch into sugar, and produce "vegetable acid." I believe a disease exists which is thus allied to diabetes, and characterized by a great amount of acid, probably *lactic*, in the system and in the urine. The chief transitional changes of starch enumerated by Dr. Bence Jones are as follows; Starch into sugar, into vegetable acid, into carbonic acid; and he supposes the arrest to occur at the acid stage, instead of at the sugar stage, in the morbid condition he describes.

The second condition is an abnormal production (probably in the liver) of sugar from nitrogenous food—*i. e.*, an amount of production which is abnormal—combined with arrest in the transformation of sugar normally formed (PARKES). A diabetic patient, kept on the most rigorous meat diet, has been known to pass a quantity of sugar corresponding to *two-fifths* of the entire meat food, or *three-fifths* of the *albuminate* contained in the meat (GRIESNER, quote by PARKES).

A third condition may be also distinguished—namely, one in which the *tissues* themselves, and especially the muscles of the body, contribute to the morbid formation of sugar. This occurs under circumstances of extreme inanition, when almost no food is being supplied to the body, and yet a constant quantity of sugar is eliminated. Traube, Parkes, and Ringer have each recorded such cases. In one of Dr. Parkes's cases the blood, after seventeen hours fasting, was found to be still very rich in sugar (PARKES, *l. c.*, p. 350). It is, however, a question whether this is an abnormal formation of sugar merely, or a further and more advanced stage of the disease. There are also cases which may be noticed here, in which the patients continue to lose ground although the quantity of sugar lessens, and in which the substance known as *inosite* or *muscle-sugar* is found in the urine (Fig. 3). It increases in quantity

FIG. 3.\*



as the sugar lessens, and at last as much as eighteen and twenty grammes of pure *inosite* have been procured from the day's urine. The *inosite* may be obtained in the form of colorless prismatic crystals, which are efflorescent. It does not reduce the oxide of copper to the state of suboxide, as is the case with *diabetic sugar* and *grape-sugar*; and it is said to have not quite the same composition as the latter substance, but is represented by  $C_6H_{12}O_6$ ; so that one atom of *grape-sugar* would thus represent six atoms of *inosite*. It may be detected by evaporation of the suspected fluid nearly to dryness in a platinum basin, when,

\* *Inosite* or *muscle-sugar*, crystallized partly from alcohol and partly from water (after FUNKE).



if a little ammonia and chloride of calcium be added, a rose color is produced, especially if the mixture be again concentrated by evaporation. M. Hohl records a case of diabetes in which, while the proportion of sugar gradually diminished, the *inosite* gradually increased in amount till upwards of 300 grains were passed in the twenty-four hours (PARKES, BEALE). The observation is one of great interest in connection with the pathology of this remarkable constitutional disease.

Professor Sidney Ringer, of University College, has made some observations of great interest, to show the amount of *urea* and of *sugar* respectively furnished by the tissues of the body and by nitrogenous food. (1.) During inanition one series of observations showed an enormous disintegration of tissues (48 grammes of urea and 105 grammes of sugar being passed in twenty-four hours), the relation between the urea and the sugar being tolerably constant. In the second series, when nitrogenous food was taken, the urea increased about the third hour after food, and reached its maximum about the fifth hour, after which it continued to diminish, and reached the inanition amount in the eighth hour. The sugar followed the same rule, and almost in an exact ratio; but the urea was in slight relative excess to the sugar, showing that the nitrogenous food raised the urea slightly more than it did the sugar. During inanition the urea was to sugar as 1 to 2.235, while after nitrogenous food the urea was to sugar as 1 to 1.9. It thus appears certain, as Dr. Parkes observes, that there is some close connection between the amounts of urea and of sugar in such cases as diabetes. The amount of urea may be double or even treble the normal amount, and that to an extent much greater than can be accounted for by the food taken, and due probably to some peculiarity in diabetes, causing heightened metamorphosis of tissues, such as might arise from the excessive action of oxygen on them (PARKES, *l. c.*, p. 342). The amount of sugar is still further increased if diabetic patients take more water than their thirst demands; and it is probable that the urea is also increased, although exact experiments are wanting.

On standing, diabetic urine soon begins to ferment, with the appearance of *lactic*, *butyric*, *acetic*, or *formic acids*, and develops the yeast plant; and during this fermentation the urea entirely decomposes. Dr. Christison gives the following formula for ascertaining the amount of solid matter in *diabetic urine*: "Multiply the excess of the specific gravity over 1.000 by 2.33, the result is the number of parts of solid matter in 1.000 of urine."

[**Etiology.**—It would appear, from the Registrar-General's Report for England and Wales, for the decade 1851–60, that of 4546 persons who died of diabetes, of a mean population of 19,000,000, 3032 were males and 1514 females, the excess of males being 50 per cent. Up to the age of puberty, the two sexes appear to be about equally liable to the disorder, but from that period to old age, the greater liability of the male is shown in a regularly increasing ratio, as will be seen by the following:

TABLE SHOWING THE NUMBER OF DEATHS FROM DIABETES IN THE TWO SEXES AT DIFFERENT PERIODS OF LIFE (ROBERTS).

PERIODS OF LIFE.	Under 5 yrs.	5-10 yrs.	10-15 yrs.	15-25 yrs.	25-35 yrs.	35-45 yrs.	45-55 yrs.	55-65 yrs.	65-75 yrs.	75 yrs. & upwards.	All ages
Deaths in males, . . .	28	40	97	378	468	502	550	500	364	105	308
Deaths in females, . .	28	42	78	220	282	261	247	191	144	26	161
Total males and females,	51	82	175	598	750	763	797	691	508	181	454

The age of the youngest diabetic patient that Dr. Bence Jones has known was three and a half years. A case in an infant of twenty months is recorded by Dr. J. L. Brown, in the *Am. Journal of Obstetrics, &c.* May, 1868.

The inhabitants of towns and of manufacturing districts would appear to be more liable to diabetes than those who live in rural districts. Hereditary influence appears to be small, though there are instances where the disorder has run in families.

Jaksch believes that diabetes sometimes is the result of syphiloma of the brain, the syphilitic process affecting the floor of the fourth ventricle, and causing symptomatic diabetes in the same way as it was artificially produced by Bernard in his experiments already referred to.

The *exciting causes* are very obscure. Among them have been named, exposure to wet and cold, drinking largely of cold fluids while the body is overheated, excessive use of saccharine and amylaceous articles of food, abuse of alcoholic drinks, and violent mental emotions.]

**Prognosis.**—The ultimate issue of every case of diabetes is probably fatal; at least the number of cases in which the urine is rendered permanently natural is extremely small, and many of them, at the moment the disease seems to have yielded, die of phthisis. Even when the presence of the saccharine principle has been so far conquered that it alternates with lithic acid deposit, or that lithic acid becomes the prominent feature, the circumstance is anything but favorable, for such individuals generally die of some sudden and overwhelming attack of internal inflammation or of apoplexy (PROUT). On the other hand, where the source and nature of the mal-assimilation can be discovered, and either rectified or held in check, and if the patient will submit to a regimen, a favorable result may be hoped for in some cases.

[On this point Dr. Roberts observes: "A not inconsiderable number, however, recover completely, and many more attain to a state of conditional amelioration—that is, an amelioration which is conditional on the observance of a certain diet and regimen." Dr. Trousseau says: "By hygiene and a proper regimen, aided by the action of certain remedies, wisely and prudently used, we may hope to cure a few and to relieve a large number of diabetic patients, if the disease has not reached the last period." The younger the patient the less is the chance of ultimate re-

covery. Dr. Roberts states, that all the cases under twenty which he has seen, have eventually died of the disease. In all persons the symptoms may continue for years, with a tolerable state of health. In corpulent persons the prognosis is more favorable than in those of spare habit. Saccharine urine, without excessive urinary secretion, is less serious than when the secretion is very great. "*Cæteris paribus*, the longer the disease has existed, the more unfavorable the prognosis; *cæteris paribus*, also, the greater the general severity of the symptoms, the less is the hope of amendment. Cases which can be traced to mental anxiety and traumatic lesions appear to be somewhat more hopeful than those for which no tangible cause can be assigned" (ROBERTS, *loc. cit.*, p. 193). Great diminution in the amount of, or the total disappearance of sugar in the urine, when saccharine and amylaceous articles of diet are not used, is a favorable sign; also a soft, perspirable skin with a moderate appetite, or the symptoms generally remaining stationary. Albuminuria, thoracic and intestinal complications, and permanent cataract or amblyopia, are always fatal.]

**Treatment.**—There are few diseases in which the treatment has been more varied than in *diabetes mellitus*. Every conceivable medicine has been given, but exact quantitative determinations of the effect of them on the sugar have rarely been made. The emaciated state of the patient presents an insurmountable obstacle to bleeding. Nevertheless, this mode of treatment has often been practised, and as much as 160 to 170 ounces of blood have been taken in a few weeks. The late Sir Henry Marsh found bloodletting of service when the disease was recent, and the strength of the patient still maintained. He found that it promoted the action of diaphoretic remedies; and especially when followed by the tepid bath. He found leeches to the epigastric region of benefit, when internal heat with fulness and tenderness prevailed there, with a "gnawing" feeling about the stomach. In the face, however, of so high an authority, the pathology of diabetes, as given in the text, does not sanction general bloodletting as a mode of cure.

*Mercury*, alike with *lead*, *antimony*, *zinc*, *silver*, and *copper*, are indifferent as to their influence on the formation of sugar (PARKES). Opium has been given to the extent of 100 grains in the twenty-four hours; but with an equal want of success, although it does seem to lessen the sugar, probably by lessening the appetite and hindering the taking of food (PARKES). The whole *materia medica* has been exhausted in search of a remedy for this disease. The metals, the fixed and the volatile alkalies, the vegetable and mineral acids; the astringents, purgatives, tonics, diaphoretics, and diuretics, have in their turns been administered, and each has perhaps afforded some relief; but the disease has proceeded, and finally, it may be said, nearly every patient dies whose treatment is left entirely to drugs. Dr. Prout, who considered it merely as a form of dyspepsia, conceived that each case requires a different treatment. In the early stages of the disease the Drs. Bullar, of Southampton, have found great benefit from the *tincture* of the *muriate* of iron, now called the *tincture of the perchloride of iron*. Dr. Camplin speaks of the *citrate of ammonia* in the effervescent form, generally com-

bined with the *citrate* of iron, as more useful than any other medicine; while bitters and alkaline remedies did him great service one period of his attack. [The citrate of soda may be given in half-drachm doses.]

Opium combined with ipecacuanha is eminently useful as a sedative, especially in the form of Dover's powder; while exercise, warm clothing, friction of the surface, hot bathing, and diaphoretics, improve the cutaneous functions. The functions of the skin must always be inquired into, and must be kept active. The nitro-muriatic baths, and the internal administration of the acid, might also be employed with some prospect of benefit in cases requiring acid tonics. This is more generally the case in the young, and in the early stages of the disease, in which the *tincture of the perchloride of iron* is also of service, prescribed in the *infusion of quassia* or of *calumba*. But each case requires to be made a special study, considering that many, or at least several, organs, may be concerned in the disordered working of the system (PROUT, CAMPLIN). The *mis-tura ferri composita* is another of the iron preparations which have been found of service. The administration of alkalies, as recommended by Miahle and Contour, is also occasionally successful.

[In cases where it is suspected that the disorder results from cerebral disease, a seton at the nucha may be of service.]

The little benefit derived from medicine induced Dr. Rollo to try the effects of an entirely azoted or animal diet; and out of nineteen cases, two are said to have been cured by this means. A full and generous diet is unquestionably useful in these cases; but the patient soon gets disgusted with mutton or beef, or both, for breakfast, dinner, and supper: he consequently nauseates a meat diet, and abandons it altogether. A diet of salt fish has been attempted; but the patient in a short time so loathes it that it has to be given up. A mixed diet, therefore, if contraindicated by some theories, is at least the best to adopt in practice, and is consistent with the remarks made under Pathology, if duly regulated and aided by other means. It will be evident, however, that those vegetables which contain a large quantity of saccharine matter should be avoided in some degree, as potatoes, grapes, or other very ripe fruit, and, *à fortiori*, sugar itself.

"There are, therefore," as Dr. Camplin justly observed, "certain fixed broad principles upon which the disease is to be treated." In all cases the various influences of the stomach, liver, skin, and kidneys on the nervous system and on each other ought to be sought out and determined, and the basis of treatment arranged accordingly. *It is necessary to abstain from all amylaceous food, as well as from every solid and liquid containing sugar, or any substance readily convertible into sugar.* Fat meat and eggs may be taken, if biliary derangement is not induced by them, and fish is a most important article with which to vary the monotony of the dietary. Milk also may be indulged in occasionally, as it is not found that the sugar it contains is readily converted into glucose. Its influence, however, requires watching.

[Dr. Roberts made the following trial of milk, in a girl, with confirmed diabetes. The patient continued to gain weight, and improved in general condition, though the density of the urine and the excretion of sugar somewhat increased.

	Average daily Quantity of Urine.	Average Quantity of Sugar daily excreted.	Increase of Weight.
Meat diet and bran cakes, for four weeks, . . . . . }	55 oz.	897 grains.	5 lbs.
Meat diet, bran cakes, and three pints of milk, for four weeks, . . . . . }	49 oz.	1260 grains.	5 lbs.
Meat diet, gluten bread, and cabbage, for three weeks, . }	41 oz.	1020 grains.	7 lbs.]

It is desirable to vary the food as much as possible during the day, taking the lighter kinds in the later meals. When soups are taken, they ought to be really good, and flavored with aromatics or onions, to the exclusion of carrots, turnips, and pease. They may be thickened with some bran *finely powdered*. Lettuces Dr. Camplin found to agree well, when eaten sparingly with oil and vinegar, or with a little salt only, if the vinegar is likely to disagree. Pickles in small quantities may be permitted to convalescents. If cocoa agree, it may be taken prepared *from the nibs only*. [Tea and coffee, without sugar, may be permitted.] With regard to drinks: if milk is found to agree, it may be used as a drink, combined with half its bulk of lime-water, or in the form of what is known as "buttermilk" in Scotland and in Ireland, but which in England is generally given to pigs, not yet being sufficiently appreciated by natives of the country south of the Tweed. Dr. Camplin eventually found it necessary to abstain from all alcoholic drinks; but, where they are found desirable or necessary, a selection may be made from those wines and spirits which are freest from sugars. Of these, clarets may be chemically considered the best, then Burgundy. The so-called "fruity wines" must be entirely interdicted, and of all alcoholic beverages *weak* brandy and water is the safest. The amount of brandy must be always *measured* and taken as directed by the medical attendant. A *teaspoonful* in a tumblerful of water is generally sufficient for an ordinary dinner drink; and Dr. Camplin candidly and feelingly observes, from his own experience, that no diabetic need expect to recover or continue well who cannot exercise self-control, and make up his mind to be temperate *in all things*.

Seeing that under this system of diet the patient is deprived of the use of ordinary bread, Dr. Camplin devised a form of bread prepared solely from *bran*; and the great value of *bran cakes* as a substitute for bread in cases of *diabetes* has now been established by the



experience of so many individuals that its use ought to be insisted on. The bran used should be *thoroughly washed*, so that it may be as free from starch as possible; and *finely powdered*, so that it may not irritate the susceptible mucous membrane of the intestine. Such carefully-prepared and finely-powdered bran may be obtained from Mr. Batchley, of 362 Oxford Street, London, near the Patheon; also of Mr. Donges, Gower Street, London, North. But it is desirable to prepare the powder at home as it is required, special mill and sieve for this purpose are necessary (Dr. Camplin *Monograph on Diabetes*).

The *formula* for bran cakes is thus given by him: "Take a quantity of wheat bran (say a quart). Boil it in two successive waters for a quarter of an hour, each time straining it through a sieve; wash it well with cold water on the sieve, until the water runs off perfectly clear. Squeeze the washed bran in a cloth, as dry as possible, then spread it thinly on a dish, and place it in a slow oven. When it is perfectly dry and crisp it is fit for grinding into fine powder.

"The bran thus prepared is ground in the mill for the purpose, and must be sifted through a wire sieve of such fineness as to require the use of a brush to pass it through, and what remains on the sieve must be re-ground till it is sufficiently soft and fine.

"To prepare a cake, take of this bran powder three or four ounces, three new-laid eggs, one and a half or two ounces of butter, and about half a pint of milk. Mix the eggs with a little of the milk, and warm the butter with the remainder of the milk; stir the whole well together, adding a little nutmeg and ginger, or any other spice that may be agreeable. Bake in small tins (pattipans, which must be well buttered), in a rather quick oven, for about half an hour. The cakes when baked should be a little thicker than a captain's biscuit.

"These cakes may be eaten with meat or cheese for breakfast, dinner, and supper, and require a free allowance of butter; and the cakes are more pleasant if placed in the oven a few minutes before being placed on the table.

"When economy is an object, when a change is required, or if the stomach cannot bear butter, the cakes may be prepared as follows: Take of the prepared bran four ounces, three eggs, about twelve ounces of milk, with a little spice and salt, to be mixed and put into a basin (previously well buttered). Bake it for about an hour; the loaf may then be cut into convenient slices and toasted when wanted; or, after slicing, it may be re-baked, and kept in the form of rusks.

"Nothing has yet been discovered of equal utility to these bran cakes, combining, as they do, moderate cost with freedom from starch, and sufficient pleasantness as an article of food" (Camplin *On Diabetes*, third edition).

Consistently with the experiments of Bernard, "cod-liver oil holds out some prospects of a natural plan of treatment by its use."

Dr. Pavy recommends ground almond powder, made into biscuits, rusks, and bread, with eggs, as a substitute for ordinary bread. Mr. Hill, 60 Bishopsgate Street, London, makes such biscuit.

An abstinence from water lessens the formation of sugar; but it probably accumulates in the body, so that when fluid is again given, an excessive elimination of sugar occurs (RINGER, GRIESINGER); and



patients become extremely depressed and ill if water is withheld from them, probably from the impregnation of the body with sugar (PARKES). Coffee lessens the sugar, but increases the urea. Rennets, as recommended by Dr. Gray, of Glasgow, at first lessen the sugar and water; but they afterwards increase again. Warm baths lessen the amount slightly; so does bicarbonate of soda. Hence the recommendation of Miahle and Contour.

When the diabetic symptoms subside, congestions, especially of the head, are apt to supervene. Such congestion Dr. Camplin found to subside gradually under the use of *citrate of ammonia* and small doses of *colchicum wine*.

The great difficulty in the treatment of diabetes is to manage the dyspepsia and impaired digestion, and, at the same time, to diminish and keep in check the formation of sugar. Warm flannel ought to be worn next the skin in all cases, and residence in a warm climate will often be of service as an aid to the means of cure employed.

[However surely an exclusive animal diet may lessen, or entirely remove, the sugar in the urine of a diabetic patient, it is certain that it cannot long be tolerated. Under its use, the appetite fails, and a loathing of all food soon happens. The necessity of a mixed diet for man has been shown (vol. i, p. 731); and it is as necessary for him when suffering from diabetes as in a state of health. In this disease the nervous system is undoubtedly implicated, and a rigid adherence to animal food alone, were it practicable, would soon be followed by an aggravation of the nervous troubles. There is, from the very beginning, and throughout the course of the disorder, a strong tendency to devitalization, and this, too, must be guarded against. Fortunately the sugar in the urine may be kept down, and, at the same time, the general strength of the system maintained, by a properly adjusted diet of mixed food. Along then with the carnivorous dietary, whose importance is not to be undervalued, certain vegetables may be permitted, not only with impunity, but with advantage, and these are cabbage, cauliflowers, onions, spinach, water-cress, sorrel, endive, lettuce, &c. Trousseau has found no ill consequences from eating acid fruits, as strawberries, gooseberries, cherries, and he has allowed apples, pears, and grapes. He even suffers his diabetic patients to eat a small quantity of bread, if they greatly crave it, for, as he remarks, there are many persons who are unable to make a meal without it. Such a regimen is more likely to keep the disease stationary, and secure the general comfort of the patient, by upholding his strength, than by confining him to one kind of diet, which his stomach soon revolts against, and which must result in innutrition, general debility, the development of intercurrent affections, and, sooner or later, death. Enforced daily exercise in the open air, when possible, just short of fatigue, is as of much importance in the treatment of diabetes as diet. Gymnastics should be practised. Trousseau says that he has repeatedly seen, during the hunting season, diabetic patients, abroad with their gun and dog, cease both to drink and urinate to excess, and regain their strength, and even their virile powers. A suit of flannel or buckskin should be worn next the skin. Warm alkaline baths should be frequently taken, and an occasional Turkish bath will be found serviceable, if it produces no disposition to boils; or daily packing with the wet sheet, may be tried, carefully watching the effects. The whole body should be hand-rubbed daily. Cream,

so long as it agrees, may be permitted, and cod-liver oil is in many cases well borne, and would seem, as a nourisher, to do good. The alkaline theory has been proved to be false and absurd by Cl. Bernard and Poggiale, still the benefit of small doses of the alkalies, not continued, however, longer than a week or ten days at the time, is incontestable. They are best given in the form of the Vichy, Marienbad Kreutzbrun, or Carlsbad Spoudel water. Legroux thought he derived benefit from the administration of arsenic. Dr. Richardson, of London, has recommended the respiration of oxygen, and Béranger-Féraud has shown that during its use there is great diminution in the amount of sugar in the urine (*Bul. d. Thér.*, t. lxvii). The ethereal solution of the peroxide of hydrogen (ozone ether) has been given with asserted benefit in half drachm doses, in an ounce of distilled water. Dr. T. K. Chambers has prescribed iodide of potash on purely empirical grounds. On no pretext should any form of alcoholic drink be permitted. The preparations of iron are indispensable and should be varied from time to time. To lessen the thirst and craving for food, small doses of opium are useful. By the use of the mixed diet recommended, particularly if fatty articles be taken, the obstinate constipation, so common in the earlier stages of the disorder, is often obviated, without a recourse to drugs. Should this not be the case, castor oil in capsules, or rhubarb and podophyllin, must be prescribed.

This mode of treatment, which secures a proper but varied diet, one that will not disgust the patient, but will nourish his body, though it may not remove the symptom glycosuria, will, in a large number of cases, give the diabetic a fair share of conditional health, so that, as Dr. Trousseau remarks, he would not know he had sugar in his urine, if an occasional chemical analysis did not remind him of its presence.]

#### (SPASMODIC) ASTHMA.

LATIN Eq., *Asthma*; FRENCH Eq., *Asthme*; GERMAN Eq., *Asthma*; ITALIAN Eq., *Asma*.

**Definition.**—*A constitutional disease which culminates in paroxysmal attacks of difficult breathing, which are of longer or shorter duration. The dyspnœa seems to be immediately dependent on more or less extensive contraction of the smaller bronchi, and due to tonic spasm of their circular fibres. The breathing is accompanied by a wheezing sound, a sense of constriction in the thorax, great anxietas, and a difficult cough. The attack usually terminates by the expectoration of a quantity of mucus from the lungs, which varies considerably in appearance and in amount. In some instances the mucus is thick and heavy, in others it is light and frothy, whilst in the severer forms of the disease there may be only a few dark pellets coughed up before relief is obtained (PRIDHAM). In the hours immediately succeeding the fit a remarkable diminution of the urea and chloride of sodium may occur, which would imply a considerable arrest either of formation or elimination, probably the former (RINGER, PARKES); or to the starvation that is generally enforced at that time (SALTER).*

**Pathology.**—Few diseases have been the subject of greater doubts and differences of opinion as to its nature than asthma. Not unfrequently it has been confounded with dyspnœa; and the terms *dyspnœa*, *asthma*, and *orthopnœa* were formerly employed to desig-

nate different degrees of difficulty of breathing. Their signification must now be much more precisely defined. *Dyspnœa* is a term which is now used to denote difficulty of breathing generally, and may be due to various causes. The significance of *asthma* is defined above, and its pathology is about to be considered; while the term *orthopnœa* signifies that great difficulty of breathing in which the patient is incapable of respiring except in the erect posture.

When asthma has once expressed itself, it seldom fails of recurring, though the intervals between the paroxysms are of very uncertain duration. In severe cases the fits will return periodically every ten days or a fortnight, and in still more severe cases they will recur every night or early morning, at exactly the same hour.

It has been observed to recur in females just after the menstrual discharge, or immediately before it. It is also apt to recur in the spring and autumn, and after exposure to cold and wet. The disease is not only paroxysmal, but often periodic; by days, weeks, months, or even years. Diurnal asthma is very common, especially when associated with chronic bronchitis, heart disease, and impaired digestion. Asthma occurring once a year is usually winter asthma complicated with bronchitis.

The stomach and bowels are extremely liable to disorder in asthmatic persons; colic-like pains, flatulence, loss of appetite, and an irregular state of the bowels, are not uncommon.

There appears to be no period of life at which asthma may not make its appearance—from the earliest infancy to old age. In thirty-eight cases noted by Dr. Hyde Salter, the first access of the paroxysm occurred in *seven* during the first year of life. In one of these, symptoms of asthma were noticed at *fourteen days* old; in another at *twenty-eight days*; in another at *three months*; in another at *one year*; and in three “during the first year.” Many of the best-marked and purest asthmatic cases date from early infancy—so early in some that it were difficult to say the disease was not truly congenital. It is evidently very often dependent upon hereditary transmission and conformation; and in all such cases of its early development there was a history of its inheritance (HYDE SALTER). Mr. T. L. Pridham, of Bideford, has traced its hereditary origin in nine out of ten cases, and relates a case of asthma which commenced as early as seven years of age. Out of *thirty-five* cases in which Dr. Salter has noted this circumstance, he found distinct traces of inheritance in *fourteen*. The melancholic temperaments, the sanguineo-melancholic, the nervous, and the irritable are most liable to the affection; and the male sex is much more disposed to it than the female (WOOD). According to the experience of Mr. Pridham, about 80 per cent. of the cases are men.

It is believed by not a few to be connected with the gouty or rheumatic diathesis. The disposition of the attacks to recur at distant but gradually diminishing intervals; the division of each attack into nightly paroxysms, with marked remissions during the day; the duration of the earliest fits for several days or a week, are all circumstances which point to the constitutional nature of

the affection. Many of the cases recorded by Mr. Pridham inherited gout; and in some instances both diseases could be traced in the family; and in more than one instance where gout and asthma had prevailed in previous generations there were alternate attacks of asthma and of gout. He found that when women were the subjects of asthma, gout prevailed in their families in a larger proportion than in men.

The best descriptions of the phenomena of asthma have been given by medical men who have themselves suffered from it—for example, Dr. Bree and Sir John Floyer, both of whom were asthmatics (Good). Asthmatic patients generally live to a good old age. The lungs undergo dilatation of the air-cells, which dilatation does not much interfere with their normal action when free from attack. It produces emphysema in the same way that bronchitis does, and leads to hypertrophy and dilatation of the right side of the heart. In those cases of asthma which have been described as dyspeptic the powers of digestion are insufficient to assimilate the food taken, and such patients can never with impunity eat and drink as other people do; and wherever the disease has occurred in any member of a family for one, two, or even three generations back such exciting causes as imprudence in eating or drinking, an attack of bronchitis or influenza, atmospheric influences, certain odors, mental excitement, and the like, may at any time or period of life bring to light this peculiar disease; but unless in constitutions predisposed to the disease, such exciting causes have no influence upon its development. Asthmatic people, for the most part, are said to be gifted with great energy, and talents far beyond the lot of ordinary people. They are generally courageous and resolute; and those in humble life possess intellectual attainments far beyond their station; and all generally excel in whatever subject of study they are disposed to follow (PRIDHAM). The experience of Dr. Hyde Salter, however, does not bear out this statement. A knowledge of the state of the blood and of the physiology of digestion, embracing a knowledge of the amount of the urinary and other excreta, and especially of the peculiar expectoration, in relation to diet and temperature, is of great importance to be known in cases of asthma. Dr. Sidney Ringer, the Professor of Therapeutics in University College, has made one series of accurate observations relative to the urinary excreta in a case of asthma. He found in the hours immediately succeeding the fit that a remarkable diminution of the urea and chloride of sodium occurred, which implied a considerable arrest either of formation or of elimination, probably the former (PARKES, *l. c.*, p. 319). The urine at the commencement of an attack is generally abundant and colorless, like what is known by the name of "nervous urine;" later in the attack it is often extremely dark and scanty, and is sometimes almost suppressed (HYDE SALTER).

Mr. Pridham observed in one of his cases that the urine was suspended during an attack, on the subsidence of which there occurred an enormous secretion of it, of a dark color, and loaded with lithates.

Dr. Hyde Salter has clearly shown that the dyspnoea of the asthmatic paroxysm is due to spasmodic contraction of the bronchial tubes; that the phenomena of the paroxysm are in a great measure those of an excitomotory kind—in other words, due to reflex action.

The exciting causes of the paroxysms are mainly due to fatigue and physical exhaustion—sudden or violent mental emotion—certain conditions of the digestive organs—gastric irritation—the irritation of a loaded rectum—irritation of an eruption on the skin and its sudden subsidence—the irritation of certain substances and articles of food, such as cheese, nuts, almonds, and raisins, sweet things generally, salted meats, condiments, preserved and highly seasoned foods, fermented liquors, especially malt liquors, and sweet wines.

**The Forms of Asthma** have been variously described under the following names: *peptic* or *dyspeptic asthma*, *congestive asthma*, *hay asthma*, *hysteric asthma*, and *spasmodic asthma*; but seeing that all true asthma is spasmodic, the classification proposed by Dr. Hyde Salter is perhaps the best, inasmuch as it suggests the necessity for examining into the various influences which act on the nervous system in each case, and enables the physician to arrange the basis of treatment accordingly. It is as follows:

**Class I.** Those cases in which the provocatives of the attack are manifest, in which the lungs seem to be mainly concerned, the source of irritation being applied to them, as some material respired which provokes the bronchial tubes to spasms by direct contact with their mucous surface (*e. g.*, Asthma from fog, smoke; fumes of various kinds, *Ipecacuanha powder*, or that of hay; from animal emanations; from certain atmospheres; and, lastly, from blood poisoning, as after beer, wine, and sweets).

**Class II.** Those cases which acknowledge a reflex source of their development. Of these, five varieties are easily indicated: (1.) Those in which the asthma follows an error in diet, or supervenes on a full meal; (2.) Those in which the source of irritation is transmitted from a loaded rectum or from uterine irritation; (3.) Those in which it arises from the sudden application of cold; (4.) Emotional asthma; (5.) Periodic asthma.

**Class III.** Asthma complicating bronchitis, heart disease, or pulmonary emphysema.

On inspecting the chest of a patient laboring under a severe paroxysm of asthma, the whole upper part seems almost motionless, while the inferior portions are acting within a very confined range. All the muscles passing from the head to the shoulders, clavicles, and ribs are rigid. The abdominal muscles, however, act most powerfully to increase the capacity of the chest, and its walls are kept fixed in a condition of extreme inspiration. The chest is enlarged in every way, the diaphragm descends, the abdomen seems fuller, and its girth is increased. The stethoscope teaches us that the whole of the lungs, but particularly the posterior lungs, are laboring with a loud and deep sibilous, sonorous wheeze, accompanied with a mucous rattle, sometimes loudest on inspiration and some-



times on expiration. No respiratory murmur exists. Dry tubular sounds alone are heard—rhonchus and sibilus of every variety of note, and pitch. There is complete stagnation of air in the chest. The sounds are so small that they seem to indicate spasmodic constriction of the smaller tubes; and the universal diffusion of the sound shows that the constrictions are universal over the small tubes. These spasms may also be observed to be constantly changing their place, disappearing in one place and making their appearance in another, so that the sounds are continually changing the character and their site. Percussion shows that the lungs are distended with air; and should an air-cell have burst, a rubbing sound will be heard, denoting the effusion of air into the cellular substance of the lung. As the fit subsides, the respiration becomes puerile and by degrees the breathing returns to its usual state. In fatal cases the respiration becomes tracheal, slight hemorrhage takes place, and after a severe struggle the patient dies; but this is an event so extremely rare that, from Dr. Hyde Salter's very extensive experience of this disease, he believes death never takes place immediately from uncomplicated asthma.

The duration of the fit varies. In some cases it lasts a few minutes, in others two or three hours, in others the whole night, in others three or four days, and in others as many weeks.

When the expressions of the disease are fully marked, the appearance of an asthmatic person is very characteristic. The countenance often bears the signs of distress. The shoulders are more or less elevated. The stomach is apt to be greatly distended after eating; the tongue becomes coated, and there is a tendency to fissures in it; the eyes red and prominent. Emaciation generally progresses, and there is inability to rest horizontally in bed, or to walk up a hill. The secretions from the bowels are more or less abnormal, and the urine passed generally shows a variable deposit. In extremely severe cases, when remedial measures are not taken to subdue the constitutional affection, the nights may be passed by the patient in a state of great distress. Unable to lie down in bed, his paroxysm may sometimes be so severe that he almost anticipates death before morning—were it not that he becomes accustomed to the severe nature of the symptoms—till a copious heavy expectoration is with difficulty thrown off from the lungs; and as the day advances he becomes somewhat relieved, although it may be passed still in great discomfort. Asthmatic patients are generally large feeders, although the desire for food may not be remarkable, believing that it is necessary to eat and drink well, in order to sustain the strength to encounter the paroxysms of the disease. In some the difficulty of breathing is constant, and always worst after a meal; and, as a rule, they can never, without aggravation of the disease, eat and drink as other people.

The pulse increases in frequency towards night, and subsides in the morning.

Many premonitory phenomena indicate the approach of a paroxysm, but the precursory symptoms are liable to great variation in different persons, according to the proximate cause of the par-



oxysm. Some patients suffer from fearful headaches, the approach of which they dread; and the heart labors with so much palpitation, and such irregularity of action, that rupture of a bloodvessel seems imminent. Eruptions on the skin sometimes lessen, and even disappear. There may be also some warning during the night of the immediate approach of an attack, in the shape of huskiness of the throat; and during the middle of the night, or towards early morning, the patient is awakened by an oppression which renders it impossible for him to lie down again. In a short time the paroxysm gains strength, and the patient breathes as it were by jerks, each aspiration being accompanied by a spasmodic effort, which seems as though it would burst open the chest. The contractions of the muscles in the neck and below the ribs in front of the chest are very great, and often, at the same time, most painful. The attacks are not of equal violence at all times.

The majority of asthmatics know that an attack is coming on, by certain feelings in themselves, or by certain conditions of the system, of which they are aware. The precursory symptoms generally show themselves on the night previous to the attack, or sometimes for two nights before it, or even for a longer time. Extreme drowsiness and sleepiness are common precursory phenomena which indicate the approach of a paroxysm—the commencement of the nervous condition of which the succeeding respiratory phenomena are the more complete development; and such precursory phenomena must be looked upon as an integral part of the paroxysm (HYDE SALTER).

Extreme wakefulness, unusual mental activity, and buoyancy of spirits, constitute another set of premonitory phenomena seen in some other asthmatics; and Dr. Salter mentions a case in which ophthalmia always ushered in the paroxysm of asthma. At other times, and with other patients, the premonitory symptoms are connected with the stomach, and consist especially of loss of appetite, flatulence, costiveness, and certain peculiar uneasy sensations in the epigastrium.

The time, according to Dr. Salter, at which a paroxysm commences, is almost invariably in the early morning, from three to six o'clock. In some the usual time is the evening, just after getting into bed, and before going to sleep. Even in such cases as night watchmen, who turn day into night and night into day, Dr. Salter relates that, though the ordinary times of sleeping and waking were transposed, the paroxysm came on at the usual time—from five to six in the morning, towards the end of the vigil, when the patient was up and awake.

Profuse *diuresis* not unfrequently attends the first stage of a paroxysm of asthma. The urine is then a pale limpid water, exactly like the urine of hysteria. This abundant watery secretion comes on soon after the paroxysm commences, and generally lasts for the first three or four hours, when it ceases altogether.

*Neuralgic* pain constitutes another early symptom, in the form of deep-seated, aching, constant, and wearing pains in the limbs, joints, or testicles.

The characteristic wheezing of a commencing paroxysm generally commences while the patient is yet asleep; and as the difficulty of breathing increases, he gradually or partially awakes, sits up in bed "in a miserable half-consciousness of his condition. A temporary abatement occurs, and sleep may again overtake him, to be again awoken, and again to sit up. By and by the struggle ceases between sleep and the full expression of the paroxysm. The *dyspnœa* increases, so that he can lie back no more. He throws himself forwards, plants his elbows on his knees, with fixed head and elevated shoulder, labors for his breath like a dying man. A most distressing spectacle is now presented by the asthmatic. If he moves at all, it is with the greatest difficulty, creeping by stages from one piece of furniture to another. Most commonly he sits fixed in a chair, immovable, unable to speak, even perhaps to move his head in answer to questions. His back is rounded, his gait stooping. His chest, back, shoulders, and arms are fixed; and, when he looks from object to object, he merely turns his eyes like a person with a stiff neck. His shoulders are raised almost to his ears, and his head thrown back and buried between them. The better to raise his shoulders, and to spare muscular effort, he fixes his elbows on the arms of the chair; or he plants his hands on his knees; or he leans forward on a table; or sits across a chair, and leans over the back of it; or he stands grasping the back of a chair, and throwing his weight upon it. In this latter attitude Dr. Salter has known a patient stand for two days and nights, unable to move. Sometimes the patient may lean against a corner of drawers or some piece of furniture sufficiently high to rest his elbows upon in a standing position. At every breath the head is thrown back, the shoulders still more raised, and the mouth a little opened, with a gasping movement. The expression is anxious and distressed. The eyes are wide opened, strained, turgid, suffused. The face is pallid; and if the *dyspnœa* is extremely prolonged, it becomes slightly cyanotic. The labor of breathing is so great that beads of perspiration stand on the forehead, and even run down in drops upon the face, which the attendant constantly wipe off, for the patient is so engrossed with his struggles and the labor of breathing that he is almost unconscious of what is going on around him; or he is impatient and intolerant of the assiduities of those who are in vain trying to give him relief.

If the bronchial spasm is protracted and intense, the temperature falls: the oxygenation of the blood is so imperfectly formed, from the sparing supply of air, that it is inadequate to the maintenance of the normal temperature. The extremities especially get cold, blue, and shrunken; but while the temperature is thus depressed the perspiration may be profuse. This union of coldness and sweat, combined with the duskiness and pallor of the skin, gives to the asthmatic so much the appearance of a dead man that sometimes even the initiated may fear that death is pending.

The pulse during the paroxysm is always small; and small

feeble in proportion to the intensity of the dyspnoea—due to pulmonary capillary arrest; and immediately the paroxysm yields, the pulse begins to resume its normal volume.

Itching under the chin is a common symptom of an approaching paroxysm of asthma. This itching is incessant, of an indefinite creeping character, and scratching does not relieve it. It often extends over the sternum and between the shoulders. It appears the moment the first tightness of breathing is felt, and subsides when the paroxysm has become confirmed.

The most exhaustive monograph on this disease is that by Dr. Hyde Salter, F.R.S., Physician to the Charing Cross Hospital; and the reader desirous of more full details will do well to consult his work on *Asthma; its Pathology and Treatment*.

**Diagnosis.**—The preceding account of the pathology and symptoms of asthma renders it obvious that it may not easily be confounded with any disease of the chest or larynx, if auscultation and percussion are carefully attended to, and with a due regard to the history of the case. The sudden attacks of the paroxysms, the short periods of their duration, the violence of the symptoms at the time, their returning after intervals of comparative ease and of tolerable health, are sufficient to characterize the disease.

It is only when asthma complicates other diseases that its diagnosis may be obscure and its treatment uncertain.

The diseases with which it has been confounded are: (1.) Spasmodic affections of the larynx; (2.) Severe cases of sudden and *acute bronchitis*; (3.) *Angina pectoris*; (4.) *Hydrothorax*.

The disease is also sometimes associated with the development of lesions of the heart and great vessels, and ultimately leads to them.

The character of the *dyspnoea in asthma* is also quite peculiar. It is unlike the dyspnoea of heart disease, or of that of bronchitis, or of that of emphysema; the distinctive features of each of which are as follow (SALTER):

*Heart dyspnoea* is intolerant of the slightest exertion, or of the recumbent position, and sitting up or stillness may cure for the time the most violent paroxysm: the breathing, too, of heart dyspnoea has a panting and gasping character, and not the wheezing, laboring character of asthma.

*Bronchitic dyspnoea* is short, crepitous, and accompanied with cough.

The *dyspnoea of asthma* is often long-drawn, dry, and without cough; it gives the most positive evidence of narrowing of the air-passages, and is of such a nature as to shut off the air-supply. The wheezing, or shrill sibilant whistle, is positive evidence of bronchial contraction, which is ever changing its place. Spasmodic stricture of the minute air-tubes thus explains, as Dr. Hyde Salter clearly demonstrates, the sudden access and departure of the dyspnoea in asthma.

The *dyspnoea of emphysema* is abiding, varies but little, and has no wheeze.

The Treatment of Asthma comprises what should be done during

the fit, and what should be done during the intervals, with a view to correct the constitutional state which every now and then terminates in a paroxysm of asthma.

When the patient is laboring under a fit of either of the forms of asthma, our efforts must be directed to tranquillize his sufferings and to shorten the attack; but so capricious is this disease that what will benefit the patient in one attack will be of little use in another. As a general rule, however, any exciting cause actually present and in operation must be removed: an undigested meal, constipation, must be got rid of—relieved by an emetic in the one case, or by an enema in the other; the patient should be supported by a strictly tonic regimen; and *camphor mixture*, to the extent of about an ounce and a half, combined with a drachm of the *spirit of nitrous ether* and some *morphia*, may be given every hour, or even two hours, for a short time. If the head should be affected by the use of opium, some milder narcotic should be substituted, as *tincture of hyoscyamus*, to the extent of about fifteen drops for each dose. In other cases, or in other attacks, *asafoetida*, *castor*, *musk*, or *hydrocyanic acid*, to the extent of ℥iij every six hours, may be substituted. Again, if the fit should occur after a hearty meal, and after an emetic has been given to empty the stomach, the *tincture of rhubarb* or the *sulphate of magnesia* should be continued in repeated small doses. If the attack be long, arrow-root or sago, with small quantities of wine or brandy, should be given to support the patient under his laborious and exhausting sufferings. *Ipecacuanha*, *tartar emetic*, and *tobacco*, are the drugs which most rapidly relax spasms as direct depressants. There is, however, great danger in the use of the latter, from unmanageable and dangerous collapse. And tobacco ought never to be indulged in by the asthmatic, except as an agent in the cure of his disease; for then only can he look to it for relief. *Ipecacuanha* is the most manageable of these remedies and ought to be given in a dose of twenty grains at the onset of the paroxysm. The tobacco should be smoked from a pipe.

The feelings of the sufferer should be consulted as to the temperature to which he should be exposed during the paroxysm. Where there is organic lesion of the heart and large vessels, the fresh air is extremely grateful and reviving, its coldness giving power to the circulating organs, and, by lowering the temperature of the body, enables the patient to live on a smaller quantity of oxygen. It is on this principle that the dog, barbarously asphyxiated by the effluvium of the Grotto del Cane, for the amusement of travellers, is thrown into the water, where he is able to breathe at the temperature of the water, when he would have died at the temperature of the atmosphere. The toad, also, when cooled down, will live for an incredible length of time incased in plaster of Paris; but if his body has a high temperature the experiment is soon fatal. On the contrary, when the paroxysm is purely a spasm of the bronchial tubes, warmth, by relaxing the spasm of the bronchial tubes at their ultimate divisions, is generally more useful than cold.

But it is the treatment during the interval which is all-important; so much so, that few cases will be found of true spasmodic

asthma which are not entirely under the control of well-regulated dietetic management. "More is to be done for *asthmatic patients* on the side of the stomach than in any other direction;" and by many observing and thoughtful physicians dietetic treatment is regarded as the only certain treatment of asthma. Mr. Pridham, of Bideford, in Devonshire, has been very successful in the management of asthmatic cases, by extremely strict dietetic treatment and sedatives during the intervals of the paroxysms (*Brit. Med. Journal*, June 9 to December 29, 1860). His plan of treatment is somewhat as follows: The secretions from the bowels are first of all to be corrected by the following pill at bedtime, followed by a saline aperient in the morning:

R. Pilulæ Aloes cum Myrrhâ, gr. iij; Pilulæ Hydrargyri, gr. j; Extracti Taraxaci, gr. ij; Extracti Stramonii, gr. ss. M. Fiant pil. ij.

Or giving every alternate night, in the form of a pill,—

R. Pil. Hyd., gr. iv; Pulv. Ipecac., gr. j.

And on the following morning,—

Mist. Sennæ comp., ℥j; Bicarbonatis Magnesiae, gr. x; Bicarbonatis Sodæ, gr. viij; and during the day small doses of Compound Rhubarb Powder.

After having thus attended to the general secretions for about ten days, the strict dietary system is to be commenced.

The diet must be regularly weighed out, and adhered to with the greatest strictness, the hours of meals being most rigidly fixed as follows:

*Breakfast* at eight A.M., to consist of half a pint of green tea or coffee, with a little cream, and two ounces of dry stale bread.

*Dinner* at one P.M., to consist of two ounces of fresh beef or mutton, without fat or skin, and two ounces of dry stale bread or well-boiled rice; three hours *after* dinner (not sooner) half a pint of weak brandy and water, or whiskey and water, or dry sherry and water, may be taken, or toast-water *ad libitum*.

*Supper* at seven P.M., to consist of two ounces of meat as before, with two ounces of dry stale bread.

The patient is not to be allowed to drink any fluid whatever within one hour *before* his dinner or supper, and not until three hours after either of these meals. At other times he is not limited as to drinks, otherwise than that all malt liquors are to be prohibited. Soda or Seltzer-water may be indulged in at other times when thirsty.

With this dietetic treatment sedatives are to be given as follows:

Three grains of the Extract of Conium are to be taken four times a day—namely, at the hours of seven, twelve, five, and ten,—the dose to be gradually increased to five grains four times a day. To each of these pills a *fourth* of a grain of the Extract of Indian Hemp may be added, which may be gradually increased to one grain in each dose.



Under this treatment in a few days the distressing symptom may be expected to subside; and after the regimen has been strictly persevered in for at least a month, two ounces more of meat may be permitted, if digestion is found to be sufficient. The stools may be repeatedly seen by the physician, and the stomach must not have more to do than it can accomplish. The powers of digestion are known to be recovering when the stomach craves for food as the hour of nourishment arrives. If flesh is gained, strength improved and while the tongue cleans, the appetite improves, the distension of the stomach lessens, and there is sufficient evidence that the powers of digestion are recovering. Great encouragement is then given for the physician and for the patient to follow up the line of treatment which is here indicated. The patient ought also to be able to sleep six or seven hours at a time, and to lie in bed all night. If these results follow, the ultimate cure of the disease may be looked for; but it may at the same time be taken for granted that the asthmatic can never with impunity eat and drink as other people. It is only by the exercise of such self-denial as is implied in the carrying out of such instructions that the patient has it in his own power to live a life of comparative ease and comfort. Many such patients who are not possessed of such resolution, self-denial and strength of mind, will say such dieting does not suit their constitution, and that consequently they cannot or will not persevere; but no trial of the remedy can be considered sufficient which does not embrace a period of at least six months, the physician taking care to ascertain—(1.) The weight of the patient, his age and height, before commencing any treatment; (2.) The state of his excreta, and the amount of the urinary elements—especially the urea, the uric acid, and the like; (3.) During the course of the treatment, at stated times, such physiological information ought to be regularly obtained.

It is confessedly difficult to persuade many people to live so abstemiously, for many cannot control their appetite; or they believe that in so limiting themselves in regard to diet they will injure their constitution.

The abnormal ravenousness of appetite it is therefore necessary to subdue by sedatives other than opiates. Asthmatics are generally dyspeptics; and as a result of the experience of Dr. H. Salter, the most simple rule regarding the diet is: Let no food be taken after such a time in the day as will allow digestion being completed or the stomach empty before going to bed. The time when the last solid food should be taken will depend upon what the bedtime is. If ten, or half-past, then three or four should be the dinner hour, after which no more solid food should be taken.

Dr. Salter's dietary in cases of asthma would be something as follows: *Breakfast*—A breakfast cup of bread and milk, an egg, or a mutton chop, or some cold chicken or game. Tea is better than coffee, and milk and water better than either. *Dinner* (not before two or four o'clock)—Mutton ought to be the staple diet; beef or lamb rarely, pork or veal never. Succulent vegetable or potato may be eaten, and a little farinaceous pudding or stewed



fruit, or fruit out of a tart, should conclude the dinner. Water is the best fluid to drink, and there should be no cheese and no dessert.

The quantity of food eaten should be small, and therefore highly nutritious, extremely digestible, and of the simplest and plainest kind.

Open air exercise must be freely taken, but not within three hours after eating animal food, and the exercise must be always short of fatigue. The greatest punctuality is necessary to be attended to as regards the taking of food as well as medicine; and the bowels should be caused to act immediately after breakfast, either naturally or by means of an enema. In truth, success greatly depends on the regularity with which all the functions of the body can be performed; and care should be taken to rest the body and mind at least for one hour after food. Smoking stramonium, the inhalation of chloroform, and the like, although they appear to soothe and mitigate the paroxysm in some cases, yet they do not appear to *shorten* the attack in any.

The fumes of *stramonium* are to be collected in an inverted glass bowl with a narrow mouth. The bowl being charged to its full, is placed under the mouth of the patient, who is directed to inhale, to the fullest extent in his power, the smoke which has been collected in the bowl. Or the stramonium may be smoked as tobacco is smoked, then puff the smoke into a tumbler, and inhale it cold into the lungs (BULLAR). Or it might be smoked as an Eastern smokes the Oriental hookah, in which the smoke is purified by being passed through water. Savory and Moore prepare cigars and cigarettes of stramonium. The fumes of brown paper saturated with a solution of nitrate of potash sometimes also relieve the spasms.

Indian hemp, in doses of from two to four grains of the extract, or thirty minims of the tincture, will often relieve the spasm for the time being, but may fail ever after (see Watson, *Lectures on the Practice of Physic*, vol. ii).

#### CANCER—Syn., MALIGNANT DISEASE.

**Definition.**—A disease inherent by birth, or otherwise inbred. Its presence and complete development are indicated by the occurrence of peculiar growths in different parts of the body, to which the names of cancers or malignant growths have been applied. The malignant character of cancers is indicated by one or more of the following conditions: Constant progress of the growths, their continuing to return after extirpation, not only in the original seat, but in the distant and internal parts of the body; destroying and causing absorption of the invaded textures; infiltrating the tissues to an indefinite extent; and tending to suppurate, and to affect the glands to which the lymphatics lead from the seat of local lesion. The increase and nutrition of these growths produce a peculiar marasmus, which tends to terminate in death. The anatomical constitution of the new growths is heterologous.—Defined more shortly as—A deposit or growth that tends to spread indefinitely into the surrounding structures

and in the course of the lymphatics of the part affected, and to reproduce itself in remote parts of the body.

**Pathology.**—The morbid condition of the body, the ill-health, cachexia, which is associated with cancers cannot be referred to the blood alone. Indeed, we have no direct proofs that the blood is peculiarly affected. We cannot find in it anything distinctive or peculiar to the cancerous diathesis, either by microscopic or chemical investigation. But while none can doubt the existence of a cancerous cachexia, we have no positive knowledge concerning the state of the blood in cancerous states of the body. It is rather inferred, from the various considerations, that a peculiar material is separated from the blood, and is constantly being renewed in the formation of cancers. There are no germs in the blood previous to the development of cancer-tumor which can be recognized as a cancer-structure. It is evident, however, that a specific state of the system exists in the cachexia associated with cancer,—(1.) Because all parts of the body are liable to be infiltrated with the peculiar and specific growths which constitute the “cancers or malignant tumors,” and that without any direct communication with the place where the first growth took place; (2.) Because the removal of the locally diseased part does not arrest the progress of the constitutional disease; and (3.) Because the cancer-growths tend naturally towards destruction of life, not necessarily from their local position, but through a peculiar marasmus which their existence establishes, and which some think due to the demands which these new growths make upon the system for increase and nutrition. They tend also to destruction of tissue around them, and to suppuration. A continual hectic febrile state is established, and increasing emaciation follows the intense bodily suffering and mental anxiety they induce. The countenance becomes pale and anxious, with a slight leaden hue, the features become pinched, and the lips and nostrils slightly livid. The pulse is frequent and the pains are severe. At length there is generally nausea and weakness of digestion, and a tickling cough frequently supervenes. Severe stitches strike through the local morbid parts, the pulse becomes rapid and faltering, the surface cadaverous, the breathing anxious, and death alone brings relief.

“Cancerous disease,” says Mr. Paget, “or a tendency to it, is prone to pass by inheritance from parent to offspring, and to occur (probably by inheritance of common properties) in many members of the same family and generation; but while there appears to be a tendency to the hereditary transmission both of cancerous and non-cancerous growths, there is a much greater probability of the hereditary transmission of *cancerous* than of *non-cancerous* growths, and that in the proportional probability of 22.4 for the *cancerous* and 8.2 for the *non-cancerous* (*Med. Times and Gazette*, Aug. 22, 1857).

There is abundant circumstantial evidence to show that malignant tumors are of *constitutional* origin—that they are local manifestations of a *constitutional* disease—that there is something specific, probably elaborated from the blood, which accumulates in their elements of structure. This specific material which composes

cancers is different from all the natural constituents of the body ; is different from all the materials formed in other processes of disease ; and is associated in the malignant tumor with structural elements which must be regarded as specific and peculiar, both in form and in mode of life. The main grounds for this belief are—(1.) The evidences, not always absolutely demonstrable, of a pre-existing unhealthy state of the constitution ; (2.) The prolonged duration of the constitutional state which precedes the local expression of the cancerous tumors—the long-continued elaboration, often extending through several generations—thus showing sometimes a tendency to alternation in the inheritance of the inborn state of ill-health ; (3.) Secondary pyrexia, which is the result of irritation or of elimination of a morbid material, the reabsorption of which may tend secondarily to affect the blood.

[Whether the cancerous cachexy is primary or secondary ; whether, under any circumstances, a true blood-cause exists, and what is the state of the blood in the cancerous constitution, are questions which at this day, with all the lights of modern pathology, and the investigations of Bennett, Lebert, Hannover, Paget, Rokitansky, Virchow, and others, are as much involved in obscurity as they were a century ago. Virchow, in his recent work on Tumors (*Die Krankhaften Geschwülste*, 1863–67), inclines to the opinion that the *dyscrasia* or *diathesis* theory is pure hypothesis, and will disappear in a more advanced state of knowledge of the subject ; and that, in the vast majority of cases, the change in the blood is to be regarded as a secondary phenomenon, due to the absorption of matter from an existing primary focus, the malignant growth being due to a local irritant. Mr. C. R. Moore, of the Middlesex Hospital, London, in a recent memoir on “The Antecedents of Cancer,” after stating the grounds on which cancer is held to be originally of constitutional nature, viz., *a.* Its final universal effusion throughout the body ; *b.* Its occasional commencement in many primary tumors simultaneously ; *c.* Its capacity to grow in various textures ; *d.* Its local recurrence after an operation on the primary tumor ; *e.* Its appearance in internal organs, notwithstanding the extirpation of the primary tumors ; *f.* Its repetition in families ; *g.* Its relation to tubercle—is of opinion that it arises as a local disorder, independently of a constitutional cause, assigning as evidences : 1. Its invariable origin as a single tumor ; 2. The manifest dependence of the later tumors upon the first. (This view is supported by the observed similarity of the morbid substance, in whatever organ or texture it may grow ; by the order regulating its dissemination ; by the interruption of the progress and dispersion of the disease, if the primary tumor be removed ; and by the possibility of extirpating that tumor by an early and adequately extensive operation.) 3. Because of the remarkable manner it is inherited as a local, and not as a constitutional, peculiarity—a disease of a corresponding organ of the plural members of one family ; whilst at the same time any inheritance of the disease is uncommon, and that by infants extremely rare ; 4. And because of its preference of the healthiest persons. He writes : “The general conclusion to which I am led by the foregoing considerations is, that cancer has no dependence on any malady anterior to the appearance of the first tumor, but that it originates in persons otherwise healthy and strong. If this conclusion is inconsistent with prevailing opinions as to the cause and nature of the disease, the collision of the

facts proves the need for more satisfactory evidence on behalf of the opinions than is at present in our possession. The existence of an antecedent general malady is, as far as I can perceive, pure conjecture, being entirely destitute of proof, or even of reasonable support. The idea sprang up in error; and it has been perpetuated mainly by the erroneous conclusions drawn from repeated want of success in surgical operations. Virchow (*loc. cit.*) shows that those organs which have a soft surface, and are most often in contact with irritant foreign substances, are more liable to be primarily the seat of cancer, than those organs which are inclosed and have no communication with external objects. He quotes, in support of this opinion, from large statistics, collected by Tanchou, Maud'Espine, and himself, to show that 80, 87, and 78 per cent. (according to these observers) of all cases of cancer occur in the alimentary canal, the uterus, and the breast, the large preponderance being of those in the alimentary canal. Virchow remarks, and with justness, that "the most marked and really demonstrable contamination of the blood with cancerous matters is, the more manifest is the relatively acute course of the process. . . . I shall have to examine this question more closely when I come to consider the theory of the propagation of malignant tumors, in the case of which recourse is so often had to the supposition that the malignancy has its root in the blood which gives rise to the local affections. And yet it is precisely in the course of these processes that it is comparatively most easy to show the mode of propagation, both in the immediate neighborhood of the diseased part and in remote organs; and it is in them we find that there is one circumstance which especially favors the extension of such processes, namely, the abundance of *parenchymatous juices* in the pathological formation" (*Cellular Pathology* Am. ed., p. 250-1). Nor should be forgotten, among the arguments for the local origin of malignant tumors, the fact of the rapid and great improvement of the general health after the extirpation of a cancerous growth, and which again begins to fail on its recurrence (from some of the germ-matter having been left behind), with, sometimes, a repetition of the same phenomena, after a second operation; to account for which, Mr. Paget is obliged to bring forward what he styles "the secondary cancerous cachexia." This great pathologist is a decided partisan of the hypothesis that cancer is the local manifestation of a disease which already, in its specific material, exists in the blood. "The existence of the morbid material in the blood, whether in the rudimental or effective state, constitutes the general predisposition to cancer. . . . The morbid material is the essential constituent of the 'cancerous diathesis or constitution;' and when its existence produces some manifest impairment of the general health, independently of the cancerous growth, it makes the primary cancerous cachexia" (*Lect. on Surg. Pathology*, 3d Am. ed., p. 669); and yet, as a practical surgeon, he admits, in his description of the disease, that the cancerous cachexia "may at first have been absent" (p. 553). Most unsatisfactory, if not unphilosophical, too, is his conclusion that "the reconciliation, not only of the two conflicting opinions, but of the seemingly conflicting facts upon which they chiefly rest, is to be found in this, that the complete manifestation of cancer, the formation of a cancerous growth, is suspended till such a time as finds both the constitutional and the local conditions coexistent—till the blood and the part are at once appropriate" (p. 669); words without practical meaning.]

It has not been proved that the inoculation of cancerous matter is followed by even the growth of cancers, far less by the develop-

ment of cancerous cachexia; and there is every reason to believe that this loathsome disease cannot be communicated by the secretions from a cancerous ulcer. Alibert has made dogs swallow the ichorous serosity collected from such a source, but the health of those animals was not impaired. Dupuytren has likewise introduced portions of cancerous parts into the stomachs of many animals—has injected the pus into their veins, and into their different serous cavities, but without producing results different from what any other irritating matter would have caused. Women, also, having the neck of the uterus destroyed by carcinoma, have conceived and borne children, yet neither the husband nor child have appeared to suffer in consequence. Alibert and others have likewise inoculated themselves with cancerous matter, and yet no contagious effect has followed. Neither has this disease at any time been known to result from accidents incident to the examination either of the living or the dead person so affected. The more recent experiments of Harley and Lawrence upon dogs have similarly failed to inoculate cancer. There are several cases known, however, in which inoculation of cancers having failed at the time, yet, nevertheless, the subjects of inoculation have died of cancer at an advanced period of life; but the value of the observation does not as yet extend beyond a mere coincidence.

Women appear to be more liable to this disease than men; and the increase is chiefly due to cancers of the breast and uterus, while in man it is chiefly the skin, the bones, and the digestive organs which suffer.

The mortality from the disease goes on steadily increasing with each successive decade until the eightieth year (WALSHE).

Age has also much influence in determining the forms of cancer and the part affected. Hard cancers are rarely observed till after forty, and from that period the liability increases with age. Its most usual seats are also those on which age and functional activity have left their marks upon the organs. Thus it seldom occurs in the mammæ, uterus, or in the ovaries, till after the cessation of menstruation, nor in the organs of generation of the male till towards old age, nor in the different portions of the alimentary canal till after forty.

Soft cancers are most common in the earlier periods of life; but then also they are observed to involve textures whose functional activity has been ever active, such as the glandular parts—for example, the lymphatic and lachrymal glands.

The cancerous cachexia seems to be particularly influenced by physical climate, mental distress, and anxiety; and according to Dr. Walshe the maximum amount of cancerous disease is found to occur in Europe, and is rare amongst the hospitals of Hobart Town and Calcutta, and the natives of Egypt, Algiers, Senegal, Arabia, and the tropical parts of America.

We know very little, however, as to the conditions which give rise to cancers. In the words of Mr. Paget: "The richest and the poorest alike seem to be subject to it; so do the worst and the best fed; those that are living in the best conditions of atmosphere



and those that are immured in the worst; those that are clean and those that are foul; those of all temperaments and of all occupations; those that appear healthy and those that are diseased. We can hardly lay our hand upon any one of the various circumstances of life, in the various orders of society in this country, which we can refer as rendering one more or less liable than another to the acquirement of the cancerous constitution."

The history of the development of cancers makes up some of the dark pages of Pathology; and in many respects the origin of such tumors is confessedly mysterious. Their first beginnings are generally hidden, being deeply buried in the tissues, so that when a swelling manifests the possible existence of a tumor, it is regarded with *doubt* in the first instance, and then by astonishment as well as dismay at the rapidity of its growth. In such doubt, astonishment, and dismay, we must recognize and acknowledge our ignorance.

The normal course of cancerous tumors we now know to be that of steady increase—a steady and certain progress towards death. No radical cure for cancer is yet known. The hand of death is most unmistakably stamped upon those who are the subjects of cancerous growths; and we seek, in all humility, to learn something from an examination of the history of such a disease. But, to learn anything from pathological growths, they require to be studied and examined *in stages*, from time to time, in the course of their development. There are *certain stages* in pathological as in physiological growths, at which periods alone the actual mode of origin and development can be traced. A large cancerous mass does not look very promising; and we might as well expect to be able to discover, by an examination of the mature foetus, the different steps by which its organs and parts had been formed, as hope to be able to determine in what way a tumor which at first was scarcely visible had been converted into a mass of obviously malignant growth. Thanks to Walshe, Bennett, Paget, Virchow, and many others, we are able to explain many of the phenomena of growth of such tumors, while there is much that is still dark and mysterious in their history.

It is during the time of active increase that tumors ought to be studied, having regard to their origin, development, growth, and maintenance as independent parts.

The idea that local cancers were due to entozoa, originally advanced by Adams and older writers, has never met with any proof, although it is yet entertained as probable by some; and there are many facts in the history of malignant tumors which indicate that their existence is attended with consequences very analogous to those of the existence of parasites. For—(1.) Every new formation which contributes to the body no serviceable structure must be regarded as a parasitical element in that body, alike foreign and injurious to it. (2.) The elements of malignant growths withdraw nutrition from the body, and in some instances elaborate specific secretions, to be stored up in their own structures. (3.) The malignant growth, whatever it may be, in the body or constitution, is



the result of long-continued elaboration, and passes through a life of continual change before it attains its highest degree of malignant influence on the constitution. The alternate generation of the malignant growths are also exemplified in the frequent hereditary transmission of the cancerous constitution. (4.) In active growth and partially independent existence we have another remarkable analogy to parasites. Malignant tumors grow and thrive while the normal tissues far and near them are only able to maintain existence.

The local exudations which constitute the tumors or cancers consist in general of two parts, namely,—(1.) The material peculiar to cancer, consisting of very varied forms of nuclei, nucleated cells, and juice, all of peculiar natures, distributed through an intercellular medium; (2.) Areolar tissue which constitutes the stroma or skeleton part of the new growth. The first part is the essential and heterologous part of cancer. The relative quantity of these crude materials gives the most marked and obvious ground—that of consistence—on which cancers have been classified—namely, into *hard* and *soft* cancers; but the grounds of classification are by no means fixed. The nature of the substance also affords a ground of classification. When the fibrous stroma is predominant, the new growth is *hard*, and has received the name of *scirrhus*. When the cellular elements predominate, the new growths are *soft*, and have received the name of *encephaloid* or *medullary* tumors. Sometimes the supposed nature of the substance is a ground for classification,—*e. g.*, *meleceris*, *atheromata*, *steatomata*. Many of the names given to cancers are only stop-gaps, tending to retard inquiry,—*e. g.*, *colloid*, *alveolar*, and the like. The true nature, rather than form, ought to be the ground of classification.

These, again, are subdivided into a considerable number of varieties by the minuteness of anatomical analysis to which they have been subjected. For instance, the stroma takes various forms, like net- or trellis-work, with large interspaces, or it grows papillary or villous in ramifications or vegetations. Sometimes it undergoes ossification, and then the skeleton of the cancer is formed of a network of true bony texture. This stroma part of the cancer is to be distinguished from the common binding tissue of the organ or texture into which the new growth is infiltrated. It is really and truly part of the cancer-exudations, and forms their basis. In the soft cancers it is more deficient, and then the cellular element predominates; and in them, rather than in the stroma, has the more distinctive elements of cancer-exudation been said to exist. The questions are, indeed, often asked, What are the characters of the true cancer-cell? Has the microscope discovered any structure which is decisive *per se* of cancer-growths wherever found? It may be said that it is significant of cancers that the forms of the elements of the tumors are after the pattern of those elements whose office is to separate whatever is refuse or abnormal from the blood—*i. e.*, the glands. Cancer-cells are formed on the types of the excretory gland-cells, and although they have no special anatomical characters, it is highly probable that malignant tumors

eliminate something specific—something peculiar to each of the—which may yet be discovered. Much importance has therefore been assigned, and with justice, to the character of the cell-element as affording a specific distinction between malignant and benign forms of growth. Viewed, however, with reference to single isolated cells, it is now agreed that they offer in themselves nothing anatomically characteristic.

[Mr. Collis says that the cancer-cell is a large, soft, weak, and delicate cell, and probably lighter—in other words, that there is the same amount of material in the larger cancer-cell as in the smaller lymph-cell (*Diagnosis and Treatment of Cancer, &c.* London, 1864).]

The practical questions to solve when a growth is first apparent are—(1.) Is it a mere hypertrophy—a growth from a spot, of the same nature as the structure of the spot, or is it different? (2.) Will it produce a fluid, or germinate elements which will spread and contaminate neighboring parts? In other words—Is it simple and innocent? or, Is it complex and malignant? To judge of these things it is necessary to remember that all tumors are composed of a conglomerate mass, made up of numerous little lobules. The first development takes place at a definite point or points. Round the original rudiments of the tumor (that is to be), and which are produced by the proliferation of a limited group of cells, little new foci are formed, which, increasing in size, group themselves round the first, and thus gradually give rise to a continuously progressing enlargement of the original *tuber*. At the peripheral portions of tumors, therefore, the most recently formed portions of the new growth are to be found; while in the centre of these tumors the elements are disintegrating. Thus the last zone of new growth may extend a considerable distance beyond any line of degeneration or alteration of structure visible to the naked eye; and hence in some tumors there may exist sources of local recurrence after extirpation. The more the anastomosis and the greater the facilities for the passage of juices, the more readily do the surrounding parts become diseased. Cartilage is slow to become cancerous by contiguity, so also are white and elastic fibrous tissues. Hence joints are often unaffected in the midst of cancerous tumors.

The more readily the morbid juices are transferred from the original seat of disease, the anastomosing elements will be found more numerous. Thus nerves are often the best conductors for the spread of cancers, not because they are nerves, but because they are parts with soft interstitial tissue. Diffusion also takes place readily by means of vessels.

While simple tumors are *overgrowths*, in addition to being *outgrowths* or new growths, they imply a continuous reproduction of one or of several tissues taking place to an excessive extent in any limited territory, ultimately giving rise to a tumor by coalescence and growth. But to cancers or malignant tumors there is *something more specific superadded*. Their intimate structure is not like that

of any fully developed natural part of the body, nor like that found in a natural process of repair or of degeneration.

Malignant tumors are indicated by the following characters:

1. *Elements of structure and mode of growth of malignant tumors.*

(1.) Continuous development of simple cells, largely supplied with very fine bloodvessels, so that the tumor is very succulent. (2.) The growth is mainly a growth of cell-elements, to the exclusion of connective or fibrous tissue. (3.) In the first instance the cell-elements resemble the cells which compose the blastodermic membrane of embryos. (4.) If a new growth is composed mainly of cells, each containing growing matter within it, in the form of another cell, or cells, or granules, it must be regarded as of malignant tendency if it is supplied with bloodvessels. (5.) *Generally* a constant or continuous repetition of the same structural element is evidence of malignancy. (6.) A tumor consisting entirely of nuclei or of minute cells abundantly supplied with fine ramifying bloodvessels is perhaps the most malignant type of all.

2. *The grouping of the elements which distinguishes cancer.* (1.)

The elements are heaped together disorderly, with seldom any lobular or laminar arrangement. (2.) Multiplicity of elementary forms are sometimes seen in the mass, due to (a.) Development or overgrowth, nodulation being an important feature in such tumors. (b.) Degeneration, calcareous, yellow, or fatty. (c.) Proliferation of cells.

3. *Infiltration by juice through parts abounding in anastomosing tissue.* Nerve-sheaths and sheaths generally are proven to give facilities for the extension of cancers—an important point for the surgeon to recognize.

4. *A peculiar tendency to ulcerate*, preceded by softening, with no disposition to heal, but a constant tendency to spread.

A peculiar softness of texture precedes ulceration, which simulates the slow fluctuation of a thick pulp, and appears to be more liquid in some places than in others. The softening is a continuous process of secretion of fluid in many respects peculiar, and is a physiological property of their structure.

5. *Malignant tumors constantly grow and progress to a fatal end*, and at the same time tend to multiply or propagate themselves.

6. *There is scarcely a tissue they will not invade.*

It is in the comparative appearance of the multitude of cells, and especially in their relative size, that most distinguishing features may be observed. While the cellular cancer-mass often shows merely small pus-like or large lymph-like cells of an oval form with many nuclei, yet when all kinds of cancer are compared, "*typical cancer cell-elements*" in quantity may be described by the following general characters:

While the external cell-wall presents the greatest multiformity of shape, the dimensions of the cell are comparatively fixed to a mean of about  $\frac{1}{1200}$ th to  $\frac{1}{1000}$ th of an inch in diameter. Its nuclei, however, are more constant in appearance and more characteristic than the cells (LEBERT, BENNETT, PAGET, LAWRENCE). They are always voluminous in themselves, as well as in relation to the area

of the cell in which they are inclosed; of a more regular form, being generally oval, or nearly rounded, clear and well defined, with distinct single nucleus, and rarely two. Their average mean longitudinal diameter is from  $\frac{1}{800}$ th to  $\frac{1}{600}$ th of an inch.

While Lebert attached too exclusive an importance to "a specific cancer-cell," he fully admitted the possibility that the most active and baneful cancers may vegetate through the system without exhibiting any of the cells now described, corroborating in this respect the general statement previously made by Dr. Walshe, "that a tumor may present to the naked eye the character of encephaloid and be the seat of interstitial hemorrhage, affecting the communicating lymphatic glands, run in all respects the course of cancer and nevertheless contain no cells but such as are undistinguishable from common exudation-cells." Müller, Bennett, Paget, and Lawrence have also since stated the impossibility of distinguishing the cell-element of cancer, as now described, in all cases, from the cells in certain other abnormal and even normal tissues, so that no *single* element can be considered as characteristic of cancer.

The general microscopic characters of cancer-tumors, so far as they can be determined, may thus be summed up as follows:

### I. *The Structure which characterizes Cancerous Growths:*

1. Nucleated cells—free and not embedded in any formed interstitial substance.

2. The arrangement of the elements of structure in no defined order—disorder and confusion of elements being the rule.

### II. *Microscopic Distinctions of Cancer-Elements:*

I. Cells of the following characters either compose the tumors alone or are associated with other elements:

(a.) Cells in which the nucleus is very distinct and very large in proportion to the cell.

*In size* the cells range from  $\frac{1}{800}$ th to  $\frac{1}{700}$ th of an inch in diameter, with a mean of about  $\frac{1}{1000}$ th of an inch; the cells of the smaller dimensions being usually found in cancers of the quickest growth.

*In structure* the cell-wall is peculiarly thin and delicate, so as to give the appearance of cell-shaped masses of a soft but tenacious substance inclosing nuclei.

*The nuclei* are constant as to size and presence. They are large, regularly oval, or nearly round, clear, and well defined. Are not easily altered by decomposition or any usual reagent. In size about  $\frac{1}{250}$ th of an inch. Two nuclei are frequent—more than two are rare.

*In form* the cells are very various—the outline being linear, angular, or extended in processes.

There is thus great diversity in the elements which compose cancers. The diversity exists alike in the corpuscles, the basis substance, or inter-cell material. The cells are often loosely held together in an abundant, soft, almost liquid substance, as in medullary cancer.

(b.) Free round or oval nuclei alone sometimes compose a medullary cancer. They are bright, clear, perfectly defined, largely and often doubly nucleolated.

(c.) Nuclei mixed with elongated, narrow, stripe-like, caudate, or pyriform cells.

(d.) Elongated caudate cells.

(e.) Monilliform growths, or jointed and branching cells.

The basis substances proper to cancers are—

(a.) Liquid alone ; amongst which the cells are suspended, and either infiltrated amongst the tissue or contained in small cavities (*e. g.*, in cancers of rapid growth). This is “cancer-juice,” and may be thick, creamy, yellow, or pink, or of a glairy consistence like synovia or oil.

(b.) A framework of granular tissue, extremely delicate, sometimes hyaline, and containing oblong nuclei.

It is important to notice, however, that the “modes of life,” development, and growth, rather than structure, should determine whether a tumor is malignant or not. There are cancers *not* formed of such structures as those mentioned, namely :

(1.) Some fibrous tumors, the clinical history of which is undoubtedly cancerous ; also (2.) Some osteoid tumors.

The multiplicity of structures composing a tumor is often due to the mingling of cells in various stages of growth, development, or degeneration ; or it may be due to disorderly overgrowth and proliferation of cells. When the diversity is due to *development*, overgrowth, or proliferation, nuclei, brood-cells, and caudate cells are characteristic ; when due to *degeneration*, granule-masses predominate, or withered corpuscles, with fatty and calcareous masses in yellow parts.

What is supposed to be the blastema or mother-fluid amongst which the cells and stroma of cancers grow, consists of an albuminous, synovial-like, colorless, or pale yellowish fluid. It gives rise to the materials which compose the cancer-juice, and to the essential cancer-mass now described, namely, the stroma and the cellular elements. The development of cancer, therefore, proceeds *pari passu* with this fluid as amongst the interstices of textures, or on the free surfaces of the membranes ; and lastly, even by endosmosis of the specific fluid into the natural cells of parts (such as into cartilage or bone-cartilage cells), the characteristic cancer-mass may commence to grow by endogenous growth within such cells, hitherto healthy. Dr. Bennett, of Edinburgh, and Dr. Van der Kolk, of Utrecht, have made most interesting observations on the extension of cancers by means of the parenchymatous fluid ; and from all that has been observed on this subject, the latter author draws the following conclusions :

“1. Through an interchange of material, taking place between cancer-cells and intercellular fluid, the latter acquires the property of forming new nuclei and cells of a similar nature.

“2. This intercellular fluid passes, along with the parenchymatous fluid pervading the sound parts, into the textures adjoining the tumor. The parenchymatous fluid thus acquires the same constituents and ten-



dency to form similar cells, which now become developed among the healthy surrounding tissue, in the course of the areolar membrane.

“3. On account of the minuteness and small number of the last-mentioned cells, their presence cannot be detected with the naked eye; so that the surrounding parts may appear to be perfectly sound, notwithstanding that they contain the germs of the advancing formation of cancer.

“4. It is therefore of importance, in removing cancer by operation not only to take away at the same time a large quantity of the adjacent sound parts, but also to examine the innermost sectional edges under the microscope, in order to ascertain whether any trace of cancer-cells in process of formation is to be discovered in them.

“5. The existence of burning or shooting pains in carcinoma may be taken as a proof that the cancer-cells have reached the neighboring nerves, and the disease can then scarcely be looked upon as a local one, in which an operation might be permanently successful.

“6. By the absorption of the infected parenchymatous fluid through the lymphatics and veins, the whole body seems to become more or less tainted, so that secondary cancer ensues in distant situations, when, as is self-evident, operation can no longer be thought of.

“7. This altered parenchymatous fluid penetrates the organic tissues which are washed by it, the sarcolemma of the muscular fibres, the tubes of the nerves, and the like. These membranes, too (both the sarcolemma and the walls of the nervous tubes), appear to take up the altered nutritive fluid; the consequence of which is, that both within the sarcolemma and the nervous tubes similar nuclei and cells arise, accompanied with an absorption of the muscular fibre and of the contents of the nerve, and attended with the deposition of fat, by which these parts waste and are destroyed, while the surrounding membranes (sarcolemma and walls of the nervous tubes) remain” (*Brit. and For. Med.-Chir. Review*, April, 1855).

The cancer-juice is a most important element of the new growth. To the naked eye it appears as a viscid, whitish, creamy, yellowish fluid, which may be squeezed or scraped in considerable abundance from the surface of a section.

It is from the performance of the vital functions by all these elements that we are to draw our conclusions regarding the innocency or malignancy of the new growths in which they form a part, rather than from their anatomical forms. If we find adjacent textures are being infiltrated, poisoning the lymphatic current which passes from them, inducing new growths of a like nature in the lymphatic glands through which these currents pass, affecting the general system with a peculiar cachexia, marked by languor, debility, emaciation, and a peculiar sallow, leaden-like color of the skin; and if after removal such growths return, then there is no doubt that the cachexia of cancer is made manifest by such local lesions. These present so many elements in common, that all these new growths are but manifestations of one disease, which has been named “cancer,” and attributable to the constitutional cachexia already described. The varieties of this disease also run into one another by characters which are so insensible that definite lines cannot be drawn between them, and thus many species of cancers are described by authors under various synonyms, as shown in the nomenclature



adopted by the following authors who have recently written on cancer :

I.	II.	III.	IV.
WALSHE AND BENNETT (1846-49).	PAGET (1853).	ROKITANSKY (1855).	LAWRENCE (1856).
Scirrhus, or hard cancer. Encephaloma, or soft cancer. Colloid, or jelly-like cancer. All other forms of cancerous new growths are described by Bennett under the name of <i>cancroid</i> growths.	Scirrhus, or hard cancer. Medullary, or soft cancer. Epithelial. Colloid. Osteoid. Melanotic. Villous. Hæmatoid.	1. Fibrous carcinoma. 2. Medullary. (a.) Villous cancer. (b.) Cancer melanoides. 3. Epithelial cancer. 4. Gelatinous cancer. 5. Carcinoma fasciculatum. 6. Cystic carcinoma.	Scirrhus. Encephaloid. Melanosis. Nævoid cancer. Villous cancer. Osteoid cancer. Enchondromatous cancer. Colloid cancer. Fibrous cancer. Fibro-plastic cancer. Epithelial cancer.

Of these particular kinds of cancer some tend to affect certain organs rather than others,—for instance, *alveolar cancer* is more frequently found in the stomach or intestines; *epithelial cancer*, in the skin and mucous membranes.

The *primary cancer-growth* commences in the textures of some organs rather than others—for instance, in the uterus and female breast, in the stomach, the colon, the liver, the bones, and the brain. *Secondary cancers*, on the other hand, are most frequently developed in the lungs, the spleen, the salivary and lymphatic glands, in the small intestines, and in the serous membranes. But the local aptitude for cancer-growths is a subject unknown. We have no knowledge why one part rather than another should be the seat of cancer; but certain organs seem more liable than others at certain periods of life. For example, before thirty years of age the eye and the orbit are the parts first most liable to be affected, next the bones, testicles, and areolar tissue of limbs and trunk. Between thirty or fifty years of age the penis, uterus, external sexual parts, and breasts. After fifty years of age the integuments and digestive organs are more apt to suffer.

Simple tumors may also become the seat of cancers, just as any part of the body may be the seat of a cancer, although the event is rare. About one-fifth of cancers are ascribed to the effects of injury; and although in many cases the statement may be fallacious, yet the consequences of injury are sometimes too obvious to admit of doubt.

*Scirrhus*, or *Hard Cancer*, has two stages—a hard stage and a stage of softening. It is a “*cancer characterized by hardness of the primary tumor, and by a tendency to draw to itself the neighboring soft structures. When ulcerated, the sore is commonly deep, uneven, and bounded by a thick everted edge.*”

The local hard or scirrhus state constitutes the first stage. It may grow in *masses*, or may be *infiltrated* into the tissues of the organ or part affected—the latter being by far the most common form. When in masses, they are generally lobulated, dense, and often contained in a cyst; again, when these masses are cut into, we find them to consist of two substances—the one is the cancerous growth, and the other is areolar tissue; so that the appearance of the divided surface in general is that of a hard, white, semi-car-

tilaginous substance, streaked by fibres radiating from the centre to the circumference. They are of considerable density and firmness, and in hardness of texture vary from hard-boiled white of egg to cartilage—the knife making a grating noise as it cuts through them. The specific weight of these tumors is extremely great; and although in those parts which are external and more or less pendulous, as in the mamma, this fact is, by some distinguished surgeons, an element in diagnosis, our exact information relative to the specific gravity of cancerous growths is still very limited—1.040 to 1.160 is the very wide range which I have observed such tumors to indicate.

The cancerous growth, however, is much more frequently infiltrated among the areolar tissue of the different organs or tissues it affects. In this case the affected tissue becomes gradually increased in thickness and in density by a slow growth of the morbid matter, so that the part, if now divided, presents the same hard, semi-transparent character as in the masses, but more interspersed with areolar tissue, the diseased portion being gradually shaded off into the healthy structures. In the mucous tissues, as those of the stomach or uterus, the infiltrated growth has often a considerable thickness, measuring from a quarter of an inch to an inch, or perhaps even more. On the contrary, when infiltrated into the cutaneous tissue, the layer is often so attenuated as to be scarcely appreciable, and the disease commences with little other appearance than a small hard pimple, or a small erysipelatous tumor, or even by a slight fissure or crack in the skin.

After a certain but indefinite period, which varies from a few months to a few years, the scirrhus stage of *hard cancer* terminates, and the second stage, or that of *softening*, begins. In mucous membranes this softening usually takes place at their *surface*, or superficially,—as at the mucous surface of the neck of the uterus, or at the mucous surface of the stomach. An ulcer is the consequence of this softened state, and is at first superficial, presenting many remarkable varieties, such as an inverted or everted edge, and an irregular form, while its base may be granulating at one part and sloughing at another. Its course is burrowing, often penetrating between the cancerous lobules, and ultimately may perforate the limiting serous covering, such as the peritoneum. The pus secreted by this sore is fetid; often a mere ichor, or pus mixed with blood, and so acrid as to inflame the parts over which it flows. In a few instances the large vessels participating in the disease ulcerate, and the patient dies of hemorrhage.

The duration of the scirrhus stage of a cancerous tumor is very uncertain, and may terminate in a few months, or may last several years. A cancerous mammary gland, for instance, has been known to remain indolent for fourteen years, and has at the end of that time been removed by an operation. This indolent character of cancer is limited, however, to the scirrhus stage; for after it has commenced to soften, its course is rapid, and a few weeks or a few months generally terminate the patient's life, the part affected in

no instance cicatrizing, or being again restored to a healthy condition.

*Medullary Cancer, or Soft Cancer*, affects more especially the solid visceral organs. It is a "cancer characterized by a smoothly lobed surface, soft, irregular consistence, great vascularity, and usually rapid growth and reproduction. When ulcerated, it protrudes in large masses, which bleed copiously." Its cell-products are most profuse, and its course much shorter than hard cancers, the disease generally terminating in a few months. While hard cancer for the most part affects persons in the decline of life, soft cancer is most common in its earlier period, or adult age, from twenty-five to forty.

Although generally deposited in masses, it may be infiltrated: the former is the more common form, the latter the more rare. In whichever form deposited, however, it has two stages,—namely, one of induration and one of softening. If we examine a soft cancer-tumor in the first stage, we find it composed, as in hard cancer, of fibrous tissue and a peculiar morbid growth. The areolar stroma is of various densities, often extremely fine, and then again of considerable consistency and tenacity, and in either case radiating through the tumor and dividing into lobules. The morbid substance or growth is of many degrees of hardness, varying from lard to cartilage, but is generally softer than in hard cancer; it is of a bluish semi-transparent whiteness. The duration of this hard stage is from a few weeks to two, three, or four months, and only in a few instances does it exceed that latter period. "*Hard encephaloid* is a designation sometimes applied to medullary cancers of unusually firm consistence."

The first stage passed, the process of softening, or of *ramollissement*, takes place. This is evident on cutting into the tumor, and passing the handle of the scalpel over the divided surface, a milky-white substance being expressed. As the disease proceeds, the parenchymatous substance of the new growth is changed into the consistence of soft cerebral matter, or of thickened pus; it is consequently opaque, and varies in color from white to red, and even black. These variations of color appear to be owing to the different quantities of blood or of melanic matter which are effused, and with which the cancerous matter is commixed. When bloodless and white, the product is so peculiar that it has been termed *cerebriform*, and when mixed with blood, *medullary sarcoma*, *fungus hæmatodes*, and many other terms, according to the different quantities of that fluid effused, which is often so abundant that the cyst or cavity at length contains little else than fibrin. "*Fungus hæmatodes* is a term applied to some cases of medullary cancer which are more than usually vascular."

The process of softening seems to commence indifferently in any part of the tumor, at its centre, or towards its circumference; and if the tumor communicates externally, the quantity of softened matter discharged often amounts to many ounces in the course of the day. This profuseness of discharge appears to be owing to the great vascularity of the growth; for although in the hard stage only a few bloodvessels, with coats of great tenuity and delicacy,

can be traced between the lobules, yet, in the softened state, a successful injection shows the growth to be made up almost entirely of bloodvessels.

The duration of the second stage is generally a few weeks, and very rarely extends to months. It appears, however, that anything which greatly irritates the part accelerates the process of softening. Thus, if a cancerous limb or tumor be amputated, the cancerous matter primarily in a hardened state appears subsequent to the operation in a softened condition, no previous hard stage occurring. The minute organic structure of this form of disease, in its scirrhus state, is probably not dissimilar to that of hard cancer, and of the vital organic characters there can be no doubt of their similarity.

There is scarcely any organ or tissue in which soft cancer has not been found, and by some pathologists the frequency of its occurrence in certain parts is supposed to be in the following order: the liver, epiploica, the mesentery, the lymphatic glands, the brain and nerves, the spleen, the testicles, the uterus and ovaries, the eye, the bones, the heart, and lastly the bloodvessels. It has been stated that soft cancerous matter far more frequently grows in masses than infiltrates into these parts. In general there is only one tumor; but there may be, as is often seen in the liver, three or four, and in some cases they are extremely numerous. Dupuytren has met with a carcinomatous heart which contained more than 600. In size they commonly vary from a millet-seed to a large egg; but when they form in loose cellular tissue, as between the folds of the mesentery or of the *epiploica*, or in the substance of the lungs or testicle, they have been known to weigh, in extreme cases, 20, 30, 40, and even more pounds. These tumors may also be encysted or non-encysted.

One of the most constant features of this disease, and which distinguishes it from hard cancer, is, that it often appears in many organs or tissues at the same time in the same patient. Thus, it has been met with in the coats of the bladder, in the liver, and in the lungs of the same individual. It has also a greater tendency to be reproduced after an operation for its extirpation than any other kind of cancer. This reproduction may take place either at the part operated on, or in some organ or tissue distant from the primary seat of the disease.

*Melanotic Cancer*, or *Melanosis*, is a “cancer characterized by the presence of pigment,” and which may be found distributed very generally in every organ and every structure of one and the same body—often becoming first manifest in the soles of the feet or in the axillæ. The lymphatic glands subsequently become affected. Such growths are also found in the ovaries, in the mucous membrane of the bladder and intestines, in the kidneys, supra-renal capsules, heart, and brain. The “*fungus hæmatodes*” have generally been considered related to *melanotic cancer*; and the relationship is more than ever established in the connection which subsists between pigments and the changes which take place in extravasated blood. Frequent hemorrhages are common in melanotic growths of a can-

cerous nature, due to extreme dilatation and thinning of the water of the bloodvessels, and the red color of such tumors must be referred to such kind of vascularity. Pigmentation of such tumors is therefore to be considered as of secondary formation; and in the majority of instances melanotic cancer consists of encephaloid or soft cancer, with the addition of black or brownish pigment. The pigment-deposit, *per se*, is not necessarily malignant. The pigment of cancers is readily decomposed by nitric and other acids, while the *spurious melanosis* or *carbon* is not. The true melanic deposit exists with the soft cancer-cells either as an infiltration into them, or in the form of isolated granules or small corpuscles. The pigmentation may be seen in microscopic sections to take place along the course of dilated bloodvessels. In an early stage the pigment is first contained in cells, whilst later it is found in free granular masses, through the dissolution and disappearance of the cells which contained it, and a gradual transition may in general be traced from the affected part into the normal structure of tissue, where it will ultimately become developed, and which is freely traversed by bloodvessels. Cells of various forms are to be seen in such tumors, which have first a yellow appearance and then a black, according to the amount of pigment deposited in them. The cell-walls subsequently disintegrate, leaving the pigment free.

*Colloid, Colloid Cancer, or Alveolar Cancer*, is sometimes also called *gelatinous* or *gum-cancer*. In this form the meshes of the new growth are filled up by a glue-like or gelatinous substance, like half-dissolved gum-arabic. It is "*a new growth, a great part of which is formed of transparent or gelatinous substance.*" The fibrous stroma are arranged in the form of alveolar spaces, the fibroid tissue being extremely delicate and transparent. The spaces or alveolar loculi vary from a very minute size to that of a pea. The contained substance is of a yellowish or greenish-yellow color, but yields no gelatine on boiling. The most common places where colloid cancers form are the stomach and omentum, the ovaries, the bones, the kidneys, the uterus, and spleen, and they may be combined with the *hard* or *soft* cancers.

[Colloid, Mr. M. H. Collis, of Dublin, regards as structurally and clinically removed from cancer. All the tumors of the kind which he has seen contained agglomerations of the smallest lymph-cells, held together by the finest connective tissue, but never large cells like those of cancer. He rejects the name "Colloid Cancer," and calls them "Colloid Tumors" (*loc. cit.*, p. 198). These growths include Laennec's colloid, the myxomata (*schleimgeswülste*) of Virchow, and some of the softer varieties of Paget's fibro-cellular tumors. According to Virchow they have altogether the structure of the umbilical cord, having mucus in their intercellular tissue (*Cellular Pathology*, p. 526).

According to Virchow, Johannes Müller's collonema (*gallertgeschwülste*) is merely œdematous connective tissue, and cannot be separated from fibrous tumors generally. In Virchow's mucous cancer (gelatinous or colloid) the stroma, instead of being composed simply of connective tissue, consists of the same mucous tissue which we meet with in a simple mucous tumor, and differs in its gelatiniform nature from the ordinary



stroma of cancer. These tumors frequently return after extirpation, and may manifest the worst features of malignancy, as the writer has lately had the opportunity of seeing in two cases of so-called fibroid tumors of the uterus.

There is a form of colloid which belongs to the true cancer group anatomically and clinically. Such are the cases described by Lebert (Virchow's *Archiv*, Bd. iv) and by Förster. The latter writer states that both scirrhous and medullary cancer may undergo transformation into colloid. Paget (*loc. cit.*, p. 663) has suggested, as a reasonable hypothesis, that the peculiarities of colloid cancer may be ascribed to cystic degeneration in elemental cancer structures, while the elements are in a rudimentary state. Förster has traced the colloid metamorphosis in cells, and describes their advancing in three different ways. First, the cell-contents themselves become transformed into a colloid mass, which gradually increases in size and is set free. Secondly, the colloid change begins in the form of a zone round the nucleus of the cell; another zone is laid on this, and then another, until the well-known large laminated colloid body, so like in appearance to the laminated capsule of epithelial cancer, is formed: the central nucleus at the same time undergoes partition and multiplication, and a group of nuclei fill up the centre of the laminated body. Thirdly, the nucleus is itself the seat of metamorphosis, and swells up so as to be converted into a great colloid bladder. The different consistence and appearance of colloid tumors generally seem to depend on the different ways in which the colloid substance is distributed; for there may be colloid cells in pure fibrous stroma; or cells with ordinary albuminous contents in a colloid stroma; or colloid cells in colloid stroma (*Wurzb. Med. Zeit.*, Bd. iv, Hft. 5 and 6; *Brit. and For. Med.-Chir. Rev.*, April, 1867).]

*Epithelial Cancer, or Cancroid Epithelioma.*—Of late years, under these names, or that of *Cancroid*, Lebert, Bennett, Hannover, and Paget have described a form of tumor which has all the vital and malignant qualities of cancer already described, but its minute elements consist of cells resembling those of epithelium or epidermis. It is therefore defined as "*cancer characterized by its occurrence chiefly in parts naturally supplied with epithelium, and by the resemblance of its cells to those of the epithelium.*" It occurs almost solely on the skin or mucous membranes, being frequently seen on the lips or cheeks. Rokitsansky has observed it in the liver; Bennett has described it as commencing primarily in the lymphatic glands; and in two cases of what has been termed acute hypertrophy of the mamma, both of which proved fatal with secondary lesions in internal organs, which I examined after death, I believe that this form of cancer was the primary local lesion in the gland (*Med. Times and Gazette*, for 1857). It only seems to occur where there is pavement, or spheroidal epithelium. Lebert has seen it on the serous layer of the arachnoid, and Robin has described it on the interior of a vein in a horse. The principal sites are in the vicinity of the great orifices of the body; but the lips, and more particularly the upper [lower?] lip, [at or near the juncture of the skin and mucous membrane,] are most liable to the development of epithelial cancer. [Next in order of frequency it is found in the tongue, prepuce, scrotum (of chimney-sweeps), labia, and nymphæ; more rarely it occurs in very many



other parts,—as at the anus, in the interior of the cheek, the upper lip, the mucous membrane of the palate, larynx, pharynx, and cardia, the neck and orifice of the uterus, the rectum, and urinary bladder, the heart, the skin of the perineum, of the extremities, head, face, and various parts of the trunk (PAGET).] The cancer commences first as a small induration, afterwards a pustule or excoriation forms, and subsequently the deeper parts participate, and determine the form of the growth, which may be mulberry-shaped, villous, cauliflower-like, or nodulated. The surface is apt to become ulcerated at an early period, and an actual loss of substance takes place from the central parts. The resulting open sore has an irregular, gray, and often a bloody base; or it is covered with crusts, from below whose edges a fetid and ichorous discharge may be caused to exude by pressure. When the tumor is cut into at an early stage, the section presents a well-defined border of epidermis, sometimes several lines thick, while the rete between the surface-layers and the chorion is much hypertrophied. The substance is friable, and is easily separated from the surface of the chorion. The tumor soon tends to take deep root in the substance of the chorion, presenting a white and gray speckled surface, with a basement-substance, in which a variable quantity of whitish bodies [—little pearly globules—] may be seen, varying in size from a visible point to the size of a millet-seed. The fluid which exudes is milky and granular, and does not mix with water, so as to form such an opalescent, semi-transparent emulsion as the mixture of other cancer-juices with water. It mixes as if it contained fat. The tumor tends to infect secondarily the neighboring lymphatic glands; and the fatal termination is generally by exhaustion or by putrid infection, when the progress of ulceration is not arrested. The mean duration of cases of epithelial cancer is about six and a half years; and the duration seems to vary with the part as follows: Nearly three and a half years for the lower lip; three and two-third years for the penis; nearly nine years for the neck and limbs; and nine and a half for the vulva and face (LEBERT, HANNOVER.)

[Mr. Collis has made epithelioma the subject of special microscopical and clinical study. The growth for him is not a cancer nor a cancrioid tumor, for its affinities with cancer are not strong enough to place it in the cancrioid group. It originates in the deeper layers of the epidermis and epithelium, where its progressive development may be divided into three stages: 1st, the stage of simple cellular hypertrophy; 2d, that of hypertrophy with ulceration, in which the number of cells has so increased as to press down on the papillæ and cause ulcerative absorption of them; 3d, hypertrophy with infiltration, in which we find enlarged papillæ topped with masses of cells, which not only press on and ulcerate the papillæ beside them (*i. e.*, the cells), but also push the new-formed cells downward between them (*i. e.*, the papillæ). The widespread devastation which an epithelioma may cause, results from this continuous cell-formation, which gradually presses before it and destroys whatever tissues it meets with. The cells, too, may push their way between the separated papillæ, and, arriving there, irritate the bloodvessels so as to produce an interstitial effusion of plastic lymph. In this lymph the epithelial cells develop fresh

nuclei and cells like themselves, and thus become a fresh centre of irritation, the disease advancing by fresh development of cells in the interstitial effusion.

Whether epithelioma has its origin in the cells of the epidermis or in those of the connective tissue of the cutis and mucous membrane, is a difficult point to decide. Hannover, Collis, and Thiers maintain the first theory, while Virchow, Förster, and Weber hold to the latter.

In throwing out epithelioma from the cancer group, Mr. Collis says: Its superficial origin, its slow progress, its indisposition to infiltrate the deeper structures, or to contaminate the glands, the certainty of cure which follows its timely removal, and the different appearance when occupying similar localities, are of sufficient importance to outweigh the points of resemblance which it undoubtedly bears to cancer in its advanced and secondary stages. In its early stage it is strictly an hypertrophy, and in this condition it may remain for an indefinite period. Its second stage is one of hypertrophy and ulceration combined. This stage also, as far as external or cutaneous epithelioma is concerned, is slow to advance into the third or destructive stage—that of infiltration and secondary deposit (*l. c.*, p. 226). Virchow writes: It remains for a very long time local, so that the nearest lymphatic glands often do not become affected until after the lapse of years; and then again the process is for a long time confined to the disease of the lymphatic glands, so that a general outbreak in all parts of the body does not take place until late and only in rare instances (*loc. cit.*, p. 531).]

*Villous Cancer* is a name given to “cancer in mucous membrane, when covered by a villous growth;” and

*Osteoid Cancer* is a “tumor usually commencing in the bones, consisting almost entirely of bone, and followed by similar growths in the glands and viscera.”

**Diagnosis.**—Cancerous affections of internal parts may be simulated by many nervous disorders, and also by chronic inflammation of the respective parts; but the long continuance of the symptoms, their gradual augmentation, the severe pain, which admits of no permanent relief, together with the loss of health and slow emaciation of the patient, at last give a moral conviction that it must be cancerous growth in some organ from which the patient suffers.

**Prognosis.**—Cancer, though long latent, and its course slow, pursues its destructive progress unimpeded, and in rare instances does amendment or a return to health await the patient, who ultimately falls an inevitable victim to the disease.

**Treatment.**—No remedy has yet been found which can in any degree be considered curative of the constitutional state associated with cancer, and the efforts of the practitioner are consequently limited to relieving symptoms, and to the adoption of such palliative measures as may prolong life. It has generally been believed that to remove a cancerous growth where it is practicable must, on theoretical grounds merely, be attended with as much benefit to the constitutional disease as would attend the removal of a leg for acute rheumatism chiefly expressed in the knee-joint. The statistics of cancer show, so far as they go, and as Dr. Walshe long ago showed, that “excision of a cancerous tumor seems to awaken a dormant

force. Cancers spring up in all directions, and enlarge with a power of vegetation almost incredible." Nevertheless, there are good reasons for removing cancers in some cases, especially mammary cancers, and others accessible to the knife. The greatest measure of good may be done, as Mr. Paget has clearly shown, "by making a careful selection of cases fit for operation, and rejecting all the rest as unsuited for operation" (*Med. Times and Gazette*, September 27, 1862, p. 319).

With regard to excision of the breast for cancer, the main objection in the first instance is, that even of cases selected with some care, 10 per cent. (PAGET) to 16 per cent. (LEBERT) die of pyæmia, or erysipelas, or tetanus, or secondary hemorrhage, or some calamity subsequent to the operation. On the whole, however, taking the results of some hundreds of cases, it is certain that the average duration of life of those operated on is not less than those in whom the disease runs its course. In well-selected cases it will be found always greater. A recent tabulation of hospital and private cases by Mr. Paget showed that 85 cases operated on lived an average of 55.6 months; and 62 cases not operated on lived an average of three months. It has also been said that the recurrent disease is more painful than the primary one; but in very many cases Mr. Paget has found that the recurrent disease was much less severe than the continued disease. Considering, therefore, the danger of the operation, and the fact that in every case a recurrence of the disease may be expected, is it reasonable to submit a patient to the risk of dying from the primary operation for the sake of that interval of health between the operation and the recurrence of the disease, for the good probability of adding a year to life, and for the chance of having a less severe disease? The average length of the interval before recurrence is little more than *thirteen* months; more than a half return within twelve months; and two-fifths return within six months. The extremes between which the average is drawn are very wide. In some cases the return may be within *three* months; in others not for *ten, twelve*, or more years.

It is of great importance to determine in what cases the risk of life is greater, and in what the probability of a speedy return of the disease is greater than the average.

The old after 60; the very large-breasted in cases of mammary cancer; the fat and plethoric; the cachectic; the overfed on animal food; the drunkards; the gouty; the habitually bronchitic; the albuminurians; the very dejected—not timid merely; in short, those with any organic disease of the internal organs—all such cases are "doubly hazardous" to interfere with by operation (PAGET).

The probability of rapid recurrence is great in acute cancers—*i. e.*, all those that are rapid in their progress—and in those which are observed to increase very quickly before the operation. Great pain, however, is often saved by performing the operation even under such circumstances. In illustration, Mr. Paget records the case of a lady "whose breast he removed when she was five months advanced in pregnancy. She recovered well from the operation, and the benefit procured by its performance was very great. She went

to her full term, bore her child, and was able to suckle it for a year before she died, with her most anxious wish fulfilled in comparative comfort."

Another condition unfavorable to operation is a brawny skin with firm œdema and wide-open hair follicles, or wide adhesion of skin, or in which the skin is cancerous, or where there are scattered tubercles of cancer in the glands and skin; or where there is considerable affection of the lymph-glands in the vicinity, and especially *numerous* diseased glands. A moderate amount of lymphatic disease Mr. Paget does not consider a serious obstacle to an operation. In very chronic cases the operation is needless—where the *breast* is small, shrivelled, knotty, and sunk down on the pectoral muscle. By thus selecting with care, on the one hand, cases fit for operation, and refusing to interfere in those cases in which the operation would be attended with more than a proper share of danger, Mr. Paget believes that the life of a large number of those who suffer from cancer may be considerably prolonged.

In whatever part the disease may be situated, one great rule is to endeavor to restore the healthy functions of that part, and to alleviate by opiates, chloroform, or chlorodyne internally the distressing pains the patient endures. These remedies are for a time successful, but make no impression on the disease, which silently proceeds, and the patient finally limits himself altogether to opiates. The quantity of opium or other narcotic known to have been taken in such cases is something enormous—five, ten, fifteen or twenty grains of opium, at a dose, or a proportionate quantity of *hyoscyamus* or of *conium*, three, four, or more times in the twenty-four hours. But although these large doses have occasionally been given, yet it may be questioned whether they are not more hurtful than beneficial; for usually they produce headache, delirium, loss of appetite, and narcotism, so that the patient is only the more rapidly exhausted. In general, therefore, the patient does better under moderate doses of opiates, as one or two grains, or its equivalent of morphine or other narcotic, every four, six, or eight hours, than when more excessive doses are given—a larger dose producing headache and much cerebral disturbance, without in any sensible degree mitigating the sufferings.

When the disease is seated in the colon or intestine, the tumor is in general difficult to make out. One loop of intestine may open into another; and death by hemorrhage may terminate the case. The symptoms vary greatly according to the position of the cancer and the part of the intestinal canal affected. Great sickness and vomiting generally attend cancer in the duodenum.

When the stomach is so irritable that it rejects everything, it is our duty to support the patient by nutritive injections, as of strong broth, egg-flip, sago, or other fluid substances. It has been attempted to impart strength to the patient by means of milk baths, or baths of strong broths; but the skin has not generally sufficient power of absorption, and it has been found that the heat of the bath has exhausted the patient in a far greater ratio than its nutriment supported him.

As a general principle, diet has little or no influence over the course of the disease, when once established, so that whatever agrees with the patient's digestion may be safely indulged in.

Preventive treatment must be directed to the infant life of those who are hereditarily predisposed.

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### CHAPTER III.

#### PATHOLOGY OF THE SCROFULOUS ORDER OF CONSTITUTIONAL DISEASES.

THE diseases belonging to this order are more strikingly wasting in their effects upon the body than those which belong to the order now just described. They comprehend more especially *scrofula*, either with or without *tubercle*, *tabes mesenterica*, *phthisis pulmonalis*, or *pulmonary consumption*, *acute miliary tuberculosis*, *tubercular peritonitis*, *tubercular pericarditis*, and *tubercular meningitis*, or *hydrocephalus*, *scrofulous ophthalmia*, and *scrofulous iritis*. Many of them are characterized by the growth of a peculiar substance in the tissue of some organs rather than in others, to which the name of *tubercle* has been given. These growths occur in the tissue of the alimentary canal; in the peritoneum, arachnoid, or pleura; in the lungs, liver, spleen, or kidney; in the tissue of the lymphatic glands, especially the cervical, inguinal, and mesenteric glands; and sometimes in the pancreas and the tonsils.

In other respects these diseases are very similar to those of the order previously described, owing their existence to constitutional conditions; and in the present state of our knowledge they ought not perhaps to have been formed into a separate order: for there can be no doubt that in the *tubercular* or *wasting* diseases, as well as in those of the *diathetic* order, there is a *latent* condition existing before the tubercles are apparent. The relation of the nutritive and other morphological changes between the solids and the fluids of the body has everything to do with the development of the scrofulous state. In cases where the tendency to the growth of *tubercles* is hereditary, the operation of agents from without act as stimuli or excitants to the growth of them.

Previous to 1829, when Sir James Clark published his classic work *On Climate*, the tendency of pathological researches into the nature of the diseases just mentioned was to keep up the idea that these diseases, and especially *pulmonary consumption*, were merely local, and referable to a local cause.

The terms *phthisis*, *consumption*, or *wasting*, were originally used vaguely to designate a variety of chronic diseases, described entirely by local symptoms and physical signs, and therefore having few characters in common, except the marked emaciation which attended them. When researches in morbid anatomy became more frequent and efficient, the local lesion most frequently connected



with the phenomena of wasting was discovered to be a peculiar morbid condition of the lungs, and to this the name of "*consumption*" was applied, and afterwards more definitely "*pulmonary consumption*." From the frequency of this local lesion the pulmonary state of necessity attracted far greater attention than any other form of disease in which *wasting* was a marked feature; and when lesions in other organs were found associated with "*pulmonary consumption*," they were regarded as complications rather than as local manifestations of one and the same general constitutional state and were looked upon as the primary cause of the emaciation. In 1819, Laennec first showed, by his accurate post-mortem observations on the state of the lungs, that growths to which the name of "tubercles" were applied formed almost the sole cause of *consumption*, and consequently he restricted the term *phthisis* to "*the disease produced by tubercles*" in the lungs. Louis and Andral confirmed the observations of Laennec; and thus the view of this morbid state, based as it was on morbid anatomy *alone*, led to the enunciation of limited and erroneous doctrines regarding the real nature of the disease which the existence of *tubercles* indicates either in the lungs or in any other part of the body. The relations subsisting between *pulmonary phthisis*, *consumption*, or *wasting*, and the occurrence of "tubercle," must now be even still more modified under the combined researches of morbid anatomy and progressive knowledge in pathology. *Consumption*, or *wasting*, in the common acceptation of the term, is not now to be found due always to tubercle (ADDISON, GAIRDNER, REINHARDT, VIRCHOW, JENNER, and MACLACHLAN), so that the terms *phthisis* and *tubercle* cannot now be considered always as synonymous. Ulceration of the lungs and partial destruction of pulmonary tissue are now found to arise from other causes than the existence of *tubercles*, which become equally efficient causes of phthisical mischief (BAYLE, ARMSTRONG, GRAVES, STOKES, and MACLACHLAN). Nay, we know also that many states of the body to which the name *scrofula* or *struma* is applied are not necessarily attended with "tubercles" at all; yet, when the local lesions of a tubercular nature are observed to be connected with marked constitutional states under various circumstances, the connection between the *tuberculous* and the *scrofulous* state is seen to be of the closest description, and their pathological history is now generally believed to be identical.

The extensive observations of morbid anatomists since the time of Laennec, elucidated by the most learned pathologists of the age, now tend to establish on a very broad foundation the doctrine first so strenuously and ably advocated by Sir James Clark, that the morbid conditions now mentioned are due to what he termed a "tuberculous cachexia;" and accordingly it is proposed to consider the *constitutional* disease, of which the occurrence and growth of "tubercles" is but a local expression, under the general title of SCROFULA, of which the varieties are (a.) *Scrofula with tubercle*; and (b.) *Scrofula without tubercle*; and the local scrofulous affections are those named on p. 213.



## CHAPTER IV.

## DESCRIPTION OF THE SCROFULOUS ORDER OF CONSTITUTIONAL DISEASES.

## THE CACHEXIA ASSOCIATED WITH THE SCROFULOUS STATE, OR SCROFULA.

LATIN Eq., *Struma*; FRENCH Eq., *Scrofule*; GERMAN Eq., *Scrophulose*; ITALIAN Eq., *Scrofola*.

**Definition.**—*A particular morbid condition of the system, attended in active or acute forms of the disease by a persistent increase of temperature, followed by a continuous wasting of the body and the growth of a substance in various tissues and organs, especially the lungs, to which the name of "tubercle" or "tuberculous matter" has been applied;—or, without the deposit of tubercle, these febrile phenomena are associated or preceded with peculiarities of outward appearance during life, and liability to certain diseases termed "scrofulous," such as swellings of lymphatic glands and of joints, carious ulcerations of bones, frequent and chronic ulcers of the cornea, ophthalmia, abscesses and cutaneous pustular eruptions, persistent swelling and catarrh of the mucous membrane of the nose, and characteristic thickening and swelling of the upper lip,—lesions which, while they are distinguished by mildness of symptoms, are peculiarly persistent, characterized by specific forms of inflammation or ulceration, and follow on the application of exciting causes which would have no effect on a healthy person.*

**Pathology.**—The *diathesis* which expresses the latent existence of the state now defined has been variously described by the terms *tuberculous*, *scrofulous*, or *strumous diathesis*. There is perhaps no subject in the whole range of medical science which the student ought to study more carefully than the cachexia associated with the occurrence of tubercle and *scrofulous affections*. As a practitioner he will find that he becomes often painfully concerned in the deepest interests of families and society, through the threatened or actual ravages of scrofulous diseases. The extensive prevalence of the scrofulous cachexia,—the great and almost inevitable mortality of the scrofulous diseases themselves, when completely developed, stamp the morbid state associated with them as a topic which at the outset of the student's career ought to engage a large share of study. Most assuredly the physician will have to turn his knowledge of the pathology of scrofula to account in every phase of his professional life; nay, further, when he knows, what experience has now adequately demonstrated, that the scrofulous cachexia springs from causes over which the public, rather than the medical profession, have control, he must be at once impressed with the belief, and encouraged with the hope, that when he acquires the confidence of the public in the practice of his profession, he may exercise a powerful influence for good in teaching how much they may themselves control the ravages of consumption by prudent marriages,

sanitary attention to offspring, and the necessity of free ventilation and of fresh air in places inhabited by man.

There are several circumstances which show the great influence of public sanitary measures in controlling the ravages of consumption and scrofula, when these measures are scientifically directed to the preservation of the general health, and especially when men are associated together in great communities,—an influence much greater than the best directed efforts of the medical profession can establish through their *materia medica*. It is by the mode of life as citizens of the world, in the social relations of husbands and wives, parents and children, and in the public relation of masters and workmen, that the extent and ravages of consumption and scrofula are to be controlled. It is by a strict attention to the rearing of offspring, and in the subsequent regulation of food, clothing, cleanliness, occupation, the choice of a profession, and by many other circumstances which have an obvious influence (perhaps at first sight inappreciable) on the maintenance of the general health, that our hopes of success as practitioners of medicine must rest in the prevention of that bad habit of body which develops and propagates the scrofulous diseases in civilized society.

• When Sir James Clark, in 1835, published his treatise on pulmonary consumption, he expressed some doubts as to tuberculous diseases being comparatively more prevalent at the time he wrote than they were some fifty or a hundred years before. He was also of opinion that while many circumstances favored the probability of a diminution of tuberculous diseases, there were circumstances which might materially counteract such an influence; and while he was convinced that tuberculous diseases had increased in the middle and upper ranks of life at least, he believed that, as a rule, the constitutions of the three past generations had deteriorated progressively from father to son.

The annual returns of our Registrar-General up to 1846 show a progressive increase in the mortality from consumption. In 1854, however, we find it recorded “that phthisis is twice as fatal as any other disease in England, but that within the last eight years it appears to have declined to some extent.” The inquiries of Dr. H. Greenhow show that this diminution is in a great degree due to those hygienic measures which have contributed to diminish the causes of the *miasmatic* order of ZYMOTIC diseases in general; and which have especially lessened the prevalence of those *febrile exanthematous diseases* which, by weakening the constitution, tend to bring about those conditions under which that bad habit of body is established which leads to the growth of tubercles and the development of scrofulous affections. To no kind of sanitary measure are we more indebted for this result than to the influence of vaccination in diminishing small-pox,—a disease which, of all others, seems to have tended to the development of the scrofulous cachexia as a sequel to its existence. Accordingly we find it recorded by Dr. Greenhow that “during the middle of last century, before vaccination was known, the scrofulous death-rate was more than five times as great as our present one; and the pulmonary death-

rate of the present time is 7 per cent. lower than the pulmonary death-rate of 1746-55."

While, therefore, such statements and careful observations, extended through long periods of time, show how much may be done by general sanitary measures in preventing the extension of scrofulous diseases, there is still great necessity for a careful study of the nature of these diseases, for we find them in reality *decimating* the civilized part of the world, cutting off, in some instances, as many as 35 per cent. of our metropolitan populations, and a much larger percentage of the army.

**Morbid Anatomy of Tubercle.**—The peculiar growth (erroneously called a deposit) which sometimes attends the diseases now under consideration is named *tubercle*, from its external form, occurring as it does in small nodules, isolated or grouped together, or as large irregular masses, dispersed through the textures of an organ. One essential character of *tubercle* is its incapacity to development beyond the state in which it first becomes visible, and in which state it may remain latent. It generally, however, exhibits a tendency to degenerate in various ways, involving with such degeneration the destruction of the tissue with which it is surrounded. It contains no trace of fibrous development (ROKITANSKY). The term *tubercle* is always understood to refer to adventitious masses of this nature, the type of which is found in the lungs as the essential anatomical constituent of pulmonary consumption. But the same material which composes *tubercles* in the lungs is also found in many different forms in other organs; and wherever it occurs it is described as *tubercle* or *tuberculous matter*; and *tuberculous disease*, or *tuberculosis*, is the usual designation of the specific malady of which the essential feature is the production of this peculiar matter (PAGET). A review of the opinions of recent writers, by Dr. Jenner, as to the nature of *tubercle*, in *The British and Foreign Medico-Chirurgical Review* for January, 1853, shows that the most eminent pathologists of the day are not at one as to the nature of this morbid product. "The opinions entertained regarding the nature of *tubercle*," writes Dr. Jenner, "may be divided broadly into two classes." One class of pathologists holds that tubercle is an exudation essentially morbid in character (ROKITANSKY, BENNETT, ANCELL, LEBERT). Another class holds that tubercle is merely a retrograde metamorphosis of pre-existing structures, tissue-elements, or morbid products (WILLIAMS, REINHARDT, HENLE, GULLIVER, ADDISON).

Virchow may be said to hold a doctrine combining both views. For, while he holds that tubercles are essentially composed of dead tissue-elements, whether these are physiological or morbid products, he also holds that a local process in all cases leads to an exudation of a material which is poured out during what he terms "a tuberculous inflammation," and which becomes organized to a certain extent, and then dies, breaks up, shrivels, and so becomes tubercle. This process Virchow calls *tuberculosis*; and *scrofulosis* is the general constitutional state in which this *tuberculizing* process occurs, and which commonly leads to *tuberculosis*; or, in the words

of Paget, "the relation between the two (terms) is, that the *scrofulous* constitution implies a peculiar liability to the *tuberculous* diseases." According to Virchow, *tuberculosis* is the local process in scrofulous affections in which there occurs an exudation of a material, nutritive or pathological, which develops into cells, and that these cells *tuberculize*, or undergo the tuberculous metamorphosis. *Tuberculization* is, therefore, the local process by which the metamorphosis of the elements of a part into tubercle is effected by endogenous development, atrophy, shrivelling, and desiccation of its textural element.

A *tubercle* is thus formed of the detritus of the metamorphosed and atrophied cells, with the remains of the vessels and other structures of the part in which they were seated (JENNER).

Whatever may be the view entertained regarding the exact nature of *tubercle*, this morbid product appears to us under two conditions, in forms more or less spherical, the contour of the masses being influenced—(1.) By the nature and movements of the surrounding tissue; and (2.) By the form of the part in which it first accumulates. The more recent and accurate microscopic observations which have been made into the nature and seat of the tuberculous deposit serve but to establish and confirm the more crude but scientific generalization made by the late Sir Robert Carswell, when he wrote that "the *free surfaces* of mucous membranes form the chief seat of tuberculous deposit." It is necessary, however, to extend the significance of the term *free surface*, and make it now apply to the ultimate and microscopic *cul-de-sac* terminations of mucous tubes.

*Gray* and *yellow* tubercle-masses are the names by which such deposits are described, and they are first visible in the form of roundish granulations about the size of millet-seeds, and isolated, or in groups of nodular masses of more or less irregular form.

The *Gray tubercle* is tough, soft, and compressible, of a pearly-gray color, and semi-transparent. Microscopically, it is seen to be composed of irregular-shaped bodies, approaching a round, oval, or triangular form, and varying in size from  $\frac{1}{8000}$ th to  $\frac{1}{2000}$ th of an inch. These sometimes appear to be embedded in a hyaline adhesive basis-substance, infiltrated with granules and molecules varying from a point scarcely measurable in size to the  $\frac{1}{8000}$ th part of an inch in diameter. The most characteristic semi-transparent gray granulations appear to contain more of the hyaline basis or connecting substance than of formed elements, the whole field of view being more transparent, and the elements less well expressed or defined. Acetic acid (weak, one part to four or six of water) dissolves many of the granules, and renders all the corpuscles more transparent, while a solution of potash completely dissolves them.

Various opinions are entertained as to whether or not *tubercle-masses* ever consist of *tubercle-cells* with *nuclei*. Rokitansky holds that there are cells present containing one or more nuclei, and which indicate an endogenous development and growth of the elements just noticed. Gulliver and Vogel believe in the existence

of such nucleated cells; Bennett, on the other hand, has never been able to discover nuclei in the corpuscles of tubercle. By many these cell-like elements are described and believed to be nuclei (BENNETT, SCHROEDER VAN DER KOLK, ROKITANSKY, PAGET, and SIEVEKING); and the view now related as entertained by Virchow, how existing tissue-elements may tuberculize, renders it highly probable that they may be the nuclei of epithelial cells, as suggested by Van der Kolk, or of other cell-elements, normal or pathological, as believed by Virchow; or they may be cell-particles, like nuclei, of slow formation and without any tendency to reproduction, but tending to disintegrate or break down into molecules, as described by Bennett. Paget enumerates the elements of tubercle as follows:

“1. Molecules, granules, and oil-particles, usually of small size and extremely predominant in *yellow tubercles*. 2. Nuclei of cytoblasts of various shapes and structures, but all degenerate or defective; some glittering, hard-edged, wrinkled, and withered; others granular; and few or none with distinct nucleoli. 3. Nucleated cells, similarly misshapen, withered, or granular. 4. Certain compound cells, as described by Van der Kolk, and consisting of epithelium charged with the nuclei which become the common tubercle-corpuscles.”

When the masses of gray tubercle exist in the substance of the lungs, their resemblance to millet-seeds has sometimes procured for the deposit the name of miliary tubercles; and when the lung is cut through, the elastic nature of its texture causes it to contract upon itself, so that the parenchyma recedes from the tubercle-deposits visible on the surface of the section, and the deposits appear slightly raised from the cut surface, and the finger may feel them as little resisting bodies set in the lung (PAGET). When examined with a moderately magnifying hand lens, the borders of these masses are seen to be irregular, with short projecting processes. The contraction of the lung and the consequent squeezing of the *tubercle-mass*, probably render these appearances secondary forms assumed by the gray granulations.

*Yellow tubercle* is of various shades of color, and occurs in masses of variable size, generally larger than the *gray* deposits. These masses are opaque, friable, and of a cheesy, lardaceous consistence. The masses of yellow tubercle are more commonly grouped so close together that the movements of the lungs cause them to become fused in uniform tubercular masses half an inch or more in diameter. Microscopically, *yellow tubercle* contains a much greater abundance of fine molecules than the gray tubercle, and there are also present in it elements similar to those in gray tubercles, which are shrivelled, indented, and wrinkled. There are thus no positive or characteristic morphological elements in tubercle, and the pathologist is therefore obliged to make the microscopic diagnosis of tubercle by a process of exclusion or elimination. For example, if he finds that the substance he is examining consists of the elements just enumerated, and that there are *no bloodvessels nor blood-spaces* in its



interior, and that there is nowhere in it any fibrous matrix inclosing cells, he is surely dealing with a tubercular formation. But, on the other hand, if he finds, besides these elements, a fine vascular provision for its nutrition and growth, with cells of a fully developed kind, and others degenerating, with blood-spaces, and having an areolar matrix-tissue, then he is most probably dealing with a malignant infiltrating growth, or a pneumonia in the lung, between the gradual invasion of which and tubercle there are points of analogy as well as of difference. The walls of the pulmonary artery are thickened in both; also the lining membrane of the bronchial tubes. In pneumonia, also, as well as where tubercle is being developed, the air-cells are increased in capillary action, followed by obstruction and breaking up of tissue.

The developmental origin of tubercle is to be sought for, in the first instance, as a growth from the elements of the surrounding tissue, nourished by the adjoining capillaries; and if an exudation of a fluid kind is present, its infiltration assumes the granular form of albuminoid elements, and is incapable of further growth, but capable of receiving a deposit of earthy salts in its substance, or of otherwise degenerating.

The fatty degeneration of tubercle seems to be a subsequent process, associated with a softer state of the mass in which the organic cell-elements still remain. To this oily or fatty degeneration is due the *yellow color* of tubercle; and hence, also, this yellow form is regarded as a secondary form to the gray granulation. It is in reality a degeneration or retrograde change. And this oily degeneration is not so favorable a local change as that by which the tubercle already formed wastes and *dries up*, while its further development ceases, and the mass assumes a cartilaginous-like consistence. At the same time calcareous salts are deposited in the mass, which then hardens or cretifies.

In the *gray tubercle* this salutary change is uniform throughout the mass; in the yellow tubercle the calcareous salts are usually found in larger amount round the peripheric portion of the tubercle, closing in a pultaceous friable substance, composed of free fat-granules, aggregated globules of fat, brown or black pigment-masses, and plates of cholesterine.

Vogel, Wedl, Virchow, and several other first-rate observers, are agreed that "an organized new formation constitutes the basis of the pathological structure known as tubercle."

To verify this the study of tubercle as a morbid growth ought to be commenced with the serous membranes, as in the arachnoid or pleura. In these textures we are not so apt to regard half-destroyed tissues as new products—a risk we always run in the examination of pulmonary tubercle.

Few objects of morbid anatomy have been submitted to more minute research than "tubercle;" and there is scarcely any organ where the growth may not occur. Considerable differences of opinion have been entertained respecting the relation of the two varieties of tubercle which have been described. Laennec taught, and it has been the general belief since his time, that the *gray tubercle* is



the earliest stage of the deposit which subsequently becomes converted into *yellow*. Dr. Walshe has also especially investigated this point, and teaches a similar doctrine. Rokitansky, in the first edition of his great work on morbid anatomy, regarded the *yellow* and the *gray* as essentially distinct forms, and considered it an error to believe that the one is converted into the other. Rokitansky now affirms that *gray tubercle* is sooner or later converted into *yellow tubercle*, as Laennec first taught, and which is now the common belief. When the gray tubercle does not pass into the yellow, it withers away. It loses its lustre, and becomes dry, dense, and hard, and shrivels into an indistinct shapeless fibrous-like mass. Sometimes such a change is associated with a calcareous degeneration, the surrounding tissue in which it is embedded becoming dark with the deposit of pigment.

It is now believed and taught by Rokitansky, Virchow, and others, that certain abnormal products, not apparently at first tubercular, afterwards *tuberculize*, and assume the appearance of yellow tubercles. To this change Virchow proposes to restrict the term "*cheesy metamorphosis*." The metamorphosis of *gray* to *yellow* tubercle generally commences in the centre of the growth, and subsequently the yellow tubercle undergoes most important secondary changes in a sanative point of view. These changes consist—(1) in softening; (2) in cretification or calcification; but it is not to be understood that the first of these two changes always passes into the second.

The descriptions already given, and the metamorphosis about to be noticed, illustrate the distinctive and peculiar characters of tuberculous growth—namely, its early degeneration and abortiveness. It seems to be a substance which acquires a certain stage of organic development, when it is arrested in its course, recedes, and degenerates. Such characters may be read from the shrivelled or granular state of the so-called free nuclei and the barren cells. The changes about to be noticed show a still more retrograde decay (PAGET).

When tubercle softens, its substance breaks down into a tolerably uniform creamy pus-like fluid, in which are to be seen an immense number of fine granular points or molecules. The basis-substance appears to soften first; but in some kinds of softening much of the corpuscular forms are retained in a thin, whey-like, flocculent fluid, approaching in character to what is known as scrofulous pus, both microscopically and to the naked eye. The softening appears on the whole due to the breaking up of the corpuscular elements, as well as of the basis-substance. When softening begins in the centre of the yellow crude masses, it leads one to believe that fluid may transude from the surrounding effusion or hyperæmic state, and penetrate to the centre of the whole mass, where it may accumulate and increase the softening, without becoming developed into the visible cell-elements of tubercle. As each tubercle or group of tubercles undergoes this softening process, the softened mass occupies a cavity, which thus becomes an abscess, though not a purulent one, and the tissue involved by the growth of the tubercle is

destroyed. It dies with the increased growth and softening of the mass. During this process of softening, tuberculous matter continues to grow in other portions of the organ, and when it occurs in the lung, generally from above downwards, so that excavations or cavities are found at the apex of the lung, while lower down yellow tubercle-masses, beginning to soften, exist; and still lower down the *yellow tubercle* is found in a crude state, as if newly changed, or in the process of being changed from the gray granulations which are disseminated through the base of the lung.

Pathologists are now agreed that the production of tubercle is quite independent of inflammation, in the ordinary acceptation of that term, implying thereby the presence of pain, heat, swelling, redness, lymph, or pus; but that in the great majority of cases inflammation is set up round the tubercle-masses, and plays an important part in promoting further growths of tubercle, which proceeds in a rapid course to softening and destruction of tissue. The phenomena of the growth of tubercle are also associated with the existence and supply of material which forms the fibrous thickenings and capsules around tubercular cavities or ulcerations. These phenomena also bring about the separation of the tubercle-mass from the surrounding parts, and if it is eliminated in any way, a cavity or an ulceration remains. When thus inclosed by fibrous thickenings or capsules, the softened tubercle may undergo the process of *cretification* or *calcification*; in other words, it becomes converted into a greasy, fatty, chalky mass, which gradually becomes hardened into a brittle substance. There appears to be either a deposit or a liberation of calcareous particles in the mass, for the elements become mixed up with gritty particles of earthy salts; and at the same time there is absorption of the animal part, so that the original size of the tubercle-growth is diminished. When tubercle is thus completely calcified, a thin section presents a granular appearance, but with no definite forms, combined with more or less pigment.

Tubercle has been analyzed by many chemists. The analysis by the late Dr. R. M. Glover, of Newcastle, appears to be the most accurate and extensive.\* His conclusions are as follows:

“1. That tubercle consists of an animal matter mixed with certain earthy salts.

“2. That the relative proportion of these varies in different specimens of tubercle. That animal matter is most abundant in recent, and earthy salts in chronic tubercle.

“3. That the animal matter certainly contains a large amount of albumen, while fibrin and fat exist in small but variable proportions.

“4. The earthy salts are principally composed of the insoluble phosphate and carbonate of lime, with a small proportion of the soluble salts of soda.

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\* This analysis, to be satisfactory, should be made on tubercles at different periods of life. The tubercle of the *young* strumous subject is believed to differ greatly from the tubercle-growth found in *advanced life*, especially in a gouty habit.

“5. That very little difference in ultimate composition has yet been detected between recent tubercle and the other so-called compounds of protein.”

Thus much is known regarding the histology of tuberculous growths, and is here stated minutely, because we have the testimony of most experienced morbid anatomists, such as Bennett, Paget, and many others, that the microscope alone can decide as to the nature of those which closely resemble tubercle when examined by the naked eye. Indeed, such are the vague definitions given of *tubercle*, that, in the words of Bennett, “every morbid anatomist must frequently have experienced much difficulty in endeavoring to determine by the naked sight whether a certain morbid product be or be not tubercle.”

[The views of Virchow respecting the nature of tubercle may be summarized as follows: “Tubercle is a granule or a knot, and this knot constitutes a new formation, which from the time of its earliest development is necessarily of a cellular nature, and, generally, just like all other new formations, has its origin in connective tissue; when this has reached a certain degree of development, it constitutes a minute knot within this tissue, and when it is at the surface, projects as a little protuberance, and consists throughout its whole mass of small uni- or multi-nuclear cells. The especial characteristic of this formation is its extreme richness in nuclei, so that when it is examined as it lies embedded in the investing tissue, at the first glance there appears scarcely anything else but nuclei. By isolating the constituents of the mass, either very small cells with one nucleus are obtained—and these are often so small that the membrane closely invests the nucleus—or larger cells with a manifold division of the nuclei, so that twelve, twenty-four, or thirty are contained in one cell, in which case, however, the nuclei are always small, and have a homogeneous and somewhat shining appearance. Tubercle is closely related to pus in this, that it has the smallest nuclei and relatively the smallest cells of any structure, and is always a pitiful production, a new-formation from its very outset miserable. From its commencement it is like other new formations, not unfrequently pervaded by vessels, but when it enlarges its little cells are so closely pressed together that the vessels gradually become completely impervious, and only the larger ones, which merely traverse the tubercle, remain. Generally fatty degeneration sets in very early in the centre of the knot, where the oldest cells lie, but usually does not become complete. Then every trace of fluid disappears, the corpuscles begin to shrivel, the centre becomes yellow and opaque; and a yellowish spot is seen in the middle of the gray translucent granule. This is the beginning of the *cheesy metamorphosis*, which subsequently characterizes the tubercle. This change advances from cell to cell farther and farther outwards, and it not unfrequently happens that the whole granule is gradually involved in it. The form is produced by the growth of the tubercle from single cells of connective tissue, by the degenerative proliferation of single groups of connective tissue corpuscles. It appears at once in the shape of a granule. As soon as it has once attained a certain size, as soon as the generation of new corpuscles which develop themselves out of the old histological elements by a continual succession of division, at last lie so close to one another as to cause a mutual arrest of development, and gradually induce the disappearance of the vessels of

the tubercle, and thereby cut off their own supplies, then they begin to break up, they die away, and nothing remains behind but debris—shrunk, disintegrated, cheesy material. The cheesy transformation is the regular termination of tubercle; but, on the one hand, it is not the necessary one, inasmuch as there are rare cases in which tubercles, in consequence of their undergoing a complete fatty metamorphosis, become capable of reabsorption; and on the other hand, the same cheesy metamorphosis befalls other kinds of cellular new-formations; for pus may become cheesy, and likewise cancer and sarcoma. This metamorphosis, therefore, being common to more than one formation, cannot well be set down as the criterion for the diagnosis of any particular structure, such as tubercle; on the contrary, there are certain stages in its retrograde metamorphosis where it is not always possible to come to a decision. There are periods in the course of development when that which is inflammatory and that which is tuberculous can with precision be distinguished from one another; but at last there comes a time when both products become confounded, and when, if one does not know how the whole arose, no opinion can any longer be formed as to what its nature is. The nature of tubercle cannot be studied after the period when it becomes cheesy, for from that time its history is identical with the history of pus which is becoming cheesy; an earlier period must be chosen when it is really engaged in proliferation. If the cells which are the real constituents of the tubercular granule are compared with the normal tissues of the body, the most complete correspondence between them and the corpuscles of the lymphatic glands will be seen, and this is a correspondence which is neither accidental nor unimportant, for it was known long ago that lymphatic glands have an especial tendency to undergo the cheesy degeneration (*Cellular Pathology*, lect. xx).]

**Nomenclature.**—The term “tubercular deposit” can scarcely be regarded as correct, although in common use. The expression ought rather to be “scrofulous or *tubercular form of inflammation* ;” just as we have been accustomed to use “tubercular meningitis” as synonymous with “scrofulous tubercular inflammation of the meninges.”

In the *peritoneum* the seat of scrofulous inflammation is immediately beneath the serous coat, where sanguineous congestions and minute extravasations first take place during the rapid formation of the tubercular granulations.

In the *mucous membrane* of the alimentary canal the seat of scrofulous inflammation is principally in the lower part of the small intestines. The tubercle grows originally in the submucous tissue and elevates the mucous membrane stretched over the granulations. They project in the form of nodules assembled together in several groups. New granulations spring up between the older ones; and as these growths degenerate and soften rapidly in the intestines, a fusion of the whole very soon takes place. The circulation in this part of the intestine is impeded, and its vitality is gradually destroyed; its mucous membrane is lost, and ulcerations form, whose shapes are decided by the form of the original growth. Thus: A single tubercle-nodule gives a crater-like ulcer. A number produces extensive ulceration, with irregularly excavated borders, the growth of the tubercle-masses being continued at the margins of the ulcers;

and hence the thick border, with particles of yellow crude tubercle embedded in it, so characteristic of these ulcers. Similar material may also be seen on the floor of the ulcers, the softening of which leads to perforation of the peritonæum.

A considerable portion of the scrofulous exudation may transude in a fluid state, in the first instance, through the capillaries, and collect in those places outside the vessels that offer least resistance (BENNETT). While, therefore, *infiltration* more or less extensive, as described by Baillie, is the first condition in which the exudation can be observed to exist, *tubercle* may be observed to accumulate on the free surfaces of mucous and serous membranes, or on the outer surface of minute bloodvessels. While, therefore, scrofulous material does not differ in its seat from the simple or cancerous exudations, when these form minute growths in the lungs or other parts, yet there is an important difference in regard to their relation with the bloodvessels, which requires to be specially noticed.\* It was first pointed out by Dr. William Stark that when attempts are made to inject a tuberculated lung, the finest injection will not reach, far less penetrate, the *tubercle-masses*. Bloodvessels which are of a considerable size at a little distance from tubercle-masses speedily become contracted, so that a large vessel, which at its origin measured nearly half an inch in circumference, could not be cut open further than one inch, and that when cut open, such vessels presented a very small canal filled by a coagulated substance. Schröder Van der Kolk made similar experiments. He sometimes found that large vessels remained pervious which crossed a tuberculous cavity in a lung, but that all the small or capillary branches which adhered and were given off from the larger trunks were obstructed and impervious. These observations lead to the conclusion that obliteration commences in the smaller vessels and proceeds to the larger trunks; and are of some importance with reference to the process of softening, and other changes which are observed to take place in this *non-vascular* growth. The process of *softening* is not so uniform in its progress as the notice already given might lead one to suppose.

It appears difficult in every instance to ascertain at what part softening commences, so that it is incorrect to give a general de-

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\* [According to Virchow, tubercle is distinguishable from cancerous growths by the circumstance that these, being more highly organized, contain large, voluminous, say often gigantic corpuscles with highly developed nuclei and nucleoli, while tubercle is always a mean production. Lebert is of opinion that cancer and tubercle do not exclude one another, but may, and often do, exist simultaneously in the same person. He found tuberculosis coexisting thirteen times in 45 cases of cancer of the uterus, twice in 34 cases of cancer of the breast, twice in 9 cases of cancer of the œsophagus, and twice in 18 cases of cancer of the bones. Cancer, though, is rarely developed in the course of the progress of tubercle, and it is doubtful whether their progress is ever coincident; the development of one suspends or stops the advance of the other (*Traité de l'Anat. Pathologique*, t. i, chap. xvi). Dr. Pollock mentions a case where the two diseases, cancer and tubercle, were undoubtedly combined. The cancerous growth was in the lungs and mediastinum, which caused death, and on post-mortem examination there was certain evidence of pre-existing pulmonary tubercle. Here the tubercular had been superseded by the cancerous disorder (*The Elements of Progress in Consumption*, p. 289, 1865).]



scription of this change as always commencing in one place. In one case it may begin in the centre and proceed to the circumference of the growth; in another it may begin at the circumference and go round the whole mass, detaching it from the surrounding textures; in a third case it may begin at once in the centre and at the margins; and in other instances it has been observed to commence at the same time in several parts of the substance of the tubercular mass; and this is especially the case where the masses are large (CRAIGIE, BENNETT). The portion or spot about to soften loses its firmness and becomes friable, the cells swell and break up, adding thus by their solution to the diffuency of the mass. Inspected with a common hand lens, the mass seems as if perforated here and there with holes, and this softening extends by degrees throughout the whole growth. The air-vesicles necessarily are destroyed by this process, and the terminal extremities of the bronchi are amongst the first structures to suffer. The mucous membrane which covers them is red and villous in the vicinity of the softened part, and as tuberculosis proceeds, the whole of the pulmonary mucous membrane presents the anatomical characters of chronic bronchitis. The softening of the growth and the inflammation of the textures seem to act and react upon each other; liquefaction seems to extend more rapidly when local inflammation is set up, and the softenings, on the other hand, appear to augment the local reaction.

**Constitutional Origin of Scrofula.**—Departing from the limited consideration of the subject which *morbid anatomy alone* affords, clinical and pathological research tends more and more to confirm the belief in the constitutional origin of scrofula. There is undoubtedly a predisposition to scrofulous growths, either hereditary or acquired; and that there is a bad habit of body—a cachexia—which precedes the development of tubercle, no one can deny.

The development of tubercles, wherever found, is undoubtedly a local lesion which indicates a *constitutional disease*. It has been hitherto, however, the custom to name the local lesions as the *disease*, rather than to describe the constitutional state. Thus, when the lesions have been most marked in the bones and glands, the name of *scrofula* has been given to the condition; when the lungs are the site of the deposits, *phthisis* or *consumption* is the name by which the condition is familiar; and when in the glands of the mesentery, it has been called "*tabes mesenterica*;" and in the meninges of the brain it has been named "*hydrocephalus*," or "water in the head." The constitutional disease under which all these affections are now comprehended is named "*scrofula*," and is shortly defined as "*a constitutional disease resulting either in the deposit of tubercle or in specific forms of inflammation and ulceration*," as stated in the second part of the definition previously given.

The changes in the general system by which the scrofulous cachexia is brought about are apparent in the physical condition of the patient, and in the exercise of some of the vital functions especially connected with nutrition. To the late Dr. Tweedy Todd, Sir James Clark, and Professor Bennett, the profession is



principally indebted for the clear and earnest elucidation of *scrofula* or *tuberculosis* considered as a constitutional affection; and on our knowledge of it as such rests our only hope of success in the prevention and treatment of this most formidable scourge of civilized society.

Many observations have been made and statements recorded with the view to connect scrofula with morbid states of the blood, but hitherto no constant morbid condition of the circulating fluid can be said to be peculiar to the disease. As Dr. Bennett observes, we must look to something beyond—we must look to the pabulum which ministers to the nutrition of the body itself through the blood; for with an impoverished state of that fluid there is doubtless an impoverished state of the tissues. But there are some curious and detached observations which, when connected together in certain pathological relations, appear to throw more light on the nature of the constitutional state which leads to the development of scrofula than any single observation of individual authors. These I would thus shortly enumerate:

1. There is to be noticed the albuminous character of the fluid material which infiltrates the tissues of an organ previous to the process of *tuberculization*, by which this infiltration is in part changed by coagulation into *gray tubercles*, each of which may block up from three to twenty air-vesicles in the lung.

2. There is in some cases, as shown by Drs. Alison, Williams, Bennett, and Rokitansky, an obvious affinity between the lymph of the blood and tubercle.

3. There is an albuminosity or venous state of the blood which is considered by some as peculiar to the scrofulous state of the constitution.

4. There is the peculiar state of the blood, amounting to an appearance of leuchæmia, which immediately succeeds digestion in healthy persons, as observed and described by Dr. Andrew Buchanan, of Glasgow, in *The Transactions of the Philosophical Society* (vol. ii) of that city, resembling the molecular and corpuscular elements of chyle or lymph, and consisting of fat emulsified with albumen. To this substance he gave the name of *pabulin*, and which is still further elaborated in the blood, in the glands, and in the lungs, before it takes part in the general morphological changes connected with nutrition.

5. The observations of Dr. Acherson, of Berlin, and of Dr. Bennett, relative to how nutrition may be impeded by diminishing the *molecular* state of the nutritive elements, and improved by increasing them.

6. The observations of Panum and Parkes relative to the precipitation of albumen by acids and neutral salts, in which Dr. Parkes especially shows that the albumen as it exists in the serum of the blood is usually in that condition in which it is most easily precipitated by acids and chloride of sodium (*Med. Times and Gazette*, July, 1850 and 1852).

7. The excess of intestinal acidity in the alimentary canal of phthisical patients, as shown by Dr. Bennett, by which, under

some conditions, the albuminous constituents of the food are rendered easily soluble, whilst the alkaline secretions of the saliva and the pancreatic juice are more than neutralized, and so rendered incapable of transforming the carbonaceous constituents of food into oil, or of so preparing fatty matters introduced into the system as will render them easily assimilable. Hence an increased amount of albumen enters the blood compared with fatty elements.

According to the observations of Mr. Jonathan Hutchinson, acid eructations were present in 62 per cent. of the cases of dyspepsia which preceded the deposition of tubercle, and were a prominent symptom in 46 per cent. of cases reported on by him in an admirable paper on the forms of dyspepsia preceding and attending phthisis (*Med. Times and Gazette*, vol. x, 1855).

Any one of these statements considered by itself does not seem of much importance, but considered as a whole, in their relation to nutrition and their influence upon morphological changes between the solids and the fluids of the body, they leave very little room for doubt that the bad habit of body in scrofulous affections associated with the growth of *tubercle-matter* must be established in the first instance through the digestive processes, as first described by the late Dr. Tweedy Todd under the name of *strumous dyspepsia*, and which has been since so fully described by Sir James Clark, Bennett, Hutchinson, and others.

The more closely these links of circumstantial evidence can be bound together the more intimate a pathological relationship will be found to exist between the albuminous constituents of the food, the blood, and tubercle, and the saline constituents of the circulating fluid, the mal-assimilation of food, and the waste of the tissues, as connected with the development of the bad habit of body associated with scrofula.

Although it must be confessed that we do not fully understand the living processes by which all the molecular changes take place to which I have just adverted, yet there can be no doubt that in the seven statements enumerated we obtain some glimpses of a rational pathology, which may yet tend to explain the very complex *constitutional morbid state* which precedes and is associated with the growth of tubercle in scrofulous affections or with the specific inflammations and ulcerations of scrofula.

**Healing of Local Lesions in Scrofula.**—It is of the greatest importance to study how some of the most unmanageable and hopeless diseases will sometimes spontaneously become cured, and how the local lesions will heal. That scrofula sometimes tends to these results we have abundant testimony daily afforded in almost every post-mortem examination of those who die of *phthisis pulmonalis*. Laennec, Carswell, Clark, and Bennett have recorded their testimony on this interesting point; and the practical result of their observations shows that if the further growth of tubercle can be arrested, the masses already existing may silently retrograde, become absorbed, or diminish in size, and the part cicatrize, or it will remain latent as a cretaceous mass in the lung.

**Symptoms of Scrofula, or of the Cachexia which Precedes and Accompanies the Growth of Tubercle.**—Ever since scrofula has become more studied as a constitutional affection, it has been rendered more apparent that impairment of the digestive organs is the primary disorder of function which ushers in the cachexia, and that a certain form of dyspepsia is not only present in the hereditary strumous constitution, “but is capable of generating the bad habit of body, and of leading ultimately to the deposition of tubercle.”

It was first observed by Dr. Wilson Philip “that there were some forms of indigestion which ended in phthisis;” and it was subsequently distinctly stated by Lepelletier that the causes of scrofulous disease were referable—(1.) To those agents which impair the assimilative action; (2.) To the elements of nutrition being insufficient, by the influence of bad diet, impure air, deficient exercise of the general functions, and obstruction of the functions of excretion.

It was reserved, however, for the late Dr. Tweedy Todd, as already noticed, to show that in cases in which the strumous cachexia was present a particular form of dyspepsia prevailed, to which he gave the name of “strumous dyspepsia.” Subsequently, the views of Dr. T. Todd have been more or less fully accepted and developed by Sir James Clark, Bennett, Ancell, and others in this country; but the most precise and most recent exponent of them has been Mr. Jonathan Hutchinson, in two valuable papers in the *Medical Times and Gazette*, vol. x, 1855. Mr. Hutchinson, from statistical evidence, shows that the tuberculous cachexia is preceded by a *peculiar form of dyspepsia*; in other words, that there is peculiar form of dyspepsia which has a natural tendency to end in phthisis; and that it is a form of dyspepsia not common to other conditions of the system—different, for instance, from that which precedes and attends gout, chlorosis, or cancer, although accurate observation is wanting on the exact symptoms of dyspepsia which are associated with these diseases.

The conclusions which have been arrived at by Mr. Hutchinson are as follow:

1. In a very large majority of cases of established phthisis a condition of well-marked dyspepsia is present as a complication. Out of fifty-six cases it was absent in four, present mildly in twenty-one, moderately in twenty-two, and severely in nine.

2. Of the form of dyspepsia most common in established phthisis, the prominent symptoms relate to difficulty in the assimilation of fatty matters. The patient acquires a remarkable distaste for all fats, which occasionally extends itself to sugar and even to alcohol; he suffers much from “biliousness,” heartburn, flatulence, and, above all, from acid eructations after taking food; everything he takes “rises acid,” to use a common expression of such patients, but more particularly everything containing fat, oil, or sugar (*butyric fermentation*).

The *dislike for fat* was present in 71 per cent. of the cases of confirmed phthisis, and had existed through life in 48 per cent. It invariably produced what is commonly called “biliousness,” and

“rose acid” from the stomach. The fat of fresh meat was generally the first to disagree; then salted meats, such as bacon, and lastly butter; but many could enjoy butter who could not touch any other kind of animal fat. Such an intense dislike to an important element of a mixed diet indicates a deficiency on the part of the patient to digest it. Coincident with the development of such symptoms *emaciation* usually becomes apparent, the adipose tissue of the body already existing being reabsorbed by the blood, to supply the respiratory element deficient in that fluid. The fat disappears from the subcutaneous tissue, hollows of the cheeks, orbits, and mammary glands, and other parts where it generally abounds in quantity, just as it disappears from the body of an animal during hibernation. The patient now gradually loses weight and becomes thinner. Other tissues of the body, such as the muscles, skin, and areolar tissue, subsequently begin to be used for a similar purpose, till the patient is wasted to a living skeleton.

The *acid eructations* were present in 62 per cent. of the cases, and were a prominent symptom in 46 per cent., small quantities of an extremely acid fluid being repeatedly brought up into the pharynx at various periods after taking food, and in bad cases these eructations were persistent throughout most of the day, and almost always attended by heartburn. *Sick headaches* and *biliousness* were very commonly present.

3. The majority of cases of *phthisis pulmonalis*, whether hereditary or otherwise, are *preceded* by a well-marked stage of dyspeptic symptoms. By this it is meant that symptoms referable to the digestive organs have preceded those connected with the lungs. Out of the fifty-two cases in which, as has been shown, dyspepsia was present, it had followed pectoral symptoms in nine, was developed nearly coincidently with them in ten, and preceded them in thirty-three.

4. The symptoms of the dyspepsia premonitory of phthisis are the same in character with those which complicate it when developed. The very earliest are alterations in the tastes, and the most constant of all is a disrelish for fat.

5. The subjects of phthisis have in a large number of cases had peculiarities of likes and dislikes for different articles of food even from very early life, and whilst seemingly in perfect health. Amongst those peculiarities the dislike of fat, often amounting to extreme aversion, ranks first. Thus it might be predicated of a family in which one child distinguishes itself from its brothers and sisters by its refusal to eat fat, that it will, *cæteris paribus*, be more prone to become the subject of tuberculous disease, in after-life, than any of the others.

[Dr. Pollock gives only a qualified adhesion to the views of Hutchinson, Bennett, and others, of the frequency of acidity of the primæ viæ in the earliest stages of phthisis. “Of 50 cases of incipient consumption, observed with peculiar care to elucidate this point, in 3 only was acidity a marked symptom; in 27 the digestion was more or less impaired (generally slightly); and in 20 it was decidedly good” (*The Elements of Prognosis in Consumption*, p. 82). The acid order of digestion, he thinks, has only a constant relation to the acute form of the disease, of which it

constitutes a distinctive character. Laennec found the stomach diseased in four-fifths of his phthisical autopsies; Louis' figures are about the same, with softening of the gastric mucous membrane in one-tenth. Dr. Pollock's experience would give even a higher proportion than this (*l. c.*, p. 313). The frequency of gastric disorder is said to diminish with age, and in senile phthisis to be rare.]

Besides these indications afforded by the functions of digestion, there is a peculiar modification of the whole organization, as regards structure, form, and the exercise of functions generally, which impresses distinctive characters on the scrofulous cachexia when it is of hereditary origin. Miller gives the following concise description of this organization:

"The complexion is fair, and frequently beautiful, as well as the features. The form, though delicate, is often graceful. The skin is thin, of fine texture; and subcutaneous blue veins are numerous, shining very distinctly through the otherwise pearly-white integument. The pupils are usually spacious; and the eyeballs are not only large but prominent, the sclerotic showing a lustrous whiteness. The eyelashes are long and graceful—unless *ophthalmia tarsi* exist, as not unfrequently is the case; then the eyelashes are wanting, and their place is occupied by the swollen, red, unseemly margin of the lid.

"In the phlegmatic form the complexion is dark, the features disagreeable, the countenance and aspect altogether forbidding, the joints large, the general frame stunted in growth, or otherwise deformed from its fair proportions. The skin is thick and sallow; the eyes are dull, though usually both large and prominent; the general expression is heavy and listless; yet not unfrequently the intellectual powers are remarkably acute, as well as capable of much and sustained exertion. The upper lip is usually tumid, so are the *columna* and *alæ* of the nose, and the general character of the face is flabby; the belly inclines to protuberance; and the extremities of the fingers are flatly clubbed, instead of presenting the ordinary tapering form" (*Principles of Surgery*, p. 55).

The growth of the body is generally unsteady in its progress; very often it is slowly and imperfectly developed; in other cases it is unusually rapid, and particularly towards puberty. The physical powers are generally feebly developed, and incapable of sustained exercise. The muscles of the limbs, though full, are soft, flabby, and weak, and have neither the form nor the firmness of health. The general circulation is feeble, the weak pulse and cold extremities indicating the debility. Digestion is feebly and imperfectly performed, the bowels being irregular, and more frequently slow in their action than the reverse.

The mucous membranes generally are very susceptible to disordered action. Discharges from the nose, ears, or eyes are not uncommon; the tonsils enlarge, and the air-passages inflame from the slightest causes. The insensible perspiration is defective, and is said to be unduly acid, and loaded with sebaceous matter; while, on the other hand, copious partial perspirations are common, particularly on the feet, where the odor is often fetid.

In children the impaired functions of the digestive organs are indicated by increased redness of the tongue, especially toward the



extremity and along the margins. The anterior part is thickly spotted with small red points of a still brighter color, the central portion being more or less furred, of a white or of a brown color in the morning. Thirst prevails; the appetite is variable, more frequently craving than deficient, seldom natural; and the breath is fetid. The bowels are occasionally loose, and the evacuations are always unnatural, generally of a pale grayish color, of the consistence and appearance of moist clay; and they are often mixed with mucus and partially digested food. While the urine is often turbid, and sometimes high-colored, it is also often abundant and pale. The skin is generally harsh and dry, or subject to cold perspirations, particularly the hands and feet, which are habitually cold; and copious partial night-sweats are common. The sleep is seldom sound; the child is restless, talks in his sleep, or grinds his teeth (T. TODD, CLARK).

Subsequently, when the disordered state has continued for some time, the internal fauces become full and red, and inflammatory sore throats are common, the tonsils often becoming permanently enlarged. The nostrils are generally dry, or thick mucus may be discharged in large quantity. Epistaxis occasionally occurs.

The nervous sensibility is sometimes greatly increased, and the intellectual functions are often performed with a preternatural degree of activity. So frequently is this observed that it has become a popular belief and saying regarding such a child "that it may be too wise to live long."

Inflammation in any tissue in a scrofulous subject generally assumes a slow chronic type, accompanied with little pain or heat; and suppurating parts heal very slowly.

When the scrofulous cachexia becomes fully developed into *phthisis pulmonalis*, the duration of the disease ranges from about nine months to two years; but in what are called "acute" cases of consumption it may terminate fatally in three or even in two months, and occasionally in as short a period as three weeks or even less.

[There are great difficulties in the way of determining the actual average duration of phthisis; hence the difference of opinion amongst skilled observers. The frequent latency of the disorder, the obscurity of the early symptoms, the greater rapidity of its progress in some countries and climates than in others, a neglect to separate the several varieties of the disease in calculating its continuance, the exclusion of certain modifying conditions, as age, pregnancy, and lactation, are all sources of inevitable fallacies. Portal considered that phthisis "might last from eleven days to forty years;" Laennec states the mean duration of his cases at 24 months; Louis and Bayle (314 cases observed), 23 months; Andral, 24 months; Sir James Clark, taking cases in the British upper classes, 36 months; and Dr. C. B. Williams says that the average duration is prolonged by cod-liver oil to 48 months. The most complete, philosophical, practical, and, it is believed, reliable tables on the actual duration of phthisis have been prepared by Dr. Pollock, with the help of a skilled mathematician, from the observation of 3566 cases, and should be carefully studied, particularly as an aid to the prognosis of the disorder. A few only of the general results can be given here, the reader

being advised to consult the original work. It would appear that, viewing the disease as a whole, in which are included all its varieties, it lasts much longer than has been generally stated by writers, or than is believed by the public. There are many instances of an early invasion of the disorder with subsequent subsidence of the symptoms, and long tolerance of the deposit. It often manifests an undoubted tendency to extreme chronicity. Dr. Pollock maintains, that owing to a greater accuracy of diagnosis, the revelation of a lengthened duration of consumption, formerly suspected but now certified, has taken place, and that of this fact we may be as secure as of any other observation of the natural history of disease. The average duration of all his cases taken together, *while under observation*, was *two years, six months, and three-fifths nearly*; this table includes cases of the most acute form. The table shows, too, that out of 3566 cases, only 129 had died at the end of two years and (a little over) six months. Of 690 cases of ordinary phthisis, the average duration of each case was 15.22 months.]

**Causes.**—In describing the pathology and the symptoms of the scrofulous cachexia, the cause of the constitutional disorder has already been in some measure indicated.

The tendency to the formation of tubercle in scrofula is not equally great at all periods of life, nor in all parts of the body. Tubercle in the bronchial glands, the lungs, the cervical glands, the mesenteric glands, the spleen, the pleura, the liver, the small intestines, and the brain, is most common in infancy, childhood, and early adolescence. But the occurrence of tubercle, which forms so large a portion of the lesion in all scrofulous affections, although it has been found in the fœtus, and at every period of life up to eighty, yet it will be observed that tubercle, after the age of fifteen, is more frequently met with in the intestines, the mesenteric, cervical, and lumbar glands. Scrofulous disease is frequently developed during infancy; and “I have met with several cases of infants,” writes Sir James Clark, “dying of consumption within the first year of life, in whom the lungs were not only extensively tuberculous, but contained large caverns, with all the characters of those found in the lungs of adults.” After the second year of life, pathologists agree to the universal prevalence of scrofulous disease. Age exercises a paramount influence on the generation of scrofula. It is peculiarly a disorder of childhood and youth (GUERSENT, LOMBARD, PAPA VOINE, ALISON, CLARK, BENNETT). Dr. Alison demonstrated that the mortality from scrofulous diseases in the children of the lower orders in Edinburgh and other large towns was so great that they died in the enormous proportion of forty-five or fifty to five and even three, as compared with the agricultural and upper classes. Tubercles prevail most through the third, fourth, fifth and sixth years, when the annual growth does not exceed one-tenth of the child’s weight, and the mortality declines to nearly one in a hundred. More than a fourth of those who die in the interval from birth to puberty are affected with scrofulous disease; yet the disease itself causes death in about one-sixth only of the cases (CLARK). The greatest number of deaths occur between the ages of twenty and thirty; the next in proportion between thirty and forty; the next between forty and

fifty. The mortality is at its maximum about thirty, and from that age declines.

[Phthisis, though a disorder of all ages, appears to attach itself remarkably to the age of growth and the age of decay. The first Medical Report of the Hospital for Consumption, Brompton (1849), states that the period most affected by phthisis, in all its forms, is the decade from 25 to 35 years; the second Report (1863) shows a large preponderance of cases between the ages of 20 and 30 years; on either side of this decade the numbers diminish in about an equal ratio. Out of a total of 6134 cases—

From 10 to 20 there were 1381 cases.						
"	20	"	30	"	"	2708
"	30	"	40	"	"	1444
"	40	"	50	"	"	551

Dr. Pollock has collected from various authorities instances of the frequency and mortality of phthisis in advanced life. At Chelsea Hospital, tubercles, tubercular concretions, and traces of ancient or recent cicatrized cavities occurred in the lungs of about one-half of the men examined, whose ages varied from 60 to 80 and upwards (MACLACHLAN). In 100 females over 60 years, examined at the Salpêtrière, in 51 there were cretaceous or calcareous concretions in the lungs, or other evidences of cured or existing phthisis (ROGER). Out of 852 cases of deaths from phthisis in males in the Edinburgh Royal Infirmary, 25 per cent. were above 40, and 8 per cent. above 50 years. In London, in 1845, 3624 males died of phthisis, of which 36 per cent. were over 40, and 15 per cent. over 50 years. The number of deaths from phthisis among women insured in an Edinburgh Life Office was 48 per cent. between 30 and 40, and 22 per cent. between 40 and 50 (CHRISTISON). Though after 45 years the disorder is usually slow in progress, still cases of rapid phthisis happen in advanced life; one in a male æt. 73, death in less than three months, and within six weeks of cavities forming; another æt. 73, death in six weeks (BLAKISTON); and another æt. 63, fatal in three and a half months, with cavity formed (POLLOCK).]

The broadest fact established regarding the exciting cause of scrofula is, that the domesticated animal is more liable to scrofulous disease than the same animal in a wild state. The stabled cow, the penned sheep, the tame rabbit, the monkey, the caged lion, tiger, or elephant, are almost invariably cut off by scrofulous affections—no doubt due to deficient ventilation and the abeyance of normal exercise of the pulmonary function.

[There appears to be little doubt that persons who breathe continuously air vitiated by respiration furnish a large percentage of phthisical cases. This cause is potently aided by the coincident conditions of deficient exercise, poor feeding, and, often, excessive work. Baudelocque, years ago, asserted that impure air was the chief cause of phthisis. Carmichael, in his work on Scrofula (1810), gives a number of examples of the influence of foul air and want of exercise in the production of the disorder. Lepelletier, Neill, Arnott, Toynbee, Guy, Greenhow, Baly, and others, have collected a good deal of evidence in support of this cause of tuberculosis. Of 104 compositors who worked in rooms of less

than 500 cubic feet for each person, 12.50 per cent. had had hæmoptysis; of 115 in rooms having from 500 to 600 cubic feet, 4.35 per cent. had had hæmoptysis; and in 101, in rooms of more than 600 cubic feet, 1.98 had had hæmoptysis (GUY). In the prison of Leopoldstadt, at Vienna, very badly ventilated, in the years 1834–1847, 51.4 per 1000 died from phthisis, there being 42 cases of acute tuberculosis; while in the well-ventilated House of Correction, in the same city, the deaths from phthisis were only 7.9 per 1000. The great prevalence of phthisis in the Indian jails is ascribed to bad air and bad food (PARKES, *loc. cit.*, p. 96, 2d ed.). Dr. Henry MacCormac is, perhaps, the most enthusiastic advocate of the doctrine of the effects of rebreathed air in the etiology of tuberculosis; so far does he carry his doctrine that he makes it almost an exclusive cause of phthisis. “Wherever the air, habitually respired, has been respired in whole or in part before, there tubercular deposits are found.”\* The greater relative frequency of phthisis in females Dr. Lawson attributes to their indoor occupations and sedentary habits.† The chief cause of the extraordinary frequency of phthisis in the European armies, to be presently spoken of, Dr. Parkes says “can scarcely be accounted for in any other way than by supposing the vitiated atmosphere of the barrack-room to be in fault. This is the conclusion which the Sanitary Commissioners for the British Army came to in their Report, after assigning all probable influence to exposure on duty, intemperance, syphilis, and faulty diet. The disease is found to prevail in the most varied stations of the army, and in the best climates, but where there was always the one common condition,—the impure air of the barrack system. And, as if to clench the argument, there has been of late years a most decided decline in phthisical cases in these stations (Gibraltar, Malta, Ionia, Jamaica, &c.); while the only circumstance which has notably changed in the time has been the condition of the air. So also the extraordinary amount of consumption which prevails among the men of the Royal and Merchant Navies, and which, in some men-of-war, has amounted to a veritable epidemic, is in all probability attributable to the faulty ventilation” (PARKES, *l. c.*, p. 97, 2d ed.). The statistics of the Hospital for Consumption and Diseases of the Chest, London, show that among 3214 men, who composed all the cases of decided phthisis which were registered as in-patients in 10 years, more than one-half—1812—had followed indoor occupations; and of the 2413 females nearly all had.

TABLE SHOWING THE OCCUPATIONS OF 5627 PERSONS OF BOTH SEXES  
AFFECTED BY PHTHISIS.‡

MALES.				MALES.			
Bakers,	.	.	64	Mechanics,	.	.	176
Bookbinders,	.	.	17	Painters,	.	.	105
Bricklayers,	.	.	109	Printers,	.	.	108
Butchers,	.	.	8	Publicans,	.	.	46
Carpenters,	.	.	295	Railway,	.	.	38
Clerks and Shopmen,	.	.	894	Sailors and Watermen,	.	.	74
Coachmen,	.	.	211	Servants,	.	.	285
Gardeners,	.	.	82	Shoemakers,	.	.	171
Laborers,	.	.	589	Soldiers and Police,	.	.	108

\* [Consumption, as Engendered by Rebreathed Air, &c., 2d ed., London: 1865.]

† [A Practical Treatise on Phthisis Pulmonalis. Cincinnati: 1861.]

‡ [Second Medical Report of the Hospital for Consumption and Diseases of the Chest, 1863.]

MALES.					FEMALES.				
Smiths, . . . .	.	.	.	89	Servants, . . . .	.	.	.	984
Teachers, . . . .	.	.	.	42	Domestic, . . . .	.	.	.	447
Tailors, . . . .	.	.	.	145	Milliners, . . . .	.	.	.	397
Weavers, . . . .	.	.	.	11	Laundresses, . . . .	.	.	.	77
At Home, . . . .	.	.	.	68	Governesses, &c., . . . .	.	.	.	80
Various, . . . .	.	.	.	49	Other Trades, . . . .	.	.	.	150
				—	At Home, . . . .	.	.	.	278
Total, . . . .	.	.	.	8214	Total, . . . .	.	.	.	2418

Dr. Pollock, one of the physicians, states that the occupations of out-patients would give the same result. It must then be admitted, from overwhelming testimony, "that deficient ventilation and crowded apartments are eminently productive of tubercular disease" (POLLOCK, *l. c.*, p. 367). Those trades which require a stooping posture—as tailors, bootmakers, weavers—which by mechanical hindrance to the free entry of air into the lungs, restrict the expansion of the chest-walls, and result in imperfect performance of the respiratory function, and consequent interference with the proper nutrition of the pulmonary tissues, and general lessened vitality of the system, furnish, no doubt, a large proportion of cases of tuberculosis. The influence of lessened breath-motion as an exciting cause of phthisis has been particularly insisted on by Dr. Edward Smith, in his excellent work on *Consumption, its Early and Remediable Stages.*]

Parents whose digestive organs are habitually disordered, who suffer from gout, the injurious influence of metallic and other poisons, such as *mercury, malaria, syphilis, chlorosis, or paludal anæmia, the debility of advanced age*, so that a cachectic state is induced, entail upon offspring begotten during such morbid states of the parent, to an extent still undetermined, but obvious and appreciable, a tendency to the scrofulous constitution, not necessarily to be expressed by the growth of tubercle in the lungs, but certainly and surely by various forms of dyspepsia and a precarious state of health.

There are many circumstances in the state of the parents' health, presumed, with justice, to influence the health of the children born under them. Among these may be mentioned a disordered state of the health of the mother, interfering with the state of the fœtus *in utero*, depressing passions, and generally an unhealthy mode of life. "In the present state of our knowledge," writes Sir James Clark, "it is not possible to determine the various circumstances in the health of the parent which may give rise to scrofulous growth in the child, much less to explain the development of scrofula. I rather allude to them as subjects deserving the investigation of the general pathologist and practical physician. There may be differences of opinion as to the particular condition of the parent which induces the tuberculous constitution in the offspring, and also as to the degree in which this constitution may exist in the child at birth."

Let the topics suggested in this quotation be diligently inquired into by those who have the opportunity; and let the initiative be



followed up so ably set forth by Dr. Walshe in the model paper from which the previous statements have been quoted.

[In a table constructed to show some of the leading facts regarding 1200 cases of phthisis, observed at the Hospital for Consumption at Brompton, by Dr. Pollock, the disease was hereditary in 362, or 30.16 per cent. of all the cases. Dr. Pollock writes, of the remote causes of tuberculosis: "Of all of these, doubtless the most important is hereditary predisposition; for, in its absence, how many struggle through influences and errors which destroy others in early life, and attain a considerable longevity? Not all the predisposing causes united could, in a given instance, induce consumption with certainty, without some subtle agent to precipitate, concentrate, and shape those elements of disease into tubercle. . . . . In the absence of more accurate knowledge than science yet possesses, we are inclined to refer the actual precipitation of the disease which we call tubercle to the influence of hereditary predisposition acting in conjunction with, or occasionally even in the absence of, all or any of the other remote agents which give rise to phthisis. . . . . If to any or all of these conditions that of inherited tendency to phthisis be superadded, very few indeed escape the disease. . . . . Finally, the forms of disease, those modifications on which depend its rapidity, development, and duration, are most distinctly reflected in families. How often do we witness child after child carried off at the same age, by the same variety of tubercular disease. Tuberculosis, though not present in the parents, may have an ancestral origin generations back." Dr. Lawson says: "The concurrent opinions of the entire profession fully establish the fact that the tuberculous predisposition is capable of transmission from parent to offspring. . . . . In a majority of cases, however, it appears to be only the *predisposition* which is transmitted, and the open disease becomes subsequently developed. . . . . It is probable, also, that the tuberculous constitution may be congenital, while the ancestors were altogether free from that form of disease" (*loc. cit.*, p. 183-4).]

The scrofulous constitution has been observed to manifest itself in the child under the following circumstances:

1. At birth tubercles may exist in one or more organs (CHAUSOIER, OEHLER, HUSSON, BILLIARD.)

2. The scrofulous cachexia, already defined and described, may rapidly show itself.

3. By the rapid occurrence of tubercles commencing very soon after birth, subsequent to the gradual appearance of symptoms of the scrofulous cachexia.

4. By a disposition to the various forms of dyspepsia, whose characters have been already noticed (page 229, *ante*).

*Race* has an influence in the production of phthisis. In this country the tendency of the Creole and the Negro to phthisis is notorious. But it is notorious also that the Creole and the Negro, when removed beyond certain limits of the land of their nativity, become tuberculous in other lands besides Great Britain.\* In the

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\* [Dr. Pietra-Santa states that a frightful mortality from phthisis almost annihilates the negro emigrants from Central Africa to the Mediterranean coast (*Climate of Algiers in reference to the Chronic Affections of the Chest*, 1862).]

West Indies some of the black races are by no means exempt from this disease, and the Creoles die of phthisis in large numbers in Martinique (RUFZ, NOTT, MORTON). This is the more unlooked for, because as children they live almost in the open air, bathe daily, or still more frequently, and are singularly cleanly in their persons.

“Among the predisposing causes of phthisis,” writes Laennec, “I know of none more certain than the *depressing passions*, especially when they are profound and long indulged; and this perhaps is the cause of the greater prevalence of this disease in larger towns, where bad habits and bad conduct are more common, and often the cause of those bitter regrets which neither time nor consolation can assuage.” He adds, “I had under my own eyes for ten years a most striking example of the influence of melancholy in the production of phthisis. There existed in Paris for that space of time a nunnery of a new foundation, and which had not been able to obtain from the ecclesiastical authorities anything but a temporary tolerance on account of the severity of its rules. The alimentary regimen of the nuns, although extremely severe, was still not beyond the bounds of nature; but the spirit of the rules of the nunnery, directing the mind to the most terrible rather than to the consoling truths of religion, as well as compelling the inmates to resign themselves in everything to the will of the abbess, produced effects as sad as unexpected. These effects were the same in all. At the end of two months’ sojourn in this house the *menses* became suppressed, and in a month or two afterwards symptoms of phthisis appeared. As the nuns had not been allowed to take the usual vows, I entreated that they would leave the house; and all who followed this advice recovered. But during the ten years I was physician to this establishment the members were renewed twice or thrice, with the exception of the superior, the *tourière*, the sisters who had the care of the garden, of the kitchen, and of the infirmary, or of such as had more frequent intercourse with the city, and consequently greater distraction. The rest died of phthisis.”

[Dr. Lawson regards mental depression of all bad influences in the production of phthisis the worst, and the late Dr. Baly regarded it as a powerful causative factor of the disorder amongst the prisoners of the Millbank Penitentiary.]

These circumstances now noticed,—namely, hereditary predisposition, or the influence of parental cachexia generally, of various sources, especially syphilis, the influence of *race*, and of depressing passions,—all co-operate, where they exist, in establishing the scrofulous cachexia; but the efficient cause seems to be “the impoverished nutrition resulting from *impure air*, and an *improper quantity, quality, or assimilation of food*; and so long as misery and poverty exist on the one hand, or dissipation and enervating luxuries on the other, so long will the causes be in operation which induce this terrible disease” (BENNETT).

It is, therefore, *the constitutional state* previous to the actual occurrence of local scrofulous affections like tubercle which it is of the

GROWTH OF THE HUMAN BODY FROM EIGHTEEN TO THIRTY YEARS OF AGE, INDICATED BY WEIGHT AND HEIGHT;  
 AVERAGES TAKEN FROM 100 OBSERVATIONS AND UPWARDS AT EACH AGE. RECORDS TAKEN FROM OBSERVATIONS  
 UPON 4800 CRIMINALS AT ALL AGES (*Statistical Society's Journal*, March, 1862, by J. W. Danson).

WEIGHT.										HEIGHT.					
Age.	Average.		Maximum.		Minimum.		Maximum over Average.		Maximum under Average.	Maximum over Average.		Minimum under Average.		Maximum over Minimum.	
	Stones.	Pounds.	Stones.	Pounds.	Stones.	Pounds.	Stones.	Pounds.		Feet.	Inches.	Feet.	Inches.	Feet.	Inches.
18	8	10.79	10	13	6	6	2	2.2	2	4.79	4	11	5	4.34	5
19	9	4.11	12	8	7	4	3	3.89	2	0.11	5	11½	5	4.94	5
20	9	5.58	12	8	7	13	3	2.42	1	6.58	5	11	5	5.11	5
21	9	5.02	12	0	7	3	2	9.	2	2.	5	11½	5	5.57	5
22	9	12.41	13	2	7	0	3	2.59	2	12.41	6	1	5	6.17	5
23	10	2.95	12	12	7	12	2	9.05	2	4.95	6	1	5	6.17	5
24	10	2.	12	12	7	12	2	10.	2	4.	6	1	4	5.94	5
25	10	5.65	13	8	8	2	3	2.35	2	3.65	6	0	4	6.30	5
26	10	1.06	13	8	6	12	3	6.94	3	3.06	6	1½	4	6.28	5
27	10	4.75	13	10	7	12	3	5.25	2	6.75	5	11½	5	6.38	5
28	10	2.62	13	2	7	7	2	13.2	2	9.62	6	1	5	6.65	5
29	10	5.53	13	12	8	4	3	6.47	2	1.53	6	0½	5	7.02	5
30	10	1.55	14	1	8	1	3	13.45	2	0.55	6	1	5	6.36	5

utmost importance, if possible, to recognize; and the question has been proposed, "Whether or not a predisposition to scrofula can be anatomically or otherwise demonstrable?" There can be no doubt that an assemblage of phenomena, more or less well expressed and obvious, are characteristic of the scrofulous constitution. These phenomena are mainly due to impaired nutrition of the fundamental parts of the frame, and are associated with imperfect growth, especially of the skeleton, the nervous system, and the intestinal tract,—imperfections capable of hereditary transmission to a remarkable extent. Of the structural characteristics of scrofulous persons, none are so significant as the weakness of cell-growth apparent in imperfections of the areolar tissue, as seen in the smooth, soft, delicate, blanched, and transparent skin. The nails are generally defective, elongated, and remarkably incurved. The mucous membranes are thin, pale, delicate, attenuated, and easily expanded. The debility of their function has been already fully noticed. The vascular system is thin, the vessels are transparent, more distensible, and less contractile than in the robust and healthy; and the visible tenuity of the vascular coats is sometimes rendered apparent in the tendency to hemorrhages from slight causes. The veins are conspicuous, and appear to be more numerous under the skin. The bones are imperfectly grown; and there is often a greater want of symmetry than usual in the skeleton. The weight of the man is light compared with his size and his age; and on these points in healthy men there ought to be a due concurrence. So important is the information capable of being derived from accurate observations in this direction, that the preceding and following tables are given for the purposes of comparison:

HEIGHT, AND CIRCUMFERENCE OF CHEST OF 1270 YOUNG PERSONS, RECORDED BY DR. HARRISON, OF PRESTON (*Edin. Med. and Surg. Journal*, 1835, p. 425).

Age.	Number Examined.	HEIGHT.						CIRCUMFERENCE.		
		Average.		Tallest.		Lowest.		Average.	Great.	Small.
		Ft.	In.	Ft.	In.	Ft.	In.	Inches.	Inches.	Inches.
11-12	210	4	2 $\frac{3}{4}$	4	11	3	10 $\frac{1}{2}$	25 $\frac{1}{2}$	30	22
12-13	203	4	5	5	0	3	11	26	29	21
13-14	192	4	6 $\frac{1}{2}$	5	3	4	0	26	32	23
14-15	197	4	7	5	3	4	0	26 $\frac{1}{2}$	32	21
15-16	186	4	10	5	6	4	1	27 $\frac{1}{2}$	32	23
16-17	181	5	0 $\frac{1}{2}$	5	7	4	4	28	34	24
17-18	151	5	0 $\frac{1}{2}$	5	7	4	3	27 $\frac{1}{2}$	34	23

Growth is thus most marked between the ages of fourteen and sixteen years. Its rate is as much as three inches during that time; and about ten inches from the age of eleven to eighteen; and it goes on to be marked between eighteen and twenty-five years of age, when it is about two inches.

There are also ample physiological reasons of the most cogent kind which clearly show that great care is necessary in the physi-

cal training of young persons.\* The main reasons are to be found in the correlations which obtain amongst the following elements, namely,—(1.) Age; (2.) Weight; (3.) Stature; (4.) Development and growth of the skeleton; (5.) The vital capacity of the chest; (6.) The growth of the muscles in relation to the bones; and the progressive increase of muscular force with advance of years.

There are individual peculiarities connected with the skeleton which at once ought to excite suspicion and suggest careful training. These are,—(1.) Narrowness of the thorax, especially at the expansion of the false ribs—a condition, when combined with functional incapacity or inefficiency in the acts of respiration, of most characteristic significance. The vital capacity of the lungs, as determined by (Hutchinson's) spirometer, compared with age, weight, and height, ought to be observed and recorded in all physical examinations. Hutchinson's observations show that men from five to six feet high have a vital capacity of lung ranging from 174 to 262 cubic inches, in an ascending scale according to height and age; and whenever the quantity of air is 16 per cent. deficient, there is reason to suspect some local affection of the chest. Dr. Graham Balfour has subsequently followed up these investigations with the spirometer. He has especially examined how far a capacity *under* the average may be taken as an indication either of a tendency to pulmonary disease or of a feeble constitution, rendering the man liable to a higher rate of mortality than that to which men of or above the average are subject. He found that the loss to the service by consumption was much greater among the men having a "*vital capacity*" *under the average* than amongst men of *average* capacity or *above* it; and although the proportion of deaths did not differ materially amongst those three classes, yet the invaliding was *four times as high* among men *under the average* as among the others. A "*vital capacity*" *below the average* may therefore be considered as indicating a generally feeble organization, less capable of resisting the deteriorating influences to which a soldier is exposed. ("Contributions to the Study of Spirometry," *Med.-Chir. Transactions*, vol. xliii.)

Such functional incapacity is further indicated by the "breathing being shorter," with less "breath motion." The *Expiration* is quick and forcible; and there is a minimum quantity of air taken in by ordinary *Inspiration*. Such lessened respiration tends of itself to induce accumulation of mucus in the air-cells, and thereby to set up inflammation. Everything which tends to impede or to

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\* I. Compare the average height of *full-grown* men at twenty-five years of age, as given by the following observers:

Quetelet,	.	.	5 feet 5.27 inches	=	65.27 inches.	} Average, 66.8.
Danson,	.	.	5 " 6.30 "	=	66.80 "	
Boyd,	.	.	5 " 7. "	=	67. "	
Libarzik,	.	.	5 " 8.89 "	=	68.8 "	

II. The average height of the *growing lad* at eighteen years of age:

Boyd,	.	.	5 feet 0.5 inches	=	60.5 inches.	} Average, 63.008.
Libarzik,	.	.	5 " 4.17 "	=	64.17 "	
Danson,	.	.	5 " 4.34 "	=	64.34 "	



interrupt or obstruct the regular, complete, and continuous performance of the respiratory acts has a most prejudicial effect upon the lungs (especially of "growing lads"), favoring the accumulation of growing material in the air-cells, which may eventually degenerate and form a cheesy mass in all respects resembling tubercle. Life not only depends on breathing, but the energy and the vigor of life are in a great measure ruled by the capacity and the free play of the breathing organs (SIBSON, 1844). Thus Dr. Walshe has very justly directed attention to the prevalence of anæmia amongst female servants in London, who are obliged to go up numerous and long flights of stairs very often in their daily labors; and is of opinion that the suspended inspiratory acts have much to do with the peculiar state of the blood that ensues ("Clinical Lectures," *Lancet*, 1849).

[Against the common idea of anæmia being a frequent manifestation of phthisis, Dr. Pollock protests, and as the result of careful observation of 125 cases of anæmia with murmurs, he declares, that it is a condition which is least frequently found associated with phthisis. In none of his 125 cases was there any sign of tubercle. The girl with extreme anæmia is always supposed to be consumptive. The diagnostic points are these: "In this affection the build of the patient is generally anti-phthisical, and in spite of her color and delicate appearance, there is neither loss of flesh to any great extent, nor fever. The presence of the murmurs (arterial, venous, or both) will decide as to the existence of extreme anæmia. On the other hand, the mobility, percussion, and tone of the respiratory sounds are normal, and this even when cough, dyspnoea, and hæmoptysis may have existed for months. The important blood changes which characterize this condition seem almost antagonistic to tubercle; for not only is phthisis not a concurrent state, but these cases very rarely proceed to the development of tubercle in the lung. . . . Anæmia wastes those organs and tissues dependent on an abundant supply of red blood; phthisis wastes all tissues by furnishing them with impure blood, and leaves a residual deposit. The phenomena of anæmia are those of insufficiency; those of phthisis of impurity of the vitalizing fluid" (*l. c.*, p. 93). Of course this can only hold good with respect to the pretubercular stage, and the active earlier stage of the disorder. After tuberculization has existed for any time, there is great relative diminution in the amount of the red corpuscles.]

It is very important to remember, in regard to training animals and young persons, that they have much more extent and range of lung than are required in the quiet every-day pursuits of life. Less lung is used when the animal lies down or sleeps, or is depressed; and on the contrary, walking, running, wrestling, the force of the passions, each or all of them bring the greatest amount of lung into action or use. In short, the amount of lung in use is an ever-varying quantity; and just as much lung *may come to be used* as a HABIT as the listlessness or vigor of the moment requires. There is every range, every variety, till the top of the wind, the top of the speed (the full vital capacity) is attained (SIBSON). Practically, therefore, the more fully the lungs are judiciously used, the more is their capacity nursed; and conversely, the less they are used and expanded, the more useless are they likely to become, if not

absolutely diseased. Under a judicious system of training, an undeveloped man, even although he may be feeble, narrow-chested, and sickly, may yet become active, full-chested, and healthy. We have numerous examples of this among the boys in our training ships for seamen. The over-fed, short-winded pugilist, rower, or cricketer, may in a few weeks be changed, by training alone, to the firm-fleshed, clear-skinned, long-winded winner of the boxing fight, the foot race, or the rowing match.

It is this want of use that probably renders the apices of the lungs more liable to the growth of tubercle in them than in any other part of their substance. It is a portion of the lungs which has less play or expansion than any other portion, and is apt to be bound down by the surrounding parts. The apices are, therefore, the parts most likely to remain in a quiescent state of non-expansion, especially when acts of respiration are inadequately performed, either owing to the constrained position of the body in certain trades, or from habitual stooping of the body from listlessness of habit or want of vigor in the system. This quiescent state of the air-cells is favorable to the growth of tubercle; and thus the apices of the lungs are the most common seat of tuberculous growths (Reviewer in *Medical Mirror*, vol. i, p. 638). The effects of want of exercise and of impure air are thus most potent agencies in causing phthisis. "The much greater prevalence of phthisis in most of the European armies (French, Prussian, Russian, Belgian, and English) can scarcely be accounted for in any other way than by supposing the vitiated atmosphere of the barrack-room to be in fault." In all the places where phthisis has prevailed, in the most varied stations of the army, and in the most beautiful climates, the only common condition was the vitiated atmosphere which our barrack system everywhere produced; "and, as if to clench the argument, there has been of late years a most decided decline in phthisical cases in these stations, while the only circumstance which has notably changed in the time has been the condition of the air" (PARKES, *l. c.*, p. 91). Indeed, the air is rendered so impure by respiration, that while an atmosphere so vitiated has a most injurious effect upon the health, contamination of the air has been so great, where lung diseases abound, as to give rise to the idea that phthisis appeared to be propagated by contagion (BRYSON, PARKES).

It is therefore within the power of the medical officer to direct the physical training of young persons so that the apparently sickly and the short-winded may in time be developed into the wiry and active young man, long in wind, sound in body, and lithe of limb—a result which, however, can only be attained by judicious feeding, careful exercise throughout the development of the body, and by the gradual nursing of the breathing powers. The opposite of this is seen in the breaking down of recruits at a very early period of service in the Army—within the third year—a result likely to be greatly obviated by the gymnasia which are now being introduced by Government for the physical training of recruits before and after they are instructed in drill.

The Austrian runners are another class who are instances of breaking down by over-exertion in running at ages unsuited for their strength. They seldom live above three or four years, and gradually die of consumption (*Remains of Mrs. Trench*, p. 72).

In all physical training the condition of the heart must be considered not less than the lungs, seeing that its movements respond to the movements of the chest and the lungs. They are members of one great system; and in nursing one we nurse the other; for by giving tone and health to one we must give tone and strength to the other (SIBSON). If, on the contrary, the development and gradual training of the lungs are not successful, the lesions which become developed are of a peculiar character, and are apt to be thought very lightly of; because, in the first instance, they are so little capable of appreciation by a single observation; and, secondly, because the lesions are insidious, and take some time before they reach a stage to be apparent. Frequent observations, extended over a considerable interval of time, are necessary to appreciate the wasting of delicate tissues which sets in, and which progresses so insidiously, and creeps on almost imperceptibly, but with a result so sure "that day by day and grain by grain the mortal part wastes and dies away." It is this progressive atrophy which it is so important to detect. Time is necessary to do this, and some certain mode of detecting the gradual decay. For this reason it is absolutely necessary to have the means of weighing men, and so to determine the ratio of the weight of the person to his age, to his height, and to his respiratory and other functions. These data are absolutely necessary to be known and recognized before any conclusions can be arrived at as to those healthy or normal physiological states with which weight has to do. It may, indeed, be stated generally that every organ and function has a physiological relation to weight and age; and, as a general rule, weight increases with the height and age, and there are physiological limits beyond which the range may not extend.\*

Loss of weight is therefore *exceedingly significant*, if PROGRESSIVE, *as indicating a persistent atrophy*—the *grain by grain decay* which is frequently the earliest appreciable sign of disease.

[The first evidence of the invasion of phthisis is altered nutrition. The earliest appreciable symptom of the disorder is, without question, loss of body-weight. Its first manifestation is an absorption of the adipose tissue, and wasting of the muscles. Though the appetite is good, and the secretions regular, emaciation below the lowest average of the individual steadily goes on. It is a precedent condition to tuberculization, in all cases of phthisis, except the most acute form. It is more marked and more perilous in those who have been previously stout. It is regularly progressive, and is thus distinguished from transient alterations in weight. In senile phthisis a long period of emaciation commonly precedes the positive evidence to be derived from physical signs (POLLOCK). Sudden and great loss of weight is always serious, and indicates in the early stage

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\* Henry Pooley & Co., 89 Fleet Street, E. C., supply suitable weighing machines at a cost of £8 15s., and upwards.

rapid pulmonary changes. The most rapid reduction of weight is in males. The quickest waste of flesh that Dr. Pollock verified took place in 3 months, from 12 st. 5½ lbs. to 8 st. 5½ lbs., a loss of 4 st. The following table shows the reduction in weight in 28 phthisical cases (POLLOCK):

Period.	No. of Cases.	1 stone.	1½ stone.	2 stone.	2½ stone.	3 stone.	4 stone.
In 5 weeks .	1 had lost . . . . .	—	—	1	—	—	—
" 2 months	4 " (respectively)	—	1	2	—	1	—
" 3 "	7 " "	1	2	1	—	1	1
" 4 "	8 " "	—	—	2	—	1	—
" 6 "	8 " "	—	1	1	1	—	—
" 8 "	1 " "	—	—	—	—	1	—
" 9 "	1 " "	—	1	—	—	—	—
" 10 "	8 " "	—	1	1	1	—	—
" 12 "	5 " "	1	—	1	—	2	1
Total cases,	28						

The lowest weight in health of one of these persons was 10 st.; he lost in 5 weeks 1½ st. The rest in health averaged 12 st.; and 1 was 14 st. 10 lb., and he lost 3 st. in 8 months. Another, whose weight in health had been 13 st., was reduced to 10 st. in 3 months; 20 out of 28 observed, were between 25 and 35 years of age; 6 were in the first stage; 10 in the second; and 12 in the third of phthisis when examined, showing that the period of deposit was exceedingly short, and the softening and destructive processes accelerated.]

**General Treatment of the Scrofulous Cachexia.**—From what has been already stated in illustration of the pathology of this malady, it is to be observed that the general treatment now in most repute is founded on the doctrines—(1.) That scrofulous local lesions will heal of themselves, if the nutrition of the system can be maintained and the continuous growth of tubercle arrested. (2.) That the periods of frequent temporary arrest of all the general and local symptoms of disease ought to be diligently taken advantage of to improve and preserve health by hygienic means. (3.) That the efforts of the practitioner should be directed to the digestive rather than to the pulmonary system. (4.) That the kind of morbid nutrition in the body generally, and altered morphological change in the tissues of the organ where the deposit takes place, appear to be chiefly due to excess of albuminous and deficiency of fatty elements in the chyle.

The treatment, therefore, to be pursued must be essentially reparative of the waste of tissue generally; corrective of what has been unfit in the individual diet and mode of life; and, lastly, supplementary of the elements of nutrition which have been deficient.

During the past *thirty* years in Germany, and *twenty-three* years in this country, the treatment of scrofulous affections has gradually but steadily become more and more firmly based on these pathological doctrines, which Dr. Bennett, of Edinburgh, was mainly

instrumental in first elucidating, and in earnestly recommending to the notice of the profession generally in this country. The view here taken regarding the nature of scrofula leads to the belief that—(1.) The blood is impoverished through the preliminary dyspepsia which precedes the growth of tubercle. (2.) That in *pulmonary phthisis* the growth of tubercle results from the exudation of lymph and of new growth which is consolidated primarily in the air-vesicles. (3.) That the successive formation and softening of these tubercles lead to ulcerations of the pulmonary and other tissues, and promote wasting of the body generally.

It has been now fully shown, especially by the observations of Dr. Bennett, in the first instance, confirmed by the extensive experience of the physicians at the Brompton Hospital for Consumption in London, that such treatment as is directed to remove the mal-assimilation of fluid frequently checks the tendency to the repeated occurrence of tubercles, while those which previously existed remain harmless; that general symptoms and physical signs may disappear completely; and that even extensive excavations in the pulmonary tissue may heal up and cicatrize. According to the testimony of Dr. Wood, of Philadelphia, the fatal results have not only been postponed, but the death-rate from phthisis has diminished in the principal cities of the United States since such principles of treatment have been adopted.

The indications of general treatment are, therefore,—

First. *To improve the faulty nutrition, which is the cause of the scrofulous cachexia, and of the exudations assuming the characters of tubercle.*

Second. *To subdue the fever which attends the growth and changes going on in the tubercle-nodules, and to favor the absorption either of the entire exudation, or of such portions of it, that what remains may undergo such changes as are consistent with its future harmless existence in the organs or other parts where it may have grown.*

Third. *To prevent the recurrence of fresh exudation by careful attention to hygienic regulations, especially during the intervals of apparent return to health.*

To fulfil the first of these indications, it is of all things important that fatty matter be assimilated in large quantities, and it appears that such are most readily absorbed and assimilated when in the fluid condition. The substance of all others found most beneficial has been cod-liver oil. At the Brompton Hospital more than 600 gallons of this oil are used annually. There, on a great scale, its merits have been tested and compared with the effect of treatment conducted on general principles, and irrespective of its use. The result has been to confirm, in the estimation of the medical profession, the great value of this remedy in the treatment of phthisis, when appropriately administered, and combined with the use of such other measures as any special circumstances in the individual patient may require (THOMPSON). The general opinion of the profession with regard to cod-liver oil may be summed up in the statement originally made by Dr. Bennett, namely, that “it rapidly restores the exhausted powers of the patient, improves the nutritive functions generally, and stops or diminishes the emaciation. The



night perspirations subside, the cough is quieted, and the expectoration is diminished after the oil has been used for a few weeks. A very constant and well-marked favorable change under its use is to be noticed in the diminution of the pulse, which gradually but steadily has been observed to come down at the rate of five or six beats weekly; and during the same period the weight of the body has been known steadily to increase at the rate of half a pound to a pound. In females it is a significant and highly favorable symptom when the catamenia return after the oil has been used for some time. The usual dose adopted by the late Dr. Thompson, at the Brompton Hospital, is *one or two teaspoonfuls twice a day* at first, and gradually increasing the quantity to *half an ounce three times a day*. Dr. Bennett recommends somewhat larger doses; namely, for an adult, *a tablespoonful three times a day*, which may often be increased to *four or even six* with advantage. When the stomach is irritable, however, *a tea- or a dessert-spoonful is enough to commence with*. It appears from the observations of Dr. Thompson that no additional advantage is obtained by pushing the oil beyond the limits of the doses adopted by him at the Brompton Hospital, from the fact that where its use has most obviously increased the weight of the body, to the extent in one instance of a pound per week for twenty-one weeks, only three pints had been taken during that time. The kind of oil used, as far as coarseness or fineness is concerned, seems not to affect the beneficial result in any material degree. Some patients even prefer the coarse to the fine oil. The experience of some is, moreover, favorable to combining the oil with *liquor potassæ* as an emulsion; and as it appears that undue acidity prevails as well in the stomach as in the intestinal canal, the addition of the alkali ought, on theoretical grounds, to be advantageous.

It seems also that, when cod-liver oil was first used as a medicine, more than fifty years ago, in the treatment of rheumatism, it was then ordinarily combined with an alkali. It may be taken, however, unmixed, or it may be floated on milk, or nitro-muriatic acid mixtures, or on lemonade, soda-water, lemon-juice, or on a saline draught during effervescence, when such combinations are suited to the patient. Creasote has been recommended to be added, as it is said to render the stomach more tolerant of the remedy. The following formula, quoted from Dr. Thompson's *Clinical Lectures on Pulmonary Consumption*, yields a palatable mixture, which ought to be combined as an emulsion:

“An ounce and a half of cod-liver oil, four drops of creasote, two drachms of compound tragacanth powder, and four ounces and a half of mixed water. Of this mixture an ounce may be taken thrice daily.”

The more direct and immediate action of cod-liver oil upon the blood has been attempted to be ascertained by Simon, Snow, and Thompson. They record an increase of blood-corpuscles and a diminution of fibrine under its use; and from the researches that have been made in animal chemistry regarding the blood in phthisis, a deficient proportion of blood-corpuscles is observed to be a most con-

stant peculiarity. But phthisis is not the only disease in which this occurs, as shown by the following table (SIMON):

AVERAGE PROPORTION OF SOME CONSTITUENTS OF THE BLOOD.

	Albumen.	Corpuscles.		Albumen.	Corpuscles.
In Health, . . .	76	180	In Diabetes, . . .	105	80
In Pneumonia, . . .	80	122	In Bright's Disease, . . .	108	50
In Phthisis, . . .	100	78	In Chlorosis, . . .	72	56
In Rheumatism, . . .	100	74	In Carcinoma, . . .	45	55

Rheumatism and diabetes present the greatest similarity in these states of the blood to phthisis; and they are diseases for which cod-liver oil has been used with advantage.

Cod-liver oil, therefore, is indicated, where it can be taken, in all those diseases in which the blood-corpuscles are deficient, where nutrition is impaired, and where fat is not readily assimilated.

Besides cod-liver oil, other animal fats and oils, where they can be taken and assimilated, are sure to be followed with benefit. Hence milk, rich in fatty matter, such as asses' milk, and milk drawn from cows at a short interval after the greater part of their milk has been withdrawn, and which is known in Scotland as the "afterings," are found to be followed by improvement where they are persevered in and are assimilated. So also has it been with cream and butter. Dr. Bennett instances the partial success occasionally of caviar, bacon, pork, mutton chops, and the marrow of the bones of oxen; while Dr. Thompson instances the good effects he has obtained from the use of oil obtained from the foot of the young heifer (neat's-foot oil). The administration of any of these remedies is quite consistent with doctrines now taught regarding the pathology of tuberculosis, and it is useful to know their individual value, in order that in particular cases one may fall back upon their use where a change may be desirable.

It has been considered that some of the good effects of cod-liver oil may be due to the biliary elements with which it has been incorporated. This view is not supported by the experiment of adding ox-gall to other animal oils not derived from livers, as no beneficial results have been observed to follow. But as the active principle of the gastric juice has been now successfully isolated by chemistry, and has been successfully used to aid the digestion of food in the stomach, might not some principle be obtained from the liver which might aid the assimilation of fatty substances when mixed with the intestinal juices?

In fulfilment of the second indication mentioned (p. 246), the propriety of abstracting blood has been much discussed.

It has been already seen that febrile symptoms of a very severe kind sometimes attend the exudation and consolidation of tubercle. It is also a more or less frequent clinical observation that pneumonia, bronchitis, and pleuritis, in acute or chronic forms, are intercurrent attendants on the deposit and future changes of tubercles in the lungs. "Hence," as Dr. Bennett justly observes, "there are all kinds of intermediate changes between the simple and tubercular exudations constantly going on in the progress of a case of pulmo-

nary tuberculosis. The phenomena of phthisis, pneumonia, pleurisy, and bronchitis, in their acute or chronic forms, may appear together, and be inextricably mingled, or they may succeed each other at intervals." Thus tuberculosis, both as a constitutional and as a local disease, is scarcely ever free from exacerbations, the various local and constitutional states acting and reacting on each other.

While, therefore, on the one hand, the system requires an increased and well-directed supply of nutritive materials; on the other hand, there are constitutional states of excitement, depending on local irritation, which require to be subdued, and even demand antiphlogistic treatment. Whatever theoretical view may be taken as to how the exudation may most readily be absorbed, all physicians are now at one as to the propriety of preserving the general strength, of effecting elimination of effete material, and of meeting antiphlogistic indications rather by diaphoretics, diuretics, emetics, and purgatives, than by abstracting any considerable amount of blood, either at once or at repeated intervals. In fact, it is now observed that the administration of appropriate diet, and abstinence from lowering remedies, with cod-liver oil, while they correct the general nutrition, may be so regulated as to subdue the constitutional irritation by a perseverance in their use for a period of not less than four or six weeks. Moderate general bleedings in acute phthisis, as well as local bleedings during the exacerbations of chronic phthisis, undoubtedly confer a temporary relief in the diminution of local pain and general febrile reaction, and allow a more free respiration to be performed, if respiration should be impeded by congestion of the lungs. Dr. Bennett, however, is opposed to bleeding. His object is simply to favor excretion by means of antimonials; and, on subduing the reaction by their means, he again proceeds with the nutritive mode of treatment to fulfil the first indication. Nevertheless, it cannot be doubted that the condition of some cases demands bloodletting; but, as Sir James Clark observes, "The employment of general bloodletting in consumption requires great judgment and circumspection. The more general error is the abstraction of too great a quantity of blood at a time—treating the disease as if it were a purely inflammatory one, and forgetting that the inflammatory symptoms are merely consecutive upon tubercles, and that the constitution of the consumptive patient is little capable of replacing the blood too lavishly drawn." Simply to remove or diminish congestion, the condition of the patient and nature of his constitution being considered, "blood may be abstracted," he continues, "with advantage at any stage of consumption when the symptoms require it." The experience of Dr. Wood leads him to teach similar doctrines. Topical counter-irritants furnish the best means of subduing constitutional irritation in chronic forms of the disease. These may be in the form of setons, issues, succession of blisters, tartar emetic ointment, or croton oil rubbed on the chest in any of its regions. Amongst these the frequent use of dry cupping ought not to be omitted.

In fulfilling the third indication, the real power of the science of medicine may be demonstrated. It is by well-directed hygienic

measures, successfully and efficiently carried out, that the real strength of the physician may be put forth to prevent the recurrence of fresh exudation; and his hygienic exertions towards the patient are to be redoubled during the temporary intervals of apparent return to health. It is now almost an axiomatic truth, that of all things which deteriorate the constitution on the one hand, and influence pulmonary congestion on the other, none are so detrimental as impure and deficient air, together with frequent variations of temperature, and changes from sudden heat to chilling cold. These latter vicissitudes may be considered characteristic of the physical climate of Great Britain and Ireland. "The conditions of preventive treatment which have seemed most useful are nutritious food and proportionate great exercise in the free and open air. So important has this last condition proved to be, that it would appear that even considerable exposure to the weather is better than keeping phthisical patients in close rooms, provided there be no bronchitis or tendency to pneumonia or pleurisy" (PARKES, *l. c.*, p. 445).

Tonic treatment consists essentially in the adoption of those means which promote or stimulate the healthy nutrition of the body. Its elements exist in fresh air, abundant exercise, sufficient repose, and judicious diet. A hygienic code applicable to the tuberculous cachexia has been recently laid down by Dr. Richardson, in the second volume of the *Sanitary Review*, and very recently in a special work *On the Hygienic Treatment of Pulmonary Consumption*. It is derived from these elements of tonic treatment; and as it puts well-known truths, too little appreciated, in a formal and more important aspect than is wont, its precepts are here quoted.

I. *A supply of pure and fresh air for respiration is constantly required by the tuberculous patient.*

As it is known that if one per cent. of carbonic acid exists in a room, the air is unfit for a healthy person, it is therefore much more so for a consumptive one. The temperature of a room ought to be equally maintained at from 55° to 56° Fahr., ventilation and heating being effected by open fire-places. A single room ought not to perform the two offices of a bed-room and a sitting-room. The sleeping-room ought not to afford less than 1000 feet of space; and if larger, so much the more healthful will it be. In connection with these statements, while it is objected, for obvious reasons, with much justice, to the treatment of tuberculous patients in special hospitals, there is much on the other hand that might be improved in all our hospitals, with reference to the arrangement of the patients, to secure to them fresher air than they generally obtain. "Constant though imperceptible movement of the air is the point to be attended to"—*i. e.*, thorough ventilation. All who are able to be out of bed ought to have their meals in a common room, which is not used for any other purpose, and is apart from the wards or dormitories.

II. *Active exercise in the open air is imperatively demanded by the tuberculous patient.*

In the words of Dr. Jackson, "He must be made to feel that the risk is in staying in the house, and not in going out of it." But the

skin must be perfectly protected, and while a chill, or inclement weather, is to be avoided, the patient must go out in all seasons, without being too fastidious about the weather, walking exercise being persevered in as much as possible. "The best climates for phthisis are perhaps not necessarily the equable ones, but those which permit the greatest number of hours to be passed out of the house" (PARKES). Next to diet, exercise in the open air is, of all things, the most important: it should be carried as far as the vigor of the patient will permit. It should not be done rashly, but boldly; and, if possible, the patient ought to have faith in it, for without this he is not likely to pursue it so far as he can, and then he will not derive from it all the benefit which it can afford (JACKSON).

III. *It is important to secure for the patient a uniform, sheltered, temperate, and mild climate to live in, with a temperature about 60°, and a range of not more than 10° or 15°; where also the soil is dry and the drinking-water pure and not hard.*

The classic work of reference on this topic is that by Sir James Clark. If it is possible to give a practical abstract of his extensive and valuable experience, it may be done somewhat as follows:

1. After the functions of the digestive organs and skin have been re-established in improved action, the patient who labors under a tuberculous cachexia may derive benefit by a residence in a mild or temperate climate (such as has been defined), conforming to all the hygienic and medicinal treatment already mentioned.

2. When symptoms, however slight, indicate that tuberculous deposit has located itself in the lung, removal to a mild climate, especially if effected by a sea voyage, under favorable circumstances, may still be useful as a means of improving general health, of lessening the chance of intercurrent inflammatory affections of the pulmonary organs, and even of arresting the further progress of the disease. The nausea, squeamishness, or even sickness, which with some are always more or less associated with a sea voyage, are beneficial to cases of incipient tuberculosis. The effects of such nausea tend to increase the natural secretion and elimination from the pulmonary mucous membrane; so that minute portions of tubercular exudation, commencing to consolidate in the air-vesicles, are effectually, gradually, and gently passed out with the motion of the pulmonary mucus in the expectoration. The sensation of nausea tends to subdue any local vascular irritation; and the unceasing motion of a sailing vessel tends to keep up a constant exercise which is advantageous to the patient.

3. When extensive tuberculous disease exists in the lungs, little benefit is to be expected from a change of climate; and a long journey will most certainly increase the sufferings of the patient, and hurry on a fatal termination.

4. There are cases, however, of chronic consumption which may derive benefit from residence in a mild climate,—namely, cases in which the deposit is limited to a small portion of the lungs, and little systemic irritation prevails; or in cases in which the disease has ceased to extend in the lungs, but where a long time is required to complete repair.



IV. *The dress of the scrofulous patient ought to be of such a kind as to equalize and retain the temperature of the body.*

Under this topic waterproof coats, boots and shoes are to be condemned. Flannel ought invariably to be worn next the skin in all seasons; and in winter a chamois leather vest may be required over the flannel.

V. *The hours of rest should extend from sunset to sunrise.*

VI. *Indoor or sedentary occupation must be suspended; but outdoor employment in the fresh air, even in the midst of snow, has been and may be advantageous.*

VII. *Cleanliness of body is a special point to be attended to in the hygienic treatment of tuberculosis.*

VIII. *Marriage of consumptive females, for the sake of arresting the disease by pregnancy, is morally wrong and physically mischievous.*

IX. *The medicinal treatment must be adapted to the site of the local deposits and the general nature of the particular case.*

Medicine is utterly powerless and useless unless the hygienic means now insisted upon are carried out to the uttermost.

## RICKETS.

LATIN EQ., *Rachitis*; FRENCH EQ., *Rachitisme*; GERMAN EQ., *Rhachitis*—Syn., *Englische Krankheit*; ITALIAN EQ., *Rachitide*.

**Definition.**—*A constitutional disease of early childhood, characterized by an unhealthy state of the system, which precedes for several weeks or months a peculiar lesion of the bones, manifested by curvature of the shafts of long bones and thickening of their cancellous extremities. Some of the solid visceral organs exhibit also peculiar lesions. The growth of the bones is characterized by irregularity, by non-solidification of their growing layers, and by the progressive formation of medullary cavities in the old, thus rendering the bony laminæ thin and brittle (VIRCHOW). In the solid visceral organs, such as the spleen and liver, there is generally albuminoid (amyloid?) degeneration.*

**Symptoms.**—The earliest recognition of the cachexia associated with rickets is rarely apparent before the fourth month of infant life; and usually between the fourth and twelfth months. It does not in general declare itself until the child first begins his attempts to walk, or until he shows suffering during the first dentition; and at first the progress of the disease is so very slow as almost to be imperceptible. The number of cases happening in the first or second years of life very greatly exceeds that of other periods; and there is a period of at least six months during which a marked series of deranged actions succeed each other, and which eventually culminate in the condition known as *rickets*. Many of the phenomena of these deranged actions are common to other diseases; but some are characteristic, and when they occur in sequence are sufficient indications of the specific characters of rickets. The symptoms may be arranged into four classes: (1.) Those which are common to many diseases—symptoms which might arise from de-

ranged digestion, from improper food, or from tuberculosis, and which are often referred to the "irritation of teething;" or to the so-called "infantile remittent fever." These phenomena always denote the precursory or incubative stage of rickets; (2.) Those which at once mark the nature of the disease, render its diagnosis easy, and which enable us to predict that the bone affection will show itself; (3.) The stage of characteristic deformity; (4.) Phenomena of favorable or of unfavorable import, inasmuch as they may characterize a period of restoration to health, of irremediable atrophy of the body, or of approaching dissolution.

During the precursory or incubative period, the most ordinary symptoms of impaired general health are those which indicate gastro-intestinal irritation. The bowels are irregular in their action; sometimes confined, but more commonly there is diarrhœa, with tumidity or enlargement of the abdomen. The stools may be of a dirty brown or leaden color, and of a most offensive odor. In some respects this odor is peculiar in its resemblance to rotten or half-decayed meat. Appetite is feeble, or entirely lost, and digestion is difficult. The child becomes dull and languid; sad, or peevish; febrile irritation prevails; the skin is hot; and the temper irritable. Although drowsy, it sleeps but little. It is thirsty, and will drink large quantities of water. If it has begun to walk, it is "taken off its legs." It lies about, and is unwilling to play, or to be amused, or to indulge in any kind of action. It prefers to sit or to lie; and it appears to be feeble or indolent, and is unable to use exertion of any kind. The transition from health to these phenomena is always gradual and slow; but there are at least three sets of phenomena which, according to Dr. Jenner, being superadded to these, are characteristic of the approach of rickets. These symptoms, therefore, stand by themselves in the second class as pathognomonic of this affection.

(a.) The most remarkable is *profuse perspiration of the head, or of the head and neck*, and upper part of the chest. It arrests the mother's attention, and she seeks medical aid. She will tell the physician that the perspiration stands in large drops on the child's forehead—that it runs in streams down the face; and it is especially when the child sleeps that such copious perspirations of the head occur; but they are not unfrequent when the child is at the breast, or even when only resting its head on the mother's arms. A little increased exertion, or a little increased temperature, may induce such excessive perspiration. Such perspirations are extremely weakening and colliquative during sleep; and when they occur the superficial veins of the scalp are generally large and full; the jugular veins are much dilated, and sometimes the carotid arteries may be felt strong and pulsating (COPLAND, JENNER).

(b.) Another characteristic feature of the disease is seen in the desire and in the efforts of the little patient to be cool, particularly at night. The child kicks the bedclothes off, or throws its naked legs on to the counterpane; and this even in cold weather.

(c.) There is also, thirdly, general tenderness. The child cannot be moved without its uttering a cry: pressure on any part of its

body is followed by evidence of suffering. It ceases to play and to move, but lies with outstretched limbs as quietly as possible, for all movement produces pain; and it will cry at the approach of any one who has been accustomed to move it in play.

As the disease progresses the child becomes staid and steady in appearance. It assumes a pensive, aged, and languid aspect. Its face grows broad and square; and when placed on the mother's arms, it sits (as she says) "all of a heap." The spine bends, and the muscles are too weak to keep the spine erect. Its head thus seems to sink between its shoulders, and its face appears turned a little upwards. Before the general cachexia has lasted long, the bone deformity begins to attract attention; and usually the lesion of the bones is out of all proportion in severity to the enlargement of the ends of the long bones; and the younger the child, the softer usually are the bones. The consequences of the bone disease thus become superadded to the general cachexia; and as the disease progresses, the muscles lose their power and begin to waste. The child cannot support itself; and if it has commenced to walk before it becomes the subject of extreme rickets, it loses its power of walking. Intellect is invariably deficient. The teeth are retarded in their development, and they fall from their sockets early. The back, arms, and sides of the face are often covered with downy hair. In short, as Dr. Jenner observes, "the general aspect of the ricketty child is so peculiar that when the crooked limbs, the large joints, and the deformed thorax are concealed, you may even detect its ailment at a glance. Its square face, its prominent forehead, its want of color, its large staring and yet mild eyes, its placid expression, and its want of power to support itself like other children of its age, in its mother's arms, all conspire to form a picture which has no like in the gallery of sick children" (*Med. Times*, l. c.). *Laryngismus stridulus* is a frequent result of rickets.

**Treatment.**—Ventilation of the room in which the child lives is of first importance. Milk diluted with lime-water (about a fourth part), and a teaspoonful or two of cream added, is the best of food. Sugar ought not to be added to the milk. Liebig's food for children and Parrish's chemical food are both valuable agents in the dietary. About once a week a dose of rhubarb, soda, and calumba, in equal parts, should be given, followed next day by a teaspoonful or more of castor oil. Prepared chalk and soda may also be given twice or thrice a day. When the febrile disturbance is subdued, the child should live as much as possible in the open air. *Vinum ferri* ought then to be given, or small doses of the *syrup of the phosphates of iron, quinine, and strychnia*, along with the food, or just before meals. Cod-liver oil is of essential service; but the stools ought to be examined daily, and if any of the oil passes by stool its dose ought to be diminished, or its administration suspended for a time. (For more details on *Rickets* and its treatment, consult the admirable lectures of Dr. Jenner, in *The Medical Times*, of 1860, vol. i.)

## CHAPTER V.

## THE THEORY OF SPECIFIC DISEASES, COMPREHENDING THE SO-CALLED ZYMOTIC AND CONSTITUTIONAL DISEASES.

THE classes of diseases which have been now considered—namely, the so-called ZYMOTIC and the CONSTITUTIONAL—are sometimes regarded and described under the name of “SPECIFIC DISEASES.” The general theory regarding the nature of such diseases, so considered as *specific*, is thus expressed by Mr. Paget:

“Each of them depends on a definite and specific morbid condition of the blood; and the local process by which each is made manifest is due to the disorder produced by the morbid blood in the nutrition of one or more tissues; and generally this disorder is attended with the accumulation, and leads to the discharge or transformation, of some of the morbid constituents of the blood in the disordered part. It is held, also, that in some of the SPECIFIC DISEASES the morbid condition of the blood consists in undue proportions of one or more of its normal constituents—in others, again, some new morbid substance is added to or formed in the blood. In either case the theory maintains that the phenomena of each SPECIFIC DISEASE depend chiefly on certain corresponding specific materials in the blood, and that, if characteristic morbid structures be formed in the local process, these morbid materials are incorporated in the organs which are formed as the products of the inflammation.”

In the preceding part of this Handbook the SPECIFIC DISEASES have been considered in two distinct classes—the divisions being founded on the fact that in the first class, or ZYMOTIC DISEASES (comprehended in vol. i), *the chief or essential constituent of the morbid agent enters the body from without*; while in the second class, or CONSTITUTIONAL DISEASES, *the essential constituent of the morbid agent appears to be inbred in the body, and makes itself manifest by various constitutional indications previous to the development of local lesions, or the characteristic expression of the disease by other phenomena.* There is sufficient circumstantial evidence with regard to all of these diseases, and absolute proof with regard to some, that there is—(1.) A morbid condition of the blood; (2.) That the nature of that condition is, in many, definite and specific, inasmuch as it may be produced at will by the introduction of a definite substance into the blood, which then manifests itself by establishing a local disease, and which, within certain limits, has constant characters; (3.) That the morbid matter or poison by which the condition of the blood is changed may accumulate or augment in quantity or virulence, and at length may be discharged in various ways from the body, and under a variety of organic forms, chiefly through the excretory products. But some are also discharged at the seats of local lesions which are set up, the morbid poisons being for the time accumulated in the morbid structures.

As far as some of the *miasmatic diseases* are concerned, such as

*typhus and typhoid fevers, erysipelas, scarlet fever, cholera, and the like*, there is undoubted evidence of a morbid material in the blood, although it has never been isolated nor proved to exist in the products of the local morbid processes or specific lesions. In many of the *constitutional diseases*, too, similar evidence exists of a morbid matter *inbred* in the blood.

It will soon appear obvious to the student that the diseases already considered differ very materially in their nature from many *local diseases* about to be noticed. While the LOCAL DISEASES may be regarded as *common* or *simple* diseases, those already noticed are so very distinct from them, and are possessed of such constant features by which they may be distinguished from the local, common, or simple diseases, that they are frequently described under the term "SPECIFIC."

Certain types of morbid local action, however, are common to both; and therefore the SPECIFIC DISEASES have some constant and characteristic modification, or something in addition, which distinguishes them, such as *syphilis, gout, rheumatism, tuberculosis, and the eruptive fevers*. These additional elements appear to consist,—

1. In a certain constancy and regularity of development, metamorphosis, duration, and decline, during which certain common morbid processes become modified according to the *special* nature of the disease. "In some the most evident specific characteristics are peculiar affections of the movement of the blood, as in the *cutaneous exanthemata*; in some, affections of certain parts of the nervous centres, as in *tetanus, hydrophobia, whooping cough, and asthma*; in some, peculiar exudations from the blood, as in *gout*, and the *inoculable diseases*; in some, peculiar structures formed by the exuded materials, as in *variola vaccinia*, and other *cutaneous pustular eruptions*; in some, destruction of tissues, as in the *ulcers of syphilis*, the *gangrene of ergotism*, and the *sloughs of boils and carbuncles*; in some, peculiar growths, as in *cancers*; in some, or indeed in nearly all, peculiar methods of febrile general disturbance" (PAGET).

In the specific diseases the phenomena of these local and general morbid processes are concurrent.

2. The most striking feature (assuming specific diseases to be due to the presence of morbid poisons in the blood, which, by a morbid process, is again separated from it, and eliminated from the system during the progress of cure) is that "the whole blood for a time seems diseased, and nearly every function and sensation is more or less disturbed from its health; and the patient feels 'ill all over' before the local disease appears" (PAGET).

In the common or local diseases, on the other hand, the local phenomena precede the general or constitutional disturbance.

3. There exists in both classes of the SPECIFIC DISEASES now described an apparent want of proportion between the cause and the effect. Thus, in *small-pox, measles, hydrophobia, bites of poisonous serpents, or syphilis*, the severity of the disease does not bear any proportion to the amount of poison applied; and numerous diseases have been described in which the morbid poison appeared to act with so much intensity, and produced such severe forms of disease,



that the patient died before local lesions had time to become developed,—e. g., in *typhus fever*, *specific yellow fever*, *paludal fevers*, *scarlatina*, and the like.

The student is referred for more information on these important topics to what has been already written at pages 203 and 724, *et seq.*, vol. i, of this Text-book, and also to Lecture XX, on “Specific Diseases,” by Mr. Paget, in his work *On Surgical Pathology*.

Of the two classes of SPECIFIC DISEASES, those which have been described as CONSTITUTIONAL, or *inbred*, appear to be, in their origin, essentially *blood diseases*, the disturbance due to their development being indicated by what has been termed a *cachexia*.

Those of the ZYMOTIC class, again, recognize the introduction of *materies morbi* from *without*, and symptoms arise which indicate much constitutional disturbance. But, in whichever way these diseases are brought about, their specific nature is such as has been described. Under the term *dyscrasiæ*, the specific condition of the blood in such diseases was wont to be described by many continental pathologists, implying a defective organization or elaboration of the circulating fluid. We cannot yet, however, associate particular diseases with definite and particular morbid states of the blood, however certain we may be that changes of a morbid nature do so exist; and therefore the term *dyscrasiæ*, as applied to designate such states, is premature in application, and has not come into general use, because it takes for granted more than is known.

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## CHAPTER VI.

### GENERAL MANAGEMENT OF THE SYSTEM LIABLE TO CONSTITUTIONAL DISEASES.

IMPORTANT general indications for the treatment of the CONSTITUTIONAL diseases are suggested by their pathology as expounded in these pages. This general treatment ought to be directed towards the management of the system for the *prevention*, *control* or *arrest* of the development of such affections, or to maintain them within such limits as are consistent, at least, with the well-being of the individual.

The topics to be noticed under this head are of so comprehensive a scope that they can only be indicated as shortly as possible, with the object of directing the mind of the student to their more extended study. They embrace, in fact, the whole subject of general and individual hygienic management. The student is therefore referred at once to two standard authorities, with whose maxims and practice his mind ought to become most thoroughly imbued, in order to direct successfully the measures for the prevention and arrest of the CONSTITUTIONAL DISEASES. The works here referred to

are—(1.) *The Management of Infancy*, by Dr. Andrew Combe, ninth edition, 1860, edited by Sir James Clark; (2.) *Practical Hygiene*, 1864, the work of my colleague, Dr. E. A. Parkes, which has passed into a new edition. From the first of these books the student will learn how he must begin at the very beginning. He will learn to appreciate the influence of the constitution of parents on the health of their children, and will learn from it how to direct the management of infant life. From the second work he will learn how he may carry out the details of practical hygiene. This work of Dr. Parkes brings together an amount of material which shows to what extent exactness and certainty have been imparted to the science of Medicine. It is a work which will mark a new era in Practical Medicine.

From the nature of the CONSTITUTIONAL diseases (fully expressed at pages 33 to 36 of this volume, and described in the subsequent pages), it is to be noticed that there are three periods in the history of these diseases necessary to be recognized in the pathology and treatment of them. The first period may be described as *the period of their constitutional development*. During this period the Physician is rarely if ever consulted, unless a constitutional tendency to the disease is suspected, obvious, or perhaps hereditary. The second period may be described as the period during which the *constitutional disease is fully expressed* by the phenomena and symptoms already described as peculiar to each of them. The third period in the history of these diseases may be described as a *series of intervals*, during which the health seems to be improved between the febrile paroxysms, or fully expressed conditions of ill-health. It is for the cure of the fully expressed disease that the Physician is generally consulted—a period when he knows he can do the least good. But when the public are aware of the extent to which health may be preserved, and constitutional diseases averted and mitigated by judicious management and treatment during the first and last periods here noticed, the Physician will be more frequently consulted as to how the health is to be so preserved and improved as to ward off CONSTITUTIONAL DISEASES.

Whenever the Physician has to treat any of these constitutional affections in their fully expressed condition, if he is successful in subduing the symptoms for the time, by means of the treatment already mentioned under each of them, *the interval of comparative freedom from the paroxysmal expressions of disease is a time most precious, which ought not to be wasted, but which ought to be taken advantage of in preserving and improving the general health*. To effect this end there are three things to be considered in the management of the system during the intervals of comparative freedom from constitutional affections. These are—(1.) *Diet*; (2.) *The use of Water*; (3.) *The use of Wines and other alcoholic beverages*.

1. *With regard to diet*, one of the highest problems in physiology, as Dr. Parkes clearly enunciates, is so to regulate the supply of the *nitrogenous substances*—the *albuminates*—that the *digestive power* of the stomach and intestines may be increased, together with the *formative power* in the nitrogenous tissues, and the *eliminating powers*

in the after-stages of assimilation (*l. c.*, p. 132). All these three parts of the process must be duly balanced, otherwise health is destroyed. Half-digested food in the stomach or intestines produces irritation. It undergoes chemical changes in the alimentary canal, and quantities of gas are given off. Dyspepsia or constipation may be produced, or an irritation causing a diarrhœa which fails to empty the bowels. The fœces then contain a large amount of nitrogen, especially after eating vegetable albuminates. The tissues are unable to appropriate the excess which remains in the blood. Urea and carbonic acid, which ought to be eliminated in normal abundance, fail to be provided for from the imperfectly oxidized products of disintegration, and irritation of eliminating organs is set up. It is highly probable that gouty affections arise in this way, and partly from the use of liquids which delay metamorphosis. A great excess of albuminates without other food produces marked febrile symptoms, malaise, and diarrhœa, and ultimately albumen may appear in the urine, or extensive irritation of the skin may supervene. To increase the adaptation of the albuminates, fats and salts must be added to the diet; and the supply of oxygen must be increased by exercise, or the supply of starchy foods which appropriate oxygen must be diminished. Lessening the supply of the albuminates effects a decline or loss—first, of the muscular system; and at a later period, of the nervous system and mental powers. Such decline or loss may be delayed by increasing the supply of fats and starches, which by absorbing oxygen limit disintegration; and by perfect rest the loss of flesh may be still further delayed.

In the management of the system, therefore, the albuminous tissues can be, to a certain extent, brought under the control of the Physician by a judicious adaptation of diet and exercise alone, without the employment of drugs. Drugs, however, when judiciously employed, are important aids at two ends of the intestinal canal—namely, to aid primary digestion and to assist elimination.

*Fatty aliments* are essential to the formation of muscular, and especially also of nervous tissues. With the nitrogenous substances or *albuminates*, they are essential to the production of mechanical force. Animal fats appear easier of absorption than the vegetable fats; and the relative proportion of *fat* to *albuminates* ought to be as 1 or  $1\frac{1}{4}$  to 2.

*Starchy* and *sugary food* save the *albuminates* and *fats* from too rapid disintegration, so that, by a judicious employment of them in dietary, the elimination, and perhaps the formation of the *albuminates* and fatty tissues can be so modified that the administration or withholding of starches and sugars as articles of food must enter largely into the management of the system, according to the circumstances of the case, and the nature of the constitutional disease.

The production of *lactic acid* in the system seems probably connected with the metamorphosis of starch. To the constant introduction of an excess of alkali in the food, and the no less constant production of acid during the digestion of foods (especially starches, sugars, and fats), associated with the effects of respiration, are to

be ascribed the singular alternation of acid and alkaline fluids in the body. The relative amount of *starch foods* to the *nitrogenous* or *albuminate* substances ought to be as  $2\frac{1}{2}$  or  $3\frac{1}{2}$  to 1. Excess of starches, sugars, and fat produce excess of fat; sometimes also acidity and flatulence; and the urine may become saccharine under excess of starch as an article of diet. *Salts* and *water* are essential articles of diet. *Lime*, in the form of *phosphate*, is absent from no tissue; and when tissues degenerate and morbid growths are overgrown, or begin to decay, or when cells grow rapidly, as in *enchondroma*, lime is present in large amount. Both *lime* and *magnesia* are essential to the growth and repair of bone; and the judicious withholding of *lime* and *magnesia* in food is an important element in the constitutional management of diseases where the bones are affected, and in the repair of fractures. *Potash* and *soda*, in the form of *chlorides* and *phosphates*, are not less important constituents of diet. They form part of almost all the tissues; and, being especially concerned in the molecular currents amongst the elements of texture, seem to be less fixed than the magnesian and lime salts. The potash seems especially associated with the formed elements of texture, such as the blood-cells and the muscular fibres; while the soda salts are more largely concerned with the fluids which encompass these elements. These two alkalies (*potash* and *soda*) have been seen to be most important remedial agents in many diseases. *Chlorine*, *chloride of sodium*, *phosphoric acid*, *sulphur*, and *iron*, must be furnished in all food, either separately or combined with the foods already noticed.

The salts which form carbonates in the system, such as *lactates*, *tartrates*, *citrate*s, and *acetates*, exist chiefly in fresh vegetables; and although their nutritive power otherwise is small, it is absolutely necessary that they be supplied in the food. Scorbutic states inevitably ensue if they are withheld.

These are the main points to be inquired into and provided for in adjusting the diet best adapted for the management of constitutional diseases. Every intelligent patient may aid his physician, however, thus far,—namely, that if he consider the subject *bonâ fide* for himself, he will become the best judge of the exact diet which suits him. Dr. Parkes observes that probably 30 per cent. of the persons who consult physicians owe their diseases in some way to food, and in many cases they are perfectly aware themselves of their error or bad habit; yet, with the singular inconsistency of human nature, either conceal it from the man to whom they are professing perfect openness, or manage to blind themselves to its existence (*l. c.*, p. 461). Individual hygienic management must be inculcated upon the patient; while, by regulating diet on the principles laid down by Dr. Parkes in Chapter V of his great work *On Practical Hygiene*, the physician will find he can exercise a great power in the control and limitation of constitutional disease. Most valuable information respecting the adjustment of the dietary will be found in Dr. Edward Smith's *Practical Dietary*; and in *A Manual of Diet and Regimen for Physician and Patient*, by Dr. Horace Dobell.

2. *With regard to the use of Water*, its influence as an agent in the

management of certain conditions of constitutional ill-health is apt to be too much neglected, or not sufficiently appreciated. Ever since the powerful agency of water, under the popular name of the "*water cure*," has been so much bequacked, the medical profession have almost allowed themselves to be seized with a kind of *hydrophobia*. Nevertheless, there can be no doubt that water has a most powerful influence for good or evil in the treatment of constitutional diseases. Under its influence as a fluid of daily consumption considerable chemical changes are promoted in the body. In the intervals between the extreme expressions of constitutional diseases, such as *gout*, *rheumatism*, *asthma*, and the like, it is a useful and most efficient remedial agent, not only as a diuretic, but also as a local application, especially in obviating the congestions of the viscera associated with these constitutional affections (SIMON). "To accelerate the defective textural metamorphosis or waste is very often the practical result to be aimed at, and such means exist in the simplest and most manageable form in many of the appliances of the so-called '*water cure*.' With increased water-drinking there is increased discharge of solids by the urine. External appliances, such as the cold *sitz bath*, lasting a quarter of an hour, increase for a time the elimination of urea and uric acids. The protracted sudorific packings produce a still more considerable waste of tissue. Generally, therefore, in cases where the use of water is suitable, and conducted under competent medical advice and direction, the healthy nutrition of the body is promoted in proportion as refuse materials are actively disengaged" (SIMON, in *Holme's Surgery*, vol. i, p. 118).

3. *The properties of Wines, and of alcoholic beverages generally*, offer a wide and important field for the careful study of the physician. Although so extensively used as beverages with our daily food, and so generally recognized as valuable agents in the treatment of disease, there is not only great diversity of opinion as to their precise effects, but as yet very little is certainly known of the action of alcohol when administered in the forms of *Wine*, *Beer*, or *Spirits*. None of the general statements so frequently met with as to the composition or effects of any particular class of beverages can be relied on as a guide to the Physician in prescribing; and much error seems to prevail on the subject, not only in the popular mind, but amongst medical men.

*Alcohol* is the most potent agent for good or evil in all of these beverages; and, therefore, its amount and its effects challenge attention in the first instance. A pint of Beer (twenty ounces) may contain *one or two or more ounces of absolute alcohol*, or *less than a quarter of an ounce*. This alcohol may be associated in the beer with an amount of *free acid* varying from *fifteen to fifty grains*, and with an amount of *sugar* varying from *half an ounce to three or four times that quantity*. A glass of Sherry (two ounces) may contain from a *quarter to half an ounce or more of absolute alcohol*, with *sugar* varying in quantity from *a mere trace to twenty or thirty grains*, associated with *a very variable amount of free acid*, and with other ingredients. Even in Brandy or Whiskey the amount of alcohol is widely different in different specimens.



It is impossible, therefore, for a physician to know what his patient is drinking unless he is acquainted with the chief constituents and their amounts contained in the identical liquor which he may prescribe; and of course, before sound conclusions can be arrived at, the conditions under which these beverages are administered or taken must also be very precisely observed.

The physiological effects of alcohol have been investigated and inquired into with considerable care by Rudolf Masing, Böcker, and Mulder; by MM. Lallemand, Perrin, Duroy; by Percy, Ogston, Bence Jones, Carpenter, Spencer Thomson, Hammond, T. K. Chambers, Edward Smith, Anstie, and Parkes.

All the observations and inquiries on this subject tend to the conclusion that *alcohol* passes through the body unaltered in chemical constitution, and does not, so far as we know, leave any of its substance behind. At the same time, there is ample evidence to show that a very large amount may be retained in the fluids of the brain for an indefinite length of time (PERCY, AITKEN, and others, see vol. i, pp. 772 to 776). During that period of retention it exerts an influence for good or for evil; and although it may not be regarded as an "aliment" in the strict sense of that term, it undoubtedly aids the appropriation of aliment under some circumstances; and so far may be regarded as an "accessory to food" in comparative health, or as a "*medicine*" in disease. Under this aspect of the subject, Dr. T. K. Chambers, in his interesting clinical lectures (p. 570), lays down the following rules for the administration of alcohol:

1. Alcohol may be given with advantage when the nervous system is exhausting itself, and when the tissues of the body generally are being exhausted by an activity in excess of the other bodily functions. It lessens the destructive metamorphosis which goes on; and chemical changes in the blood are partially arrested (HARLEY, quoted by PARKES).

2. It must be given, increased in amount, or left off, under the guidance of the appetite for food. As long as a person in ill-health takes and digests food better with alcohol than without, so long will alcohol be of service to him. Beyond this general statement there is no evidence. In *very small quantities* it appears to aid digestion in the stomach; in larger amounts it checks it. A moderate use of beer or of the weaker wines (*i. e.*, of pure unbranded well-fermented wines) may increase appetite and improve nutrition. On the other hand, the use of malt liquors (even when pure and good) is injurious to persons of sedentary habits, or unless much exercise be taken in the open air; but sound, well-fermented beer is the best of all dinner drinks *for persons of good digestion, who work hard in the open air* (DRUITT).

3. When the marked features of disease consist in the retention of effete matters which ought to be discharged, the use of alcohol must be totally abstained from, as, for example, in Bright's disease.

4. The daily allowance of alcohol ought to be divided into two or three doses only.

Under all circumstances its effects must be watched; for while it may sometimes be desirable to diminish the metamorphosis of tis-

sue by its use, it must not be forgotten that large quantities of alcohol tend to cause an accumulation in the system of imperfectly oxidized bodies, such as *uric* and *oxalic acid*.

The general evidence also tends to show (but does not absolutely prove) that pure alcohol has its pernicious effects greatly lessened, and its good effects more powerfully developed, when highly diluted, and still more so by admixture with other substances, as the carbohydrates and salts contained in *beer* and *wines*. The use of strong wines (15 to 23 per cent.) undiluted should therefore be discouraged as much as possible; and if such an amount of alcohol is found necessary for the due preservation of the wine, and naturally results from the fermentation of the grape, the wine ought to be diluted with water when used as a beverage. On the other hand, light wines cannot be long exposed to the atmosphere without acetous fermentation commencing in them. Light wines with a small amount of alcohol for the use of invalids must therefore be preserved in bottles of such a size that the whole may be consumed in a day; for much mischief may arise if wines are used by invalids after acetous fermentation has commenced. Besides alcohol and ethers, wine contains several substances of great value as articles of diet,—namely, some albuminous substances, such as *sugar*, as well as other carbohydrates, and abundant salts. The vegetable salts are most valuable, and in this respect are highly antiscorbutic (PARKES, *l. c.*, p. 227).

For the purposes of the Physician, the prominence of certain substances in wines renders some more desirable than others, or more useful as “*medicines*.” These substances are—(1) *alcohol*, (2) *acids*, (3) *sugar*, (4) *solids* or *extracts*. Of these in their order:

**Alcohol**, to a variable amount, ought to exist in wines as a natural product of the fermentation of the grape; and the conditions of its existence in the wine are then very different from those which obtain when alcohol (distilled even from wine) has been added to wine. It is highly desirable, therefore, to avoid adventitious alcohol; but it is impossible to prove that spirit has been added to a wine unless the amount is absurdly excessive. One vintage sometimes produces a wine with a considerably greater amount of *alcohol* than another of the same vineyard. For example, the samples tested in London (for duty) of *Chateau Lafitte*, of vintage 1858, showed 16.5 per cent. of proof spirit; the same vineyard in 1859 showed 17.7; and in 1860 it gave 14.8; the three years varying about 3 per cent. The samples of common *St. Emilion* in 1858 showed 16.5 per cent. of proof spirit; in 1859 the same vineyard showed 15.4 per cent.; and in 1860 it gave 16 per cent.;—thus not differing more in the three years than about 1 per cent.

The amount of the alcohol by measure may be most readily estimated by the vaporimeter of M. Geisler, of Bonn, which indicates the amount of the alcohol by the tension of vapor at a certain temperature, from the fluid containing the alcohol forcing up a column of mercury. Dr. Parkes gives also a very ready process by evaporation, and the use of a urinometer (*Practical Hygiene*, p. 220).

Having thus ascertained the percentage of alcohol in *wine*, *beer*,

or *spirits*, it is easy to calculate by simple proportion the dose of alcohol administered. For example, a pint bottle of Claret (*thirteen* ounces), of the strength of *eleven* per cent. of alcohol, will be found to contain 1.43 ounces of absolute alcohol, thus:

$$100 : 11 :: 13 = 1.43.$$

It is the basis of the rule laid down by Dr. Parkes (*l. c.*, p. 224), which says,—“*To tell how much pure alcohol is taken in any definite quantity of wine, measure the wine in ounces, and multiply it by the percentage of alcohol with a decimal point before it.*”

**Acidity.**—Free acid in wine is a necessary result of its fermentation. Its presence is likewise necessary for the evolution of the *bouquet*, for the agreeableness of the wine, and for its wholesomeness. It is, therefore, a popular error to denounce absolutely the existence of acidity in wine. The *relative amount of free acid* present in any particular wine is a very important point to determine. Much error prevails respecting the relative amount of acidity in different wines, and an excessive amount of free acid is very easily disguised by the relative sweetness of the wine. The estimation of the free acid may be measured by a solution of carbonate of soda, containing 530 grains in the 10,000 grain measure—53 grammes in the litre; and the amount of acidity is represented by determining how many grains of crystallized oxalic or acetic acid a certain quantity of the soda solution will neutralize. The details of the process are as follow:

(1.) Take 50 or 100 c. c. of recently opened wine; (2.) Add from a burette a standard solution of soda, in small portions at a time (say 5 c. c., or drop by drop); (3.) After every addition, test the fluid by moistening a thin glass rod or feather with the mixture, and streak it across some well-prepared violet litmus paper;—when the streaks cease to become red, the analysis is complete; (4.) Estimate how much of the standard solution has been used, and express the acidity as equal to so many grains of crystallized oxalic or acetic acid. Good wine contains a quantity of acid that is equivalent to from 300 to 450 grains of crystallized tartaric acid in a gallon. Wines with less than 300 grains of acid in a gallon are too flat to be drinkable with pleasure. Wines with more than 500 grains in a gallon are too acid to be pleasantly drinkable; and wines with more than 700 grains in a gallon are undrinkably sour (Druitt's *Report on Cheap Wines*, p. 178).

It is also very important to get at the *quality* of the acid,—to the extent, at least, of dividing the *volatile* from the *non-volatile* (and less digestible acids). A large amount of acid and acid salts may not be readily digested; and in many diseases the supply of acid to the system is a thing to be desired or prevented (see also PARKES, *l. c.*, p. 219).

The necessary standard solutions and burette apparatus are supplied by Mr. Griffin, 119 Bunhill Row, who has prepared a set of apparatus which shows all that chemistry can teach of the quality of wine; by Messrs. Bulloch & Reynolds, Hanover Street, London; also, Harper & Sutton, Operative Chemists, Norwich.

**Sugar** is characteristic of all the *sweet* wines, and of many wines in their immature condition. Its amount tends to diminish with age, so that old wines of the *sweet* sort may be less pernicious, as regards sugar, than the new. *Sweet* wines, or wines which contain a large percentage of sugar, are to be avoided by those who are disposed to corpulence; and they are extremely injurious to those who are disposed to the formation of *oxalic acid*, or to the discharge of sugar in the urine. Its amount in any particular sample of wine is best determined by Soleil's *saccharimeter*. The principle on which the process depends is that the varieties of sugar possess the power of twisting the plane of polarization of a ray of polarized light which is transmitted through solutions containing these varieties of sugar. *Cane sugar* and *glucose* twist it to the right hand; *fruit sugar* twists it to the left hand. All gradation lists given in books as to the amount of sugar in wines are purely empirical, and apply only to the particular sample of wines examined. In stating the sweetness of Champagne, for example, nobody seems to notice that it is a manufactured article; and that the quantity of *syrup added to it* actually varies from *four* to *twenty* per cent. The Champagne known as "*still*" has no sugar. Some sorts of Madeira are also nearly free from sugar; and some sorts of Sherry are much sweeter than well-fermented Ports.

The correlation of sweetness with acidity and with alcohol, are most important points to be determined with reference to the easy digestion of wines; and hence they are all-important for the physician to know.

**The Amount of Solids** may be learned (approximately) by determining the specific gravity after the alcohol is driven off. A low specific gravity shows that alcohol has been added, or that the solids are in small amount (PARKES, *l. c.*, p. 226).

The following table gives a standard for the determination of the solids (PARKES, *l. c.*, p. 218):

Specific gravity after loss of alcohol.	Per cent. of extract.	Specific gravity after loss of alcohol.	Per cent. of extract.
1004, . . . . .	1	1024, . . . . .	6
1008, . . . . .	2	1028.1, . . . . .	7
1012, . . . . .	3	1032.2, . . . . .	8
1016, . . . . .	4	1036.3, . . . . .	9
1020, . . . . .	5	1040.4, . . . . .	10

It is much to be regretted, especially for the sake of invalids, that so few wine merchants are acquainted, or care to be acquainted, with the chemical constituents of their different wines. Medical men should, therefore, deal only with those wine merchants who will tell them the amount at least of the several constituents in their wines that have been referred to—namely, of alcohol, acids, sugar, solids—and who will guarantee the wine they supply as being in accordance with the sample of which they give the analysis. The sale-room of every wine merchant ought to be provided with the means and appliances here indicated for ascertaining the exact amount of the substances contained in the samples of wine they offer for sale. *The amount of alcohol, of free acid, of sugar, and*

of solids should be recorded also of every wine on importation, so that the wine merchant may be able to watch the metamorphoses, in respect of these constituents, effected on them by lapse of time. Wine merchants could thus materially assist the efforts of medical men in the cure of disease, and physicians could then prescribe with some knowledge of what they were prescribing; and to that extent, at least, the welfare of invalids and the sick would be better cared for.

But although by ascertaining these facts a physician may prescribe with better knowledge of what he is prescribing, and to that extent at least be better able to care for his patients, it must not be concluded that chemistry can detect every form of adulteration in alcoholic beverages. As Dr. Druitt justly observes, "the only real test for wine is the empirical one. It is impossible to say that such a wine must be good in such and such cases, because it contains certain ingredients. . . . The stomach is the real test-tube for wine; and if that quarrels with it, no certificate of Liebig and no analysis are worth a rush" (*Report on Cheap Wines*, p. 6). With all possible aids, the one safeguard against fraud is to deal only with wine merchants and brewers of established character and reputation, who will be above attempting to dispose of unsound or adulterated liquors; and to avoid being tempted by the low prices of amateur dealers in wine, or of advertising companies and others, obviously more anxious to sell than to sell only the purest and the best.

The principal analyst of the Inland Revenue Department reports, that he found illicit ingredients in no less than twenty out of twenty-six samples of beer tested last year (1865); and he believes that, owing to the difficulty of detection, the practice of adulterating beer with poisonous ingredients is much more prevalent than might be inferred from the small number of discoveries made. These samples were all from the stores of licensed brewers; and although, doubtless, further adulterations are practised by the retailers, they are probably for the most part with less noxious materials, and chiefly with the object of increasing bulk—known as "*stretching*." As to wines, the following advertisement, frequently appearing in the *Times*, shows that there is a trade openly carried on in this country in the treatment or *cure* of bad or spurious wines:

"Wine merchants having wines turned acid may have them returned to their original wholesome perfection in two or three days by applying to —," &c., &c.

It is well known that the same practices prevail abroad, so that "pure as imported" is now no guarantee for purity of production. Hambro' wine has been shipped from London to Cadiz and back—an operation which raises the price of the stuff 100 per cent. The custom-house officers have lately (1865) stopped a large quantity of stuff imported as "Sherry," which had not a drop of grape-juice in its composition; and it cannot be doubted that the increased consumption, and increased competition for low prices have greatly



added to the temptations to adulterate. Such liquors, for any medicinal purposes, must be worse than useless; and whenever Wine, Brandy, or Beer is prescribed medicinally, they ought to be obtained from wine merchants or brewers of the highest respectability. Wine merchants are generally quite willing to sell in small quantities either wine or spirits—a recent change in licensing laws enabling them to do so, and so making it no longer necessary to send to the public-house when only a single bottle is wanted.

The blindly empirical and routine mode in which alcoholic beverages are generally prescribed, in absolute ignorance of their constitution and genuineness, and the importance of them in the treatment of disease or of ill-health, renders it advisable in a text-book to insist fully on these topics, believing that the Physician cannot cope successfully with diseases, and especially with CONSTITUTIONAL DISEASES and the ill-health with which they are associated, unless he learns judiciously to use the immense power at his disposal in the influence of *diet, water, and alcoholic beverages*, as agents in the management of the system during the intervals between the paroxysms of these diseases. For much valuable information on wines, given in a very pleasing form, the reader is referred to *Report on the Cheap Wines from France, Italy, Austria, Greece, and Hungary; their Quality, Wholesomeness, Price, and their Use in Diet and Medicine*, by Robert Druitt, Esq., F.R.C.P., 1865.

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### CLASS III.

LOCAL DISEASES; OR, DISEASES IN THE COURSE OF WHICH CERTAIN LESIONS COME TO BE LOCALIZED.

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### CHAPTER I.

GENERAL NATURE OF THE DISEASES COMPREHENDED IN THIS CLASS.

THIS class is intended to comprehend all those diseases which affect the structure of special organs or particular parts of the body, which lead to marked disturbance of their functions.

Local affections are often accompanied by constitutional symptoms. Such symptoms are to be regarded as secondary when they succeed to and depend upon the existence of the local lesion. Many diseases where the lesions are strictly localized and defined are really the results of *constitutional* or *zymotic* disease; and many have been already described under the several diseases that have been considered in the previous pages of this work. The local affection in such cases is often so striking and important that it has especially challenged attention, while the constitutional state from

which it may have sprung is as yet concealed and unknown. Not a few cutaneous eruptions are of this nature; so also are some forms of dropsy.

The diseases about to be considered are essentially characterized by more or less defined local lesions, of the nature of—(a.) *Catarrh*, or increased flow of secreted fluid; (b.) *Inflammation of the suppurative, ulcerative, plastic, pyæmic, rheumatic, gouty, syphilitic, scrofulous, or gonorrhæal*; of the nature of—(c.) *Gangrene*; (d.) *Passive congestion*; (e.) *Extravasation of blood, or hemorrhage*; (f.) *Dropsy*; (g.) *Fibrous deposits*; (h.) *Altered dimensions, as dilatations, contractions, hypertrophy, atrophy*; (i.) *Degenerations, amyloid or lardaceous, fatty, atheromatous, pigmental, calcareous, fibroid*; (k.) *Syphilitic*; (l.) *Cancer*; (m.) *Non-malignant tumors*; (n.) *Cyst*; (o.) *Scrofula, with or without tubercle*; (p.) *Parasitic lesions*; (q.) *Calculus and concretions*; (r.) *Malformations*; (s.) *Injuries*; (t.) *Functional diseases*.

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## CHAPTER II.

### GENERAL REMARKS ON THE PATHOLOGY OF BRAIN DISEASES, AND DISEASES GENERALLY OF THE NERVOUS SYSTEM.

It is with nerve-texture, as composing the essential parts of the nervous system contained within the cranium, and distributed as nerves throughout the body, that we have to deal in describing the local diseases of this order.

The interest which attaches itself to the study of the nervous system cannot be surpassed either in a physiological or pathological point of view. The truth and force of this statement will be more especially apparent when it is remembered that the nerves are the channels of Sensation; that nervous textures compose the organs which conduct the influence of the will to the muscles, before motion can take place; and that while the textures which constitute the *nervous centres* associate Sensation on the one hand, they at the same time balance and co-ordinate the motions of the body on the other. And that, lastly, being the seat of the various mental processes through which *Sensation, Volition, Memory, Judgment*, and *all mental acts* are expressed, the pathological relations of the nervous texture contained within the cranium or distributed throughout the body, are perhaps the highest and most important, in a scientific, philanthropic, and sanitary point of view, to which the mind of the physician can be directed.

The student is therefore called upon to give the Brain diseases and those of the Nervous System generally, a large share of his study.

The varied phenomena connected with the morbid conditions of the nervous system must be examined from the following points of view, namely: (1.) The purely anatomical structure of the Brain

and Nerves; (2.) The chemical composition and properties of the nervous substance; (3.) The physiological relations of the several parts; (4.) The morbid and pathological relations. Each of these methods of study and investigation will mutually illustrate one another; and it is only from a consideration of all of them conjointly that we can arrive at the Natural History of a case, and so act for the best in diagnosis, prognosis, and treatment. Every student knows how very many physiological doctrines regarding the Brain and Nerves receive elucidation from accurately determined anatomical information; and so such knowledge tends to explain various points in the pathology of cerebral diseases. He need only be here reminded of the phenomena explained by the decussation of the *pyramids* in the *medulla oblongata*; how the continuity of the fibres of the spinal cord upwards to the *cephalic centres* explains various secondary lesions of the brain as a consequence of lesions in the spinal cord altered by paralysis (Drs. Brown-Séquard, Turck, and Waller); and, lastly, the interesting observations made by Drs. Walshe and H. Bence Jones regarding the excretion of sulphates and phosphates by the urine in *acute chorea*, *delirium tremens*, and *inflammation of the brain* itself. These observations show, to some extent, how accurate chemical and anatomical investigations may become valuable in the elucidation of morbid phenomena occurring in living bodies.

**Anatomical Constituents of the Brain and Nerves.**—The nervous texture may be simply considered as arranged into three great divisions:

1. A large quantity of nervous matter collected into one mass, and contained in one cavity—the cerebro-spinal. This mass is called the *Brain and Spinal cord*, or *Encephalon*, or *Cerebro-spinal axis*, or *central part of the nervous system*; and is composed of the *cerebrum*, *cerebellum*, *sensori-motor ganglia*, and *spinal cord*.

2. A nervous-texture, arranged in the form of long, continuous cords or threads, mutually connected, and running in every direction throughout the body. These are simply called the *Nerves*.

3. An accumulation of peculiar nervous substance, in the form of small, round, and somewhat oval masses, called "*ganglia*," variously connected with each other, and with the surrounding parts, and forming what is known as the *Sympathetic system*.

**Chemical Composition of Brain and Nerve-Tissue.**—The white or gray matter of the brain has been generally taken to represent pure nerve-substance. It consists of albumen, fatty matter, salts, and from  $\frac{1}{8}$ ths to  $\frac{1}{3}$ ths of water. The fatty constituents are remarkable, inasmuch as two of them, being acid compounds, contain a large amount of phosphorus, from 8 to 10 parts in 1000 of the mass, or  $\frac{1}{8}$ th to  $\frac{1}{10}$ th of the whole solid matter. This is continually being metamorphosed during functional and morbid changes of the nerve-substance; and the amount of alkaline phosphates in the urine may be taken in some measure as an estimate of the amount of nerve-tissue disintegrated, the earthy phosphates being disregarded, inasmuch as they have been shown mainly to depend on the quantity taken in the food. The phosphorus set free by disintegration of the

nervous tissue unites, in the form of an acid, with the alkaline basis in the blood, and is then separated by the kidneys and discharged with the urine.

**Weight of the Brain and its Parts.**—In appreciating morbid states of the brain and nervous textures after death, it is useful to remember that the absolute and specific weights of the brain range within certain limits, consistent with the healthy exercise of function. The following statements are made of standard numbers for reference and comparison illustrative of this point:

**Absolute Weight.**—The absolute weight of the brain or encephalic mass varies in concurrence with variations of age, body-weight, and height of persons; and generally it may be stated as in the table on p. 271, compiled from the valuable records communicated to the Royal Society on the 28th February, 1861, by Dr. Robert Boyd, the Physician to the Somerset County Lunatic Asylum.

It is by accurate observations such as these that the gradual growth of every organ of the body is demonstrated to advance slowly and in concurrence with advancing age and increase of body-weight. It is now also a matter of experience, although too often overlooked, that the functions and organs of the body, and by no means rarely the brain and the Intellect, may be injured for life by pressing upon them too hardly and continuously in early years. Whatever theory we hold (as to the functions of the brain or Mind), "it is certain that the powers of the brain are only gradually developed; and that if forced into premature exercise, they are impaired by the effort. This is a maxim, indeed, of great import, applying to the condition and culture of every faculty or function of body and of Mind, and singularly so to the Memory, which forms in one sense the foundation of intellectual life. A regulated exercise *short of fatigue* is improving to it (and to all faculties or functions), but we are bound to refrain from goading it by constant and laborious efforts in early life, and before the instrument is strengthened to its work, or it decays under our hands" (Sir Henry Holland's *Mental Pathology*).

**Bulk.**—The bulk of the encephalon varies from 65 to 84 cubic inches.

**Specific Gravity.**—The result of recent observations in Germany, France, and Britain, shows that any considerable change in the specific gravity of the cerebral substance is incompatible with a healthy exercise of the nervous functions.

To Dr. John Charles Bucknill, Physician to the Devon County Lunatic Asylum, medical science is indebted for the first most extended account of the specific gravity of the cerebral substance, and its relation to disease, and more especially to *atrophy* and *paralysis*. The following are the general results of his observations as detailed in *The Lancet*, 25th December, 1852, and for the most part made upon patients laboring under different forms of mental disease:

(1.) Average specific gravity of healthy brain, 1.036. (2.) In paralysis of a chronic character, complicated with insanity, the specific gravity ranged between 1.036 to 1.046. (3.) In some acute cases the specific gravity was as high as 1.052. (4.) In paralysis terminating

TABLE SHOWING THE RELATIVE AVERAGES OF BODY-WEIGHT AND THE WEIGHT OF CEREBRAL ORGANS AS TO AGE AND HEIGHT.

Age.	Sex.	Body-Weight.		Height of Body.	Weight of Cerebrum.	Weight of Cerebellum	Weight of Pons and Medulla.	Weight of Encephalon.
Years.		Lbs.	Oz.	Inches.	Ounces.	Ounces.	Ounces.	Ounces.
1 to 2	Male, . .	14	6	28.5	29.21	3.54	.5	33.25
	Female, .	13	2	27.7	26.19	3.15	.46	29.8
2 to 4	Male, . .	20	0	31.6	34.03	4.02	.66	38.71
	Female, .	18	7.5	31.6	30.77	3.7	.5	34.97
4 to 7	Male, . .	25	8	37.5	35.44	4.17	.62	40.23
	Female, .	24	9	37.0	35.04	4.19	.68	40.11
7 to 14	Male, . .	42	6	47.0	40.36	4.84	.76	45.96
	Female, .	38	6	45.0	35.86	4.27	.65	40.78
14 to 20	Male, . .	68	0	60.5	41.77	5.32	1.0	48.54
	Female, .	63	14	57.7	38.88	4.65	.85	43.94
20 to 30	Male, . .	92	14.5	66.75	41.98	5.19	.93	47.9
	Female, .	86	13	62.0	38.0	4.82	.88	43.7
30 to 40	Male, . .	98	3.5	66.5	42.06	5.15	.98	48.2
	Female, .	87	0	62.0	37.92	4.74	.91	43.09
40 to 50	Male, . .	102	0	66.8	41.48	5.22	1.06	47.75
	Female, .	84	9.5	62.0	37.12	4.69	.89	42.81
50 to 60	Male, . .	102	0.5	66.0	41.09	5.13	.96	47.44
	Female, .	86	0	62.0	37.38	4.62	.86	43.12
60 to 70	Male, . .	103	13	65.7	40.21	4.98	.97	46.4
	Female, .	86	14	61.5	37.13	4.68	.83	42.69
70 to 80	Male, . .	106	13	65.7	39.6	4.97	.94	45.5
	Female, .	80	4	61.0	35.58	4.47	.88	41.27
80 to 90	Male, . .	99	0	66.7	39.62	4.79	.89	45.34
	Female, .	79	0	60.0	34.47	4.47	.82	39.77

by coma, 1.040. (5.) In paralysis terminating by syncope or asthenia, 1.036 to 1.039. (6.) In general terms, a higher specific gravity was found when life terminated by coma or asphyxia, than when it ended by syncope or asthenia.



In addition to these observations, an able and elaborate paper has since been published by Dr. Sankey, showing the relative specific gravity of the *gray* and *white matter* of the brain, and of so extensive a nature as to furnish very copious data for comparing morbid states with the standard of health. The following are the general results of his researches, as given in *The British and Foreign Medico-Chirurgical Review* for January, 1853, p. 257 :

(1.) Mean specific gravity of the gray substance of the brain in either sex, 1.034. (2.) In the earlier and later periods of life the specific gravity of the gray matter is below the mean. (3.) The cerebral substance acquires its greatest density in males between the ages of fifteen and thirty, and in females between the ages of twenty and thirty. (4.) The density diminishes with prolonged illness. (5.) It decreases with a lapse of time after death in the ratio of .001 for every twenty-four hours. (6.) A density of .006 above the average indicates the existence of the following conditions during life: Acute cerebral symptoms, or chronic disease with no cerebral symptoms, or only slight delirium; also with conditions associated with hyperæmia. (7.) Mean specific gravity of white matter, 1.041.

Both sets of observations referred to above have been made upon the brain as a whole; and as the observations of Dr. Sankey show that no constant relation exists between the absolute weight of the brain and its specific gravity, it is necessary to examine the brain as we do its anatomy—namely, by comparative observations on its *central parts* or *ganglia*.

At the time Dr. Bucknill published his observations I was engaged in determining the specific gravity of the central parts of the brain, which are sometimes called the *central ganglia*, and which are now generally regarded as the parts more immediately related to the combined exercise of sensory and motor functions. These centres consist of the *corpora striata*; *thalami optici*; *tubercula quadrigemina*; and the large mass of vesicular nervous matter associated with the convolutions of the hemispheres and the substance of the cerebellum. While these parts are the immediate seats of the origins of the nerves, they may be looked upon as parts where some changes in connection with the functions of special nerves are constantly going on, of such a kind that a result is expressed through “Volition, Perception, or Emotion, or the balancing or co-ordinating of movements” (TODD.)

These parts have a specific gravity as follows: The central ganglia, 1.040 to 1.047; the cerebrum, 1.030 to 1.048; the cerebellum, 1.038 to 1.049.

The same kind of morbid states which modified the specific gravity of the brain-substance, as recorded by Drs. Bucknill and Sankey, also manifest their influence on the central parts. Thus death, by *coma*, and especially in *typhus fever*, was indicated by an extremely high specific gravity; and while it was observed that a slight difference was common in most cases when similar parts on opposite sides were compared, it is presumed that further observation, extended in this direction, especially in cases of hemiplegia, may lead to important results. In one case of *choreic hemiplegia* which I had an opportunity of carefully investigating, the specific

gravity of the *corpus striatum* and *optic thalamus* on the right side was found to be 1.025, while the specific gravity of the corresponding parts on the left side was observed to be 1.031 (*Glasgow Med. Journal*, No. 1, 1853).

**Pathological Relations of the Nervous Organs and Texture.**—Our knowledge of these relations is necessarily imperfect, and for the following reasons: (1.) The functions of the various parts which, connected together, constitute the encephalon, are not yet determined accurately. (2.) The inconstancy and irregularity of the functional disorders which accompany the morbid state of the nerve-substance render it difficult to interpret the value of the symptoms by which the *nervous diseases* are manifested. (3.) Some of the diseases of the brain and nervous system which are marked by the most violent symptoms during life, such as *epilepsy*, *chorea*, *tetanus*, and *hydrophobia*, leave after death no constant lesion capable of being detected with the unaided eye, or even by microscopic examination; while *tumors* and serious destruction of the nervous mass may exist during life without producing any severe or pathognomonic symptoms whatever. (4.) We have no means of applying physical diagnosis to the cranium, as we have to the chest, although it has been proposed by some (Drs. John Fisher and Whitney) to found diagnosis upon *Cerebral Auscultation*. For an abstract of the nature of the investigations "On the Auscultation of the Brain" the reader is referred to Wood's *Practice of Medicine*, vol. ii, p. 621, and also to a notice of Henning's "Inaugural Dissertation on the Sounds perceptible about the Head and at the upper portion of the Spinal Column in Children," in *Med.-Chir. Review* for 1857, p. 528.

The general principles on which the pathological relations of the *brain diseases* are determined, rest upon the anatomical, chemical, clinical, and physiological facts now accurately known. An accurate knowledge of anatomy is most essential in the study of disease of the nervous system.

In connection with the physiological view of the subject there are several cardinal facts which must be constantly kept in remembrance, and which may be shortly referred to here.

There are separate and distinct functions performed by the *gray* and *white* matter which enter into the structure of the nervous centres and organs. Reasoning from the general properties known to be possessed by cells in other structures, it is now a generally received doctrine, that the cells of the *gray* substance of the brain and nervous centres are the seat or source of that force which has received the name of the "*nervous force*, *nervous power*, or *nervous influence*," and which makes itself known by *sensation* and *motion*, as also by the various ways in which the *mental acts* are expressed. The *white nerve-fibres* are in connection with the *gray* or *cell-elements* of the nervous tissue, conducting from and to these centres the "influences" which are sent to, or which originate there and are thence sent forth.

The union of the nerve-tubes with the nerve-corpuscles is supposed to be connected with the transference of action from one nerve-fibre to another, as in *reflex action*.

It is likewise a remarkable fact that each nerve-fibre in a fasciculus acts quite independently from end to end,—quite isolated from the others in its vicinity; and thus at once we have the enunciation of three distinct sets of physiological phenomena associated with the diseases of the brain and nerves.

*First,—Phenomena of Isolated Conduction.*—Exalted or diminished action is presented by that nerve-fibre only which is affected by the irritating or depressing cause, and the adjoining fibre, though in ever such close approximation, is not implicated.

*Second,—Phenomena of Sympathy or Irradiation of Sensations.*—That irritation is propagated from a fibre originally excited to other centripetal nerves.

*Third,—The Phenomena of Intelligence.*—The brain furnishes the conditions necessary for the manifestation of the intellectual faculties, properly so called, such as the Emotions, Passions, Volition, and is at the same time essential to Sensation.

That the evolution of power or nerve-force immediately connected with Mind is dependent on or emanates from the hemispherical ganglia, is rendered probable by the following facts: (1.) In the animal kingdom generally a correspondence is observed between the quantity of gray matter, the depth of the convolutions, and the sagacity of the animal. (2.) At birth the gray matter of the cerebrum is very defective, so much so that the convolutions are, as it were, in the first stage of formation, being only marked out by superficial fissures, confined to the surface of the brain; and as the gray substance increases, Intelligence becomes developed. (3.) The results of experiments have shown that, on slicing away the brain, the animal becomes more dull and stupid in proportion to the quantity of gray substance removed. (4.) Clinical observation points out that in those cases in which the disease has been found to commence at the circumference of the brain, and proceed towards the centre, the mental faculties are affected *first*—e. g., *meningitis*, and the like; whereas in those diseases which commence at the central parts of the organ, and proceed towards the circumference, the mental faculties are affected *last*—e. g., *tumors* in the central white substance.

The white tubular matter in the form of the diverging fibres of the brain conduct influences, originating in the *hemispherical ganglia*, to the nerves of the head and trunk; while they also conduct, in an inverse manner, the impressions made on the peripheral parts up to the *cerebral convolutions*.

The spinal cord, by its connection with the brain, furnishes the conditions necessary for combined movements; and that its nervous force is also dependent upon its gray matter is rendered probable by the following facts:

(1.) The universal connection of the gray matter with all motor nerves. (2.) Increased quantity of the gray matter in those portions of the spinal cord whence issue large nervous trunks. (3.) The collection of gray matter in comparatively large masses at the origin of such nerves in the lower animals as furnish peculiar organs requiring a large quantity of nerve-power, as in the *Torpedo*, *Gym-*

*notus electricus*, and *Silurus*. (4.) Clinical observation shows that in cases where the *central portion* of the cord is affected previous to the external portion, the individual retains the sensibility and power of moving the limbs, but wants the power to stand or walk; whereas, when disease commences in the *meninges* of the cord, pain, twitching, convulsions, numbness or paralysis indicates lesion in the white conducting matter.

Independent endowment of nerves is shown by the fact that, whatever be the stimulus which calls their power into action, a uniform functional result is obtained; and hence it is inferred that the nerves are not altogether the mere conducting tubes of a stimulus from one place to another, but are in some respects the seats, or agents, or apparatus of power.

As far as we know, the brain alone furnishes conditions necessary for Intelligence, the spinal cord conditions essential to Movement; and together they furnish conditions connected with the balancing and co-ordination of motor and sensific power.

In dealing with the diseases of this order,—the *Cephalici*,—it is incumbent on the physician to ascertain, as correctly as possible, the *locality of the lesion*, the *nature of the affection*, and the *anatomical condition of the part affected*. Although it has been sometimes asserted that it is of little practical importance to discriminate accurately between diseases of one part of the brain or of its membranes and those of another, because the treatment may be the same for all, yet, for the sake of science—because “knowledge is power,” and because the acquisition of such knowledge must eventually alleviate the sufferings and lessen the sorrows of humanity—the sooner such doctrines are ignored the better for the Science of Medicine; and, moreover, the majority of the medical profession are beginning to appreciate the principle that diagnosis should be carried as far as possible. To the advanced student, who would desire more minute information to guide him in the differential diagnosis of brain diseases than can be given in a text-book of medicine, he is recommended to study the work of Dr. J. Russell Reynolds, Professor of Clinical Medicine in University College, on the *Diagnosis of Diseases of the Brain, Spinal Cord, and Nerves*, as well as the writings of Dr. Sanders, of Edinburgh, Dr. H. Jackson, of the London Hospital, and Mr. Lockhart Clark, and the comprehensive manual of Tuke and Bucknill *On Insanity*. From these and other works the general remarks and descriptions of brain diseases have been mainly compiled; and the method of classifying the phenomena of these diseases adopted by Dr. Reynolds has been followed throughout this text-book.

## CHAPTER III.

## GUIDES TO THE DIAGNOSIS OF BRAIN AND NERVOUS DISEASES.

1. **As to Locality or Site of Lesion.**—As yet we are able only in some cases of tumors of the encephalon to define their locality as to whether they are in the cerebrum, cerebellum, or central ganglia.

With regard to the cerebrum, it may be determined in the majority of structural diseases which lateral half is affected, and in particular cases it may be predicated with strong probability that the lesion is situated in some one of the following sites: (1.) The substance of the hemispheres (cortical or central); (2.) The ventricles,—hemorrhage into these cavities, for example, may sometimes be distinguished from effusion into the substance of the hemisphere; (3.) The base of the brain; or (4.) Its superior surface. Inflammation of the brain-substance and of the meninges presents different symptoms when occurring in the two last-mentioned situations.

In the present state of science we are unable to localize exactly either the diseases of the *cerebellum* or of the *central ganglia* of the brain from symptoms during life.

Diseases of the *pia mater* and *arachnoid* may be discovered by the history and progress of a case from those of the *dura mater*; and sometimes it is possible to distinguish *meningitis* of the base from that of the convexity of the brain, chiefly from the influence which the diseased *meninges* exercise upon the functions of the parts beneath. Disease of the *dura mater*, for example, may often also be inferred from morbid conditions discoverable in the organs of *special sense*, or from disease in the *bones* of the *cranium*, or of the *integuments* or *scalp*.

With regard to the diseases of the spinal cord, similar grounds for diagnosis exist; and the locality of the lesions may often be correctly referred to the *anatomical regions* of the cord, to *certain columns* of its *substance*, to the *white* or *gray nerve-substance* of which it is composed, or to the *coverings*. It is of importance in cases of diseases of the nervous trunks to know which set of the cranial nerves are affected; and in relation to the spinal nerves it is necessary to distinguish diseases of the *anterior* from those of the *posterior* roots, as well as the region of the cord from which the diseased roots proceed.

The grounds of diagnosis of the locality of nervous diseases generally may be summed up as follows:

I. The *brain* is presumed to be the seat of lesion when several of the special senses are simultaneously affected; when *Perception*, *Ideation*, *Volition*, and *special Sensation*, are affected: when the muscles and general sensory nerves are implicated longitudinally and unilaterally (hemiplegia); when muscles situated so high as those of the face and tongue are involved, and the *orbicularis* of the eyelids does not share in their affec-



tion. In these rare cases of bilateral (or transverse) paralysis (paraplegia) resulting from some cerebral change, the symptoms at some period of the case have generally referred to the head (by their special character), so that, by a combination of the two classes of observations, the general diagnosis may almost universally be established.

II. The *spinal cord* is presumed to be the organ affected when the symptoms of motory and sensory character are distributed transversely or bilaterally, inducing paraplegia or transverse spasms; when the mental functions are unchanged. The precise locality may be estimated sometimes from anatomy of the spinal nerves. If the lesion or disease is high, speech, deglutition, or respiration may be impaired. There is often erection of the penis, and the retention or voluntary discharge of fæces or urine.

III. The *nerve-trunks* are presumed to be the seat of lesion when the symptoms are referable to an isolated muscle, or group of muscles, or to a small portion of the sensory surface. When paralysis is the symptom, the irritability of the muscles to electric stimulation is quickly lost, and the symptoms show no disposition to wander from the special localities affected (REYNOLDS).

The distinguishing characters of *meningeal* from *cerebral* diseases may be arranged in the following tabular form, for comparison and reference:

#### CEREBRAL DISEASE.

1. From the outset, or from a very early stage of development, there is loss of some one or more of the proper nervous functions, such as paralysis, anæsthesia, loss of memory.

2. Cerebral disease is not commonly attended by high-marked exaggeration of function, such as furious delirium, convulsions, intense hyperæsthesia, pain, or tenderness.

3. Little vascular excitement attends cerebral disease, nor is there frequently any highly-marked general disturbance.

4. Paralysis and Anæsthesia, losses of Volition, Ideation, Perception, and the like, characterize cerebral disease.

#### MENINGEAL DISEASE.

1. It is not till some time after the detection of signs of disease that diminution or loss of nervous function takes place.

2. The subsequent diminution or loss of nervous function which succeeds the prolonged existence of "head symptoms" is generally preceded, in cases of meningeal disease, by extremely severe excitement or exaggeration of functions, such as pain, tenderness, furious delirium, or convulsions.

3. In meningeal affections there is usually much local vascular excitement, with general disturbance.

4. Spasms, convulsions, pain, and delirium, are the general features of meningeal disease.

In diagnosing the locality of diseases of the brain generally, it is necessary to distinguish, in the first instance, the *intrinsic diseases* of the nervous system, which properly constitute the order *cephalici*; also local diseases from nervous complications of other diseases not of a local kind. It is necessary also to distinguish affections of the

*brain, spinal cord, and nerves*, as much as possible from each other; and, lastly, to separate diseases of the *meninges* from *cerebral lesions*.

It is chiefly by the history of the case that nervous symptoms peculiar to the *Specific* or *Constitutional* class of diseases are to be distinguished. It is also generally worthy of notice that symptoms referable to altered nervous functions are the earliest indications of intrinsic or local diseases of the organs of the nervous system; and that when general disease exists of a *Specific* or *Constitutional* kind, the nervous symptoms are secondary in relation to the time of their appearance, compared with the earliest manifestation of symptoms of ill-health.

The diagnostic value of *vomiting*, as a symptom of cerebral disease, is one which must be thoroughly appreciated. Regarding this symptom, Dr. Reynolds makes the following remarks: "The intimate sympathy" subsisting between the stomach and the head is a matter of daily observation. Headache from gastric disturbance is as common as vomiting from cerebral derangement. In children especially, the existence of obstinate vomiting is indicative of head rather than of stomach disease. A consideration of the following points (for comparison tabulated) may lead to the discrimination of the pathological significance of this symptom:

#### GASTRIC OR HEPATIC VOMITING.

1. There is nausea, which is relieved, at all events temporarily, by the discharge.

2. The tongue is foul, the conjunctivæ often yellowish, and the headache secondary in respect of time.

3. Gripping pain in the abdomen, diarrhoea, and disordered evacuations frequently attend the *gastric* or *hepatic vomiting*.

4. Retching and increased salivation attend *gastric* or *hepatic vomiting*.

#### CEREBRAL VOMITING.

1. Little or no nausea, and the vomiting continues, in spite of the complete discharge of its contents by the stomach, so soon as anything (liquid or solid) is introduced.

2. The tongue may be clean, the conjunctivæ colorless or injected, and the headache primary.

3. Obstinate constipation generally attends *cerebral vomiting*.

4. In *cerebral vomiting* the stomach is emptied almost without effort, and without any increase of the salivary secretion.

Thus, while vomiting may depend upon derangement in the gastro-intestinal canal, it may also depend upon increased sensory or reflex action, and is thus a valuable indication of cerebral disease.

2. **As to the Nature of the Affection.**—The nature of the intrinsic diseases of this order may be shortly stated to be,—

I. *Acute*, but *non-febrile*, to distinguish them from the nervous symptoms which attend the febrile state of many of the diseases already noticed, as peculiar to the *Specific* and *Constitutional* classes. They are of such a kind as are marked by,—(1.) Diminution or loss of functional activity (apoplectic and paralytic diseases); (2.) In-

crease or excess of action, such as of sensibility (neuralgia), of mobility (convulsions, spasms), ideation (delirium).

II. *Chronic* diseases, the character of chronicity depending not only on the time such diseases last, but also on the severity of their course.

Such chronic diseases are marked by,—1. Excessive functional activity, as by *neuralgia*, *hallucination*, *chorea*, *hypochondriasis*. 2. Diminution or loss of functional activity,—for example, *anæsthesia*, *paralysis*, *dementia*, *epilepsy*. 3. Combinations of these conditions, such as,—(1.) Loss of mobility, with increased sensibility, as in *paralysis with pain*. (2.) Loss of mental, with increased motor activity, as in *coma* with spasms. (3.) Loss of sensibility, with increased mobility, as in *anæsthesia* with *reflex spasms*.

It is of great importance to recognize, as Dr. Brown-Séquard points out, the necessity of distinguishing between the symptoms of—(1.) Loss of function; and (2.) Irritation.

Symptoms of irritation may be arranged in two groups, according as the irritation acts—(a.) In the central parts, as in convalescence from such serious illness as typhoid fever—overtaxing the mental powers, as in the case of weak children; or, (b.) In the peripheral parts of nerve-fibres, as in the neuralgic headache of dyspeptic children during the second dentition, when the symptoms frequently resemble those of the first stage of *tubercular meningitis*. Convulsions are not rarely due to the irritation of *ascarides*; and they also occur concomitantly with the second dentition, just as they occur in the first.

The most frequent cause of local paralysis or loss of function is an irritation in certain parts of the nervous centres, or in the trunk or periphery of nerves. Such circumstances may produce paralysis in very different parts in different cases, according to the particular fibres on which the irritation has acted. Thus there are now numerous cases, capable of clinical recognition, where paralysis of the upper and lower limbs, and of the face, as well as of contractions and rigidity, which are traceable to the influence of reflex action.

3. **As to the Anatomical Condition.**—Although some of the diseases of this order are marked by excessive severity of symptoms during life, such as *tetanus*, *epilepsy*, *chorea*, *hysteria*, *neuralgia*, and the like, yet no characteristic or constant structural change can be detected in the nervous centres after death, either as a consequence or as a cause of such diseases; and although the belief is daily extending, that no morbid conditions of function can exist without some correspondent change in the organs, yet, so long as we have no means of appreciating such changes, the diseases now noticed must be regarded as “neuroses,” “dynamic,” or “functional” diseases. But there are many other diseases of this order which are attended by some physical changes in the organ, expressed by undoubted symptoms during life, and which leave evidence of their existence after death. For example, very different apparent vascularity is discoverable after death in the nervous masses and texture; and there are two very common classes of nervous symptoms

during life which evidently depend upon the variable amount of blood in the brain. These symptoms are referable to *active arterial hyperæmia*, and are distinguished by the well-known phrase of “determination of blood to the head;” or they are referable to *passive venous hyperæmia*, commonly called “congestion.” The question has been much discussed and experimented on as to whether more than a fixed proportion of blood can find its way into the brain; and there can be no doubt that all considerations of the subject lead to the conclusion that the quantity of blood within the cranium is extremely variable at different times and under different circumstances; and (as clearly stated by Dr. Sieveking) there is a peculiar property belonging to the white matter of the brain which has a strong bearing on the question, namely: the great elasticity of the medullary tissue, so much so that the resiliency afforded by this property is a sufficient counterpoise to the rigid structures which envelop the brain, and which do not, as is erroneously supposed by some, remove the intracranial contents entirely from the influence of atmospheric pressure. That pressure is exerted on a large surface, composed of columns or tubes of blood in innumerable small curved vessels which maintain, through the scalp and diploe of the skull, a direct communication with the blood within the cranium; and which is thus directly influenced by atmospheric pressure, while every anatomical arrangement of the parts within the cavity of the skull illustrates provisions made to counterbalance the varying interchange of bulk between the solid and fluid contents of that cavity. Among these may be noticed the ventricular and subarachnoid spaces, with their varying amount of contained serosity, as furnishing most prominent evidence of provision to accommodate the varying amount of fluids within the cranium.

Morbid states of the brain are also due to a poisoned state of the blood; although such a condition cannot be proved in all cases,—such, for example, as occurs in many of the zymotic and constitutional diseases already noticed (“Typhus,” “Variola,” “Rheumatism,” “Alcoholism,” “Narcotism,” “Blood Poisoning by Urea”).

**Urea in the Blood and Brain.**—It is often of importance to determine whether urea circulating in the blood is contaminating the brain and impairing its functions.

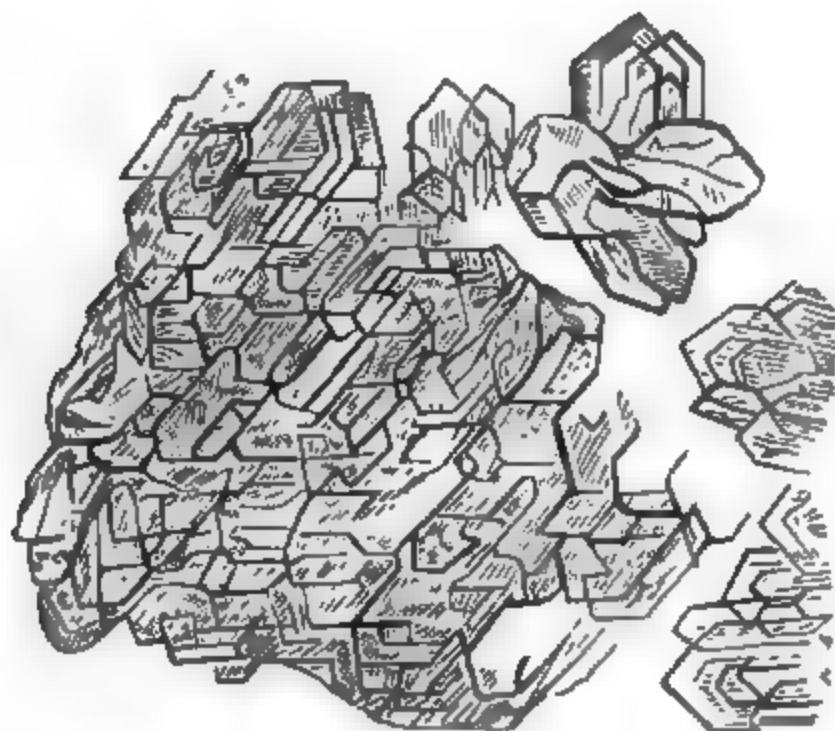
The following instructions are given for the detection of urea in the brain after death, as well as for its discovery in the blood during life:

“1. *In the Serum.*—Take the serum from a good-sized blister, and evaporate it to dryness over a water-bath. The residue is to be extracted with alcohol, which is a ready solvent of urea. This alcoholic extract is then to be evaporated to dryness, and a little water added, so as to make a syrupy mass, which should be plunged into a freezing mixture, and a few drops of pure nitric acid added to it. If urea be present, the characteristic crystals of nitrate of urea are soon found in the solution, and may be recognized either by the naked eye or by the microscope (Fig. 4).

“2. *In the Substance of the Brain.*—Take about three-fourths of a whole brain, and cut it up into small pieces. Then treat it with four

successive portions of boiling distilled water, each portion, consisting of about ten ounces, being allowed to stand six or eight hours before the next is added. The brain while thus macerating should be frequently stirred and mashed about with a glass rod. The washings, after being poured off, are to be mixed together and filtered. The filtered aqueous

FIG. 4.\*



extract so obtained must be evaporated to dryness over a water-bath, and the dry residue, after being powdered, is to be again treated with four successive portions of boiling distilled water, observing the same precautions as before. The washings, after being mixed together as before, are to be filtered, and the clear solution evaporated to dryness over a water-bath; and after being thoroughly dried in a hot water oven, the residue obtained in this manner should be finely powdered, and the powder boiled in five successive portions of ether. The ethereal extract so obtained should be evaporated to dryness at a low temperature, and then treated with a little tepid water, and allowed to get quite cold. It is then to be filtered through paper previously moistened with water, and the clear solution again evaporated to dryness at a low temperature, when a small quantity of the extract procured in this way (which would contain all the urea present in the brain operated upon) is to be placed on a glass slide, treated with a drop of strong nitric acid, covered with a bit of thin glass, and allowed to stand a little time, and then examined under the microscope. A few crystals will then be seen, having all the characters of those of nitrate of urea" (*Clinical Lectures*, by Dr. Todd, 1859).

**The Morbid Textural Changes** of the brain consist chiefly of inflammation and its *productive* consequences, or in softening, degeneration, or atrophy of the nerve-substance; and in heterologous products, such as result from *tuberculous* or *carcinomatous* infiltration. The nervous centres are also known to waste, harden, and lose

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\* Nitrate of Urea (after BEALE). No. II. Urinary Deposits, Plate III.



weight as age advances, and the nerves participate in these changes. Hardening seems less constant than wasting and loss of weight; but it is a change very generally observed in old people; and its characters are thus described by Dr. Maclachlan: "The cortical portion is thinner and darker than in adults, and the medullary portion is often void of its glistening white appearance, which gives place to a dull white or gray; and frequently to a pale drab color in old age." The membranes of the brain are also thicker, more opaque, and more resisting in the aged. This is especially the case with the arachnoid, and is associated with enlargement of the Pacchionian glands or villi. The condensed dura mater acquires immense strength, and adheres firmly to the calvarium, especially along the margins of the longitudinal sinus.

When the brain shrinks, as in atrophy, there is a marked increase in the cephalo-rachidian fluid, according to the degree of atrophy. Instead of two or three ounces, there may be ten or twelve ounces; and the subarachnoid areolar tissue is then also frequently infiltrated with serum (MACLACHLAN). The ventricles contain more than the usual quantity of serosity, and the lateral ventricles especially are filled with limpid fluid. Flattening of the surface of the convolutions becomes apparent, while the sulci are widened and diminished in depth.

It is of importance to study every disease of the nervous system as presenting—(1.) Disease or changes in tissues; (2.) Damage of organs generally; (3.) Disorder of function (H. JACKSON).

[For the *Physical Diagnosis of Diseases of the Cerebro-spinal System*, see Article by the Editor in the Appendix to this volume.]

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## CHAPTER IV.

### MEANINGS OF SOME WORDS IN COMMON USE IN DESCRIBING BRAIN DISEASES.

*Convulsion*.—The occurrence of universal involuntary muscular contraction, generally of paroxysmal or temporary duration.

*Spasm*.—Involuntary convulsive actions of less extent. Of these there are several varieties:

(a.) *Clonic Spasm*.—Consists in rapidly alternating contraction and relaxation, as in *subsultus tendinum*.

(b.) *Tonic Spasms* or *Spastic Contractions*.—Consist in contractions having a certain duration, attended with rigidity or hardness of the muscles, as in *common cramps* and *tetanus*.

*Epileptoid* or *Epileptiform Attacks*.—Imply a sudden loss of Perception and voluntary power, with more or less generally distributed spasmodic movement. The movements are quasitonic at first, then clonic, and appear to impede the respiratory process. The attack

lasts from two to twenty minutes, followed by some exhaustion and sleep (REYNOLDS).

*Coma*—Denotes the loss of Perception and Volition; in other words, the loss of Consciousness, with the appearance of profound sleep, from which the patient may be partially roused.

## CHAPTER V.

### DETAILED DESCRIPTION OF DISEASES OF THE NERVOUS SYSTEM.

#### SECTION I.—CEPHALIC DISEASES, ATTENDED WITH A FEBRILE STATE.

##### MENINGITIS.

LATIN, *Meningitis*; FRENCH, *Méningite*; GERMAN, *Gehirnhautentzündung*—Syn., *Meningitis*; ITALIAN, *Meningitide*.

**Definition.**—A complex morbid state of the immediate coverings of the brain, attended with more or less vascularity of the membranes, opacity of the arachnoid, and the formation of adventitious products between the arachnoid and the pia mater, and the effusion of serum or of pus in the same situation. Acute pain in the head, from the first, accompanies the development of these lesions, attended with intolerance of light and sound; watchfulness, delirium, flushed countenance, and redness of the conjunctivæ, or a heavy, suffused state of the eyes; quick pulse, frequent spasmodic twitchings or convulsions, passing into somnolency, coma, and complete relaxation of the limbs (COPLAND).

**Pathology.**—This morbid state is properly known as “*Meningitis*.” It may be simple and idiopathic, or it may supervene as the result of some Specific or Constitutional disease, such as of some of the fevers, or of the syphilitic, rheumatic, gouty, rachitic, or scrofulous cachexia. Three several forms (in addition to the general involvement of all) may be distinguished.

(1.) Inflammation of the *dura mater*. This is almost invariably the result of injury or disease of the bones of the skull; and in such cases the injury or disease by which it is caused may be readily distinguished.

(2.) Inflammation of the *pia mater* and *arachnoid*. In diffuse *arachnitis* the arachnoid has seldom any considerable redness or congestion, but is thickened and opaque; while the transparent serum naturally contained in the cavity of the arachnoid being now scanty, or wholly wanting, it has neither that polish nor that moisture which is natural to it in health, so that it appears brown and dry. The principal phenomena of *arachnitis* are most obvious in the *pia mater*, so that the large vessels of that membrane are greatly congested; but still, according to Dr. Baillie, the redness is not so general nor so continuous as in inflammation of other serous

membranes. Also, if the *pia mater* be attempted to be removed, it is easily torn, and separates from the brain in small fragments. The arachnoid covering the *dura mater* seldom participates in this affection. These are the appearances observed in diffuse *arachnitis*, supposing it to terminate by resolution. The inflammation, however, often proceeds, and may terminate by effusion of serum, lymph, or pus.

When serum is effused into the arachnoid cavity, the opacity of the arachnoid gives the serum a gelatiniform appearance; but when that membrane is divided, the serum is found to be fluid, and to diffuse itself in every direction; sometimes, however, it may be turbid, from an admixture of a small portion of free albumen. It is also not unusual to find a few points of lymph, of pus, or of blood, either at the exterior surface or within the arachnoid, effused along with the serum, and almost in juxtaposition with each other. The quantity of fluid effused is variable,—from two to three drachms to as many ounces. The effusion most commonly takes place at the upper surface of the hemisphere, but sometimes at the base, and sometimes into the ventricle of the brain. Lymph is the form which the exudation usually assumes ultimately if life continues, varying in density and thickness, dipping down between the convolutions; and commonly it is most abundant on the upper portions of the hemispheres. Foville says he has met with cases in which the effused lymph covered the whole of the brain, or nearly so, as far as the *tentorium*. The lymph was deposited in the arachnoid sac in two layers—one adherent to the cranial arachnoid, and the other to the cerebral arachnoid; while between them was a stratum of serum. He mentions having had six cases of this description under his care for several years, and that they were all in a state of the dullest stupidity, and apparently laboring under paralysis of every sense. They were like statues, with this difference, that placed upright, they preserved their balance; if pushed, they walked; and if food was placed in their mouths, they swallowed it (Art. “*Méningite*,” p. 406, *Dict. de Médecine*). Lymph also may be effused into the arachnoid cavity, but it is generally in small quantity.

Suppurative inflammation may take place either into the sub-arachnoid spaces or into the arachnoid sac. It is, however, by no means a frequent occurrence. Rostan gives several cases of effusion of pus into the arachnoid cavity, likewise Morgagni, Cruveilhier, and Dr. Bright. Dr. Baillie states that he once saw pus effused into the cavity to such an amount as to cover the entire upper surface of the brain. Two cases are given by Dr. Hodgkin of cut wounds of the head in which pus was found in the arachnoid sac. Dr. Sieveking relates a remarkable instance of purulent exudation of the arachnoid, occurring in a young woman aged twenty-seven, under his care, at St. Mary's Hospital, in whom sudden and unexpected coma supervened, and terminated, after thirty-six hours, in death. She had previously suffered from *otorrhæa*; but on her admission she gave no signs of cephalic disease; nor was any direct connection traced after death between the affection of the ear and the *meningitis*.

The characters of *chronic arachnitis* are—a similar opacity and thickening of the membranes, together with granulations of a pearly color, and more especially along the longitudinal sinus, and also an augmentation of the *glandulæ Pacchioni*. Much serum is effused into the cavity; and the connective tissue by which the pia mater is attached to the brain acquires considerable strength, so that portions of the brain come away with the membranes. The membranes acquire considerable tenacity; and so marked are their characters in some cases, that Dr. Maclachlan notices an instance in which a portion of the arachnoid nearly an inch square was fully an inch thick, in appearance like the boiled white of an egg, and equal to the peritoneum in toughness. The surface of the brain is pale, and sometimes slightly atrophied. Much serum abounds in the arachnoid cavity, in proportion generally to the atrophy of the brain; and the ventricles contain serum (MACLACHLAN). Ossification of the pia mater is extremely rare; but it also becomes thickened, opaque, and injected. Dr. Baillie, however, mentions one case of ossified pia mater, on the authority of Soëmmering; and Dr. Hodgkin speaks of a specimen in the museum of Guy's Hospital.

In *acute arachnitis of the ventricles* the membrane becomes thickened, semi-transparent, pulpy, and sometimes sprinkled with minute spots of blood. It is rare to find lymph effused, but occasionally old adhesions are seen between the opposite surfaces of the ventricle. Pus has also been occasionally found in these cavities. With regard to the effusion occurring in the ventricles in "*meningitis*," it is correctly remarked by Dr. Sieveking that "it offers many relations different from the arachnoidal effusions occurring on the surface of the brain; nor is it quite intelligible why the secretion into the cavity of the ventricles should so rarely be found to communicate with the superficial arachnoidal space." The anatomical relation of these parts may in some measure explain the differences; for it is very doubtful that the arachnoid lines the ventricles of the brain, an arrangement which Kölliker considers impossible. Nevertheless, there is in meningitis generally an increase in the ordinary amount of the fluid in the ventricles, to the extent of several drachms, which may perhaps be regarded rather as a result of contiguous sympathetic effusion than as an extension by continuity of the inflammatory exudation.

A microscopic examination of the smaller vessels of the pia mater in *meningitis* shows that they are studded with the exudation-corpuscles described by Gluge and Bennett, and with oily-like vesicles; and these appearances are found both within and without the vessels (SIEVEKING).

(3.) *Tubercular meningitis*—Syn., *Acute hydrocephalus*. The *meningitis* which accompanies the cachexia associated with *tuberculosis* is of a very distinctive kind, and frequently terminates the lives of tuberculous children. Its essential morbid character consists in the growth of tubercle on the arachnoid, generally in the shape of small miliary granules, resembling in appearance the Pacchionian bodies, but differing from them in minute structure and in position. They are found most frequently and most abundantly within the fissure

of Sylvius, between and upon the convolutions of the brain, and at the base of the brain, and very rarely on the cerebellum. They are generally of the nature of the gray granulations embedded in the vascular network of the pia mater. Meningitis is also apt to occur as the result of *gout* or of *rheumatism*, constituting *gouty* or *rheumatic meningitis* (MACLACHLAN); and it is an occasional if not a frequent consequence of Bright's disease (CHAMBERS, GOODFELLOW, MACLACHLAN).

**Causes.**—Every age is liable to meningitis. Children are often attacked by it whilst teething, under the form described as *hydrocephalus acutus*, and also when laboring under *scarlatina*, *measles*, or other disease caused by a specific poison. Adult age, as well as the middle periods of life, is still more liable to this affection, both from the greater exposure to the action of the typhus, typhoid, and paludal poisons, to syphilis and to mechanical injuries, as well as to the greater intemperance and greater excitement incident to this age. In old people meningitis is likewise common; and two forms of the disease are met with in the aged,—namely, the *acute* and the *chronic*, both of which present great varieties in their character and progress (MACLACHLAN), and both sexes perhaps suffer in nearly equal proportions.

*Meningitis*, however, is a disease which most commonly occurs from the action of some specific morbid poison; and there are few agents of that class which are not apt to act on the membranes of the brain. There are many instances also of persons suffering from *arachnitis* after exposure to the heat of the sun, or to what is in common language called the "*coup de soleil*," and which will be considered under the head of *insolatio*. Intemperance, as well as great mental work and anxiety, is also a frequent cause of the chronic forms of the disease; but the free use of alcoholic liquors is perhaps the most common predisposing cause; while fits of intemperance occasionally are the direct agents which induce the disease. It is especially apt to be associated with or to follow *delirium tremens* in the aged, and likewise *gout* or *rheumatism*. It is also especially connected with *insanity*, and with every structural disease of the brain; and to these causes must be added mechanical injuries, diseases of the bones, and morbid growths, especially in cases of primary implication of the *dura mater*.

**Symptoms and Diagnosis.**—*Meningitis* has usually been described in three stages. The symptoms of the first stage are those of excitement, resulting from diffuse inflammation. The symptoms of the second stage are those of compression, marking that effusion has taken place; while those of the third stage are associated with progressive recovery, or with the modes in which death may occur.

But there are symptoms characteristic of several forms of this disease, which may be classed as follows:

(a.) *Simple Meningitis*.—The type of this morbid condition is that in which the membranes covering the convexity of the hemispheres are parts generally affected. The most important facts to be ascertained in the previous history of the case, before the development of cephalic symptoms, relate to the general health, and especially



to any signs of cachexia or diathetic states, such as *tuberculosis*, *rheumatism*, *gout*, or of the specific action of implanted poison, such as *syphilis*. It is important also to ascertain whether any blow on the head has been sustained, or if the patient has been much exposed to the sun; whether any disease of the ear or nose exists; whether application to study has been intense, or to the cares of business.

Premonitory symptoms may be trifling, or absent altogether. The most common are slight but increasing pains of the head, sensorial disturbance, irritability of temper, or restlessness, with some general *malaise*. Rigors quickly supervene, or simple chilliness, with *cutis anserina* and pallor of the surface, quickly followed by febrile reaction. An attack of convulsions may supplant the rigors, especially in children. Such, however, are by no means common in the adult, and are not necessarily indicative of any severe or advanced lesion. The fever is commonly high; the pulse sharp, hard, and frequent; the respirations are irregular, performed with a sigh, and often with a moan. The skin is hot; the bowels obstinately constipated; and evacuations, when they occur, are dark and offensive. In this stage there is little or no prostration of strength. The headache of fever is supplanted by acute and intense pain; the face flushes and turns pale alternately; the eyeballs stare, and the conjunctivæ become injected.

The purely nervous symptoms, expressed by *mental*, *sensorial*, and *motorial* phenomena, are thus classified by Dr. Reynolds:

1. *Mental*.—The temper is extremely irritable. There is marked somnolence, or wakefulness, and the two sometimes alternate for several days. The most marked feature is delirium, commencing early, and of a furious character, the patient screaming and gesticulating in the wildest manner; the expression of countenance is savage and malignant, or sometimes has the fierce aspect of the brute.

2. *Sensorial*.—Marked and continuous headache (*cephalalgia*) prevails, with exacerbations of a darting, violent character, eliciting from the patient, and especially from children, a sharp, piercing cry. Pain is increased by movement, and the patient holds the head with the hands; or, if a child, frequently carries them to the head. The headache is increased by sensorial impressions, and hence the eyes are obstinately closed, and the ears, if possible, kept covered with the bedclothes. Double vision, *tinnitus aurium*, *formicatio*, and subjective sensations of various kinds, are present. The sensorial disturbances are highly marked.

3. *Motorial*.—Restlessness is incessant, sometimes general or partial. The muscles of the face and limbs twitch involuntarily. There is strabismus, or the eyeball is unsteady, and with a contracted or oscillating pupil. Vomiting is frequent, without epigastric pain or tenderness, and often without nausea.

This stage generally lasts from *one* to *four* days, and its characteristics may be shortly expressed as consisting of—the combination of *great nervous hyperaction*, with *marked fever*, a *peculiar cry*, *cephalalgia*, *vomiting*, and *constipation*.

*The second stage* is one of a transition from the first to the third. The fever diminishes. The pulse sinks in frequency and force, becoming variable in frequency between very wide limits, and in very short intervals of time. Respiration becomes peculiarly irregular. The bowels continue constipated. The tongue becomes furred and dry. The heat of the head persists, but the body generally is cool.

The nervous phenomena present remarkable intermissions during the further progress of the affection, especially in the following points:

1. *Mental*.—Delirium becomes quieter, or passes into coma; or the patient may appear collected and well.

2. *Sensorial*.—Excitement diminishes and disappears, and drowsiness is the most common feature.

3. *Motorial*.—Muscular twitchings generally are increased on both sides of the body. Convulsions are common in the child, and spasms often alternate with paralysis. A violent general convulsion may throw the patient at once into—

*The third stage*.—It may come on almost immediately, or a week of transition symptoms may intervene. The face becomes sunken, the extremities cold, and the abdomen retracted. *Sordes* form on the gums and teeth. The pulse flutters, becomes thready, feeble, and uncountable. Great prostration of strength supervenes.

The nervous functions are gradually suspended.

1. *Mental*.—Perception, Volition, and Ideation become lost, so far as can be ascertained by corporeal signs.

2. *Sensorial*.—Anæsthesia is complete.

3. *Motorial*.—There is absolute paralysis to almost every form of stimulus, observed first in the eyelids and eyeballs, and then in the limbs. Muscular relaxation becomes complete, as evidenced by the dilated pupil, stertorous breathing, involuntary micturition and defecation.

Generally there is absence of nervous action, and organic life gradually dies out.

The duration of these stages is various. For the most part each lasts a week; but one or more stages may be wanting.

The tongue in the first stage is white; in the second it becomes brown; in the third it again cleans, if the patient does not die. The pulse, likewise, in the first stage is from 90 to 100; in the second from 110 to 130; and in the last stage it either gradually returns to its natural standard, or runs on too rapidly and too feebly to be counted.

The symptoms which have been described are those which especially mark *arachnitis* at the superior portions of the brain. When, however, it occurs at the base, or in the ventricles, some differences are observable, although the condition cannot always be distinguished; but in some cases such a location of the malady is rendered probable if the Intellect is less impaired, the Passions more excited, and the patient lies fretful, impatient, morose, and, although somnolent, he occasionally cries out and grinds his

teeth very early in the disease ; while the parallelism of the axis of the eye is frequently affected at an early period.

(b.) *Tubercular Meningitis in the Child*.—It is an object to ascertain the existence of the scrofulous cachexia in the first instance, as described at page 229, *ante, et seq.* If such exists, *tubercular meningitis* may supervene on the occurrence of any febrile disturbance, with slight thirst and anorexia ; irregular and somewhat quick pulse ; vomiting and constipation ; clayey evacuations deficient in bile ; red and moist tongue ; dry and hot skin, and other phenomena of general derangement. The temperature, as measured by the thermometer, will be found to be persistently above the normal amount (RINGER).

The special nervous phenomena are often feebly marked.

1. *Mental*.—There may be irritability of temper and peevishness, with some slight delirium at night, rarely commencing early in the disease, disturbed sleep and restless manner.

2. *Sensorial*.—Pain in the head prevails, with intolerance of light and sound. Vertigo is also indicated by staggering, or clinging to objects for support.

3. *Motorial*.—Grinding of the teeth prevails, occasional vomiting, unsteady, restless movements, and dragging of the limbs. After three or four days of these premonitory symptoms,

*The second stage* commences, with heat of head and flushings of the face, alternating with pallor. The pulse is irregular, and commonly, when the child is still, it is of little frequency, but rises rapidly if the child is disturbed. The vomiting ceases, but the constipation persists, with retracted abdomen.

The following special nervous phenomena become more marked :

1. *Mental*.—The child inclines to lie quiet, and resists being moved, as if pain was increased by motion. There is delirium, which is sometimes fugitive and sometimes persistent.

2. *Sensorial*.—The cephalalgia increases. The expression of countenance bespeaks great suffering, and the face looks aged. A peculiar piercing cry is now and again given by the child. The eyes are closed, and there is a tendency to drowsiness.

3. *Motorial*.—Strabismus and muscular twitchings occur. The pupils are variable, and often oscillate, and the eyeballs are unsteady.

*The third stage* of tubercular meningitis is ushered in by the general symptoms of approaching dissolution, such as coldness of the extremities, clammy perspiration, and an excessively rapid but feeble pulse.

The special nervous phenomena are first those of exalted spinal action, and then those of general prostration.

1. *Mental*.—Drowsiness passes into stupor, with an idiotic expression of face. There is loss of Perception and Volition.

2. *Sensorial*.—All signs of activity give place to anæsthesia, and the eyes are half open.

3. *Motorial*.—Death approaches by convulsions, with partial paralysis, *subsultus tendinum*, clenched hands, retracted head, and automatic movements, giving way to general relaxation.

(c.) *Tubercular Meningitis in the Adult*.—The symptoms occasionally assume an apoplectic, sometimes a convulsive form, at the commencement; and the febrile character is generally imperfectly marked. The premonitory symptoms are those associated with the scrofulous cachexia, and *meningitis* may occur at any stage of the lung disease. The following are its features:

After some remission of chest symptoms, special nervous phenomena referable to the head may supervene.

1. *Mental*.—The patient looks bewildered, with a dull, heavy, expressionless face, often highly characteristic. There also appears to be some *intellectual* incapacity to speak; the patient, seeming to understand what is said or asked, looks at the inquirer for a few seconds, and then turns the head away without a reply. There is often marked somnolence.

2. *Sensorial*.—Pain in the head, fixed to one spot (generally the forehead), is the most striking symptom, of considerable intensity, and is persistent for many days.

3. *Motorial*.—An attack of convulsions may precede every other symptom, but paralysis is rare.

*The second stage* is of very variable duration, the pulse is highly irregular, while alternate flushings and pallor of the countenance are common, and all the symptoms already noticed become more intense, mild delirium prevails, and the face becomes increasingly stupid-looking.

Paralysis of volition alternates or coexists with clonic or tonic spasms, strabismus, or prolonged convulsive attacks.

*The third stage* of tubercular meningitis in the adult is marked by increasing stupor, immobility, and involuntary defecation and micturition.

The general characteristics of *tubercular meningitis* thus consist in the occurrence of fixed pain, vomiting, dulness of Intellect, and duskiness of the face, with partial paralysis or convulsions, slight fever, and diminution of the chest symptoms in a patient demonstrably tuberculous. The indications of inflammatory action are only feebly marked, not only during life, but after death; and very often the febrile state which had existed before becomes less noticeable at the onset of cerebral symptoms. Still, heat of head, injected conjunctivæ, and flush of face denote a condition of vascular excitement; while persistent headache with febrile symptoms in a patient otherwise phthisical, when it can be traced to no other known cause, is strong presumptive evidence of impending *tuberculous meningitis* (HILL). The aid of the thermometer should invariably be sought in diagnosis (see p. 64, vol. i). But there are also cases in which the disease of the brain is marked by the absence of ordinary symptoms. The premonitory phenomena already noticed, for example, may be absent, especially in children; and then the steady persistence of any one symptom becomes of great importance in diagnosis—for example, sickness, headache, constipation, drowsiness, heat of head (WEST).

(d.) *Acute Meningitis of the Aged*.—Dr. Maclachlan gives the best account of this form, and the phenomena he describes may be classi-

fied as the previously described forms have been. It seldom occurs suddenly, nor with the intense symptoms which prevail in the meningitis of early manhood. It commences insidiously, without rigors, and very frequently some days elapse before it can be recognized, even by persons familiar with the diseases of old age. The pulse is natural, the tongue remains clean, the bowels regular, and there is little or no vascular excitement, local or general, but the symptoms at the outset are purely of a nervous character.

1. *Mental*.—The temper is peevish or irritable, with more or less confusion of thought, inattention, and forgetfulness. “If infirm, and already an inmate of an hospital, the patient commits strange mistakes, takes possession of another’s bed, uses the spittoon instead of the chamber-pot, and is frequently found lying outside the clothes, or with his feet where his head ought to be. When addressed, his answers are rational, but still there is a peculiarity in his manner and expression of countenance, an apparent slowness of comprehension, and a vacancy of the eye, that warns the Physician of the approach of some cerebral disease” (*The Diseases and Infirmities of Advanced Life*, p. 85).

2. *Sensorial*.—The appetite is slightly if at all impaired; and the skin remains of normal temperature.

3. *Motorial*.—Restlessness prevails, although prostration is apparent. In walking the gait is unsteady, and in lifting anything to the mouth the hand trembles. In a few rare cases the disease commences with convulsions.

While such symptoms are being developed, after twelve, forty-eight, or sixty-two hours, but generally within twenty-four hours, more or less febrile reaction is expressed by slight increase of temperature merely, and without any marked redness of the face or acceleration of the pulse. The nervous symptoms are now of the following kind:

1. *Mental*.—Wandering, low muttering delirium and incessant talking become frequent and characteristic symptoms. Maniacal excitement is uncommon. For a day or two the patient may answer questions rationally, though slowly and hesitatingly, when roused from the reverie in which he is generally wrapped. At a still more advanced stage there is coma.

2. *Sensorial*.—Headache is not a prominent symptom. It is so slight or unfrequent that unless the patient is pointedly asked the question, there is never any allusion either to it or to giddiness, or to *tinnitus aurium*. This absence of headache is pointedly noticed by Dr. MacLachlan as a remarkable character; for even in the most acute pus-forming or false-membrane-forming meningitis, headache may be entirely absent from the beginning. The eyes are suffused, the pupils either slightly contracted or natural. Knitting of the eyebrows, intolerance of light, acuteness of hearing, and vomiting,—common and characteristic phenomena of the disease in early life,—are comparatively rare in the aged. The scalp may be hotter than natural, and, combined with the suffusion and injection of the eyes, is the only physical indication present of vascular excitement within the cranium. The feet are frequently cold, and the surface



generally moderately warm. There is usually great thirst, the patient drinks greedily, but seldom asks for liquid. He is apt to refuse food, or takes but a small quantity at a time, when it is presented to him.

3. *Motorial*.—Nervous twitchings and convulsions are observed in the worst cases; and if the patient be raised in bed, these symptoms are frequently induced when otherwise absent, and the head trembles, as well as the upper extremities. When coma prevails, there may be sometimes slight convulsions of the limbs.

The general features of the disease resemble typhus fever. The tongue becomes very dry, and generally brown in the centre. The bowels cease to act without medicine, but are not obstinately confined. Towards evening there is generally increased febrile disturbance, denoted by flushing of the face, where it had been previously pale and dejected; dryness of the skin, greater heat of the scalp, and acceleration of the pulse. In numerous examples while dulness of Intellect and somnolence are the first symptoms observed, in others great and unusual loquacity, with redness of the face and heat of the scalp, ushers in the disease. The progress of the disease is equally inconstant. In not a few cases the symptoms of febrile excitement are wanting; but dulness of Intellect and stupor, with more or less insensibility of the limbs and relaxation of the sphincters, are frequently prominent symptoms throughout the whole course of the disease, as if the medullary substance of the brain were congested or suffered compression from early exudation on its surface. Such examples of an apoplectic character are more frequently met with in old persons addicted to fermented liquors, in whom the cerebral vessels are enlarged from repeated engorgement. Excessive venous congestion of the brain prevails in such cases during life; and the symptoms of meningitis are marked, and apt to be confounded with effusion into the brain, or softening of its texture (MACLACHLAN, *l. c.*, p. 87).

(e.) *Chronic Meningitis of the Aged* is extremely subdued in its symptoms and insidious in its approach. It is not unfrequent as a result of albuminuria, or of repeated attacks of *delirium tremens*; or it follows *gout* or *rheumatism*. The characteristic phenomena may be arranged as follows:

1. *Mental*.—It is almost uniformly accompanied with great impairment of the mental faculties. There is very marked loss of Memory, slowness of comprehension, periodical fits of passion.

2. *Sensorial*.—Vertigo prevails, with ringing in the ears; and occasional attacks of headache occur, with or without vascular excitement.

*Motorial*.—Speech is thick. There is paralytic weakness of the lower limbs, and the gait becomes tottering and feeble. All movements, whether of the upper or the lower limbs, are performed slowly, awkwardly, and with more or less uncertainty.

The energies of the whole system are reduced. The functions of organic life are impaired. The appetite may be good, but digestion is slow, the bowels being inactive, and the various secretions vitiated or diminished. "Sooner or later the aged invalid takes to

bed reluctantly. There he lies uncomplaining, vegetating, the mere wreck of what he formerly was, both in mind and body, gradually sinking, and dying often in consequence of sloughing of the hips and nates" (MACLACHLAN, *l. c.*, p. 93).

**Diagnosis.**—*Meningitis* is distinguished from *encephalitis* by the headache, the early delirium, and by the general absence of hemiplegia. It must be admitted, however, that disease of the brain and of its membranes is often conjoined, so that *meningitis* is not in all cases a simple affection, but is ultimately conjoined with *cerebritis*, the inflammation of the membranes usually extending to the cortical portion of the brain.

From *delirium tremens*, meningitis is mainly distinguished by the character and mode of accession of the delirium, and the tremor, and by the *mental*, *sensorial*, and *motorial* phenomena.

From *typhus fever* it is mainly distinguished by the absence of the characteristic eruption or mulberry rash (see vol. i, p. 396).

**Treatment.**—As a general principle, remedies have little influence over those forms of the disease which arise during the progress of any specific or constitutional disease, independent of the general treatment indicated for that disease. When arachnitis arises from mechanical injuries, the treatment is generally by bleeding, active purgatives, especially by calomel and scammony, and by cold applications to the head. In chronic cases, Foville strongly recommends the cold *douche*, but with caution, as being a powerful depressant, yet producing less ultimate debility than bleeding. It acts, doubtless, by cooling down the general mass of the blood. (See remarks under the treatment of "Hydrocephalus.")

In advanced life it rarely happens that the symptoms are so intense as to demand general bloodletting; but in vigorous constitutions this measure is sometimes necessary. Dr. MacLachlan gives a well-marked instance of its usefulness (*l. c.*, p. 90). The cases which demand it are those attended by high cerebral excitement and vascular action. But as a rule, local bloodletting is not only infinitely more safe but more beneficial, and it can be repeated from time to time, as the nature of the symptoms may indicate; while general bloodletting can very rarely be resorted to more than once, and that only at the commencement of the attack. The beneficial effects of local bloodletting may be greatly aided by keeping the head well raised, and by the constant application of cold water to the scalp; or the occasional use of bladders filled with crushed ice.

The bowels should be opened as rapidly as possible, unless the patient is feeble, emaciated, or greatly exhausted. Four or five grains of the *compound extract of colocynth* should be given, combined with two or three grains of calomel, in cases uncomplicated with *gout* or Bright's disease, and followed in a few hours with a dose of salts and senna. A fair proportion of nutriment must be given, in the form of milk, strong beef-tea, sago, tapioca, or arrowroot; and the patient should be kept in a quiet and darkened room.

The more active symptoms being subdued, but not till then, a blister should be placed on the pape of the neck, if coma should ensue. The bowels should be kept open regularly, and the strength

supported by mild unstimulating nutriment. When nervous irritability continues during convalescence, *henbane*, or *muriate of morphia* may be given (MACLACHLAN, *l. c.*, p. 92).

In the *chronic* form, the periodical attacks of headache or of insanity may generally be relieved by a brisk purge; and the head should be kept cool by cloths dipped in vinegar and cold water. If vascular excitement prevails, leeches may be applied behind the ears. The bladder must be kept empty of urine by the catheter, and although wine must be withheld during the active stage of the disease, it is beneficial when the vital energies begin to fail (MACLACHLAN).

The dietetic treatment should be strictly antiphlogistic, the patient especially avoiding all mental excitement; and, if not secluded, he should at least be kept tranquil, not only in body, but also in mind.

### ENCEPHALITIS.

LATIN, *Encephalitis*; FRENCH, *Encéphalite*; GERMAN, *Gehirnentzündung*—Syn., *Encephalitis*; ITALIAN, *Encefalite*.

**Definition.**—*A morbid state of the brain, more or less complex, according as it is general or local, or as it may or may not be combined with meningitis. The usual evidence of inflammation is shown by symptoms during life, and certain post-mortem appearances after death, such as dusky redness, softening or suppuration of some part of the brain-substance.*

**Pathology and Morbid Anatomy.**—The most general fact that has been determined regarding this morbid state is that there are two very distinct forms of *cerebritis* to be recognized both anatomically and clinically. While dissection demonstrates the fact that inflammation may exist either in the brain alone or in the membranes alone, yet clinically it is found that *meningitis* generally complicates *cerebritis*, and thus the phenomena proper to each of these morbid states become combined; and especially in that form of the disease in which the lesion is extensive, commonly known as “*general cerebritis*,” “*phrenitis*,” or “*encephalitis*,” when the precise seat of the inflammation cannot be determined except by post-mortem examination.

There are, however, more partial or local forms of this affection, to which the name of “*red softening*,” or “*acute ramollissement*,” has been applied; and which Gluge and Bennett, by the aid of microscopic observation after death, have learned us to regard as an inflammatory affection.

There are thus two states to be distinguished, namely,—(1.) “*Inflammation of the brain-substance*,” with or without implication of the membranes, usually partial, and in many cases dependent on local injury or specific deposits or growths; (2.) *Inflammatory red softening*, or *acute ramollissement*, as contradistinguished from “*white softening of the cerebral substance*,” the result of an *atrophic* process and impaired nutrition.

The red *diffuse* inflammation of the substance of the brain appears to have many degrees. In the first degree the substance of the organ, when cut into, exhibits more bloody points than usual, so

that the medullary portion appears as if sprinkled with blood, while the color of the cortical substance is increased in intensity. If the inflammation assumes a higher degree, the most markedly red appearances generally only partially affect one of the convolutions of the brain, or a small portion of a hemisphere; and the inflamed part varies from a bright rose to a deep red dusky color. This increase of color is supposed by many pathologists not to arise from any greater vascularity of the part, but from blood escaping from the vessels and becoming effused or infiltrated into the substance of the brain, forming so many apoplectic spots (BOYER). The inflamed part is generally swollen, and sometimes considerably so, and is generally softer, though it may appear firmer than usual.

As the disease advances the exudation ultimately assumes the purulent character, or becomes more or less softened, approaching in character the condition of pus as it appears to the naked eye. Without suppuration, however, having actually taken place, the mere effect of inflammatory exudation amongst the brain-tissue is to soften its texture and increase its specific gravity, whereas in "white non-inflammatory softening of the brain" the specific gravity is diminished. When the softening results from the exudation merely, before it has undergone any subsequent change, there are traces of congestion, which give to the affected part a red appearance; and the microscope shows, as Bennett first demonstrated, a large amount of exudation-corpuscles interspersed among the broken-down nerve-matter, as well as coating the inner and outer surface of the minute bloodvessels. Wherever exudation-matter exists, although there may be no palpable indication of changed consistence in the tissues, yet the specific gravity will be found increased; just as we find that without microscopic examination we cannot tell whether or not congestion of the brain-substance exists, combined with the exudation-corpuscles of red softening.

The inflammations of the substance of the brain have thus much in them that is peculiar, depending on the nature of the tissue; and, independent of physical appliances (such as the tests for specific gravity and the microscope), the unaided senses cannot enable us to appreciate the exact pathological significance of morbid changes. It is only in a small number of cases that the red color of the brain is characteristic of inflammation; for in by far the greater number there is no increase of redness; and on account of the frequent occurrence of such cases; *ramollissement* has been described by many authors as a distinct idiopathic disease. The liberation of phosphoric acid and of fatty acids seems to be associated with yellow softening; and there is decided acid reaction of the fluid contained in the soft part (ROKITANSKY).

The varieties of softening, as characterized by their color merely, cannot be regarded as essentially different: they are peculiarities due to the nature of the tissue and the effects of the morbid process upon its component parts. It appears to be the *liquor sanguinis*, rather than the *corpuscular* part of the blood, which takes the chief part in cerebral inflammations where softening is the prominent phenomenon. The characteristic of the part thus affected is, that

it is generally whiter or grayer than the natural color of the brain, and also softer than its natural substance: and, accordingly, a rough way to appreciate the presence of this softening is to allow a gentle stream of water to flow upon the suspected part; if softening exists, the softened parts will be gradually washed away. This softening, however, has many degrees, and in some cases can be only accurately determined by a microscopic examination. In its extreme form the softened portion of the brain is absolutely diffluent, so that it can be poured out of the cranium with as much facility as a thickened cream or a thin jelly can be poured from one cup into another. Sometimes nearly a whole hemisphere has been thus destroyed (BRODIE, MACLACHLAN). In this semi-liquid state much serum can often be expressed from it.

Extreme softness of the brain, unattended with inflammatory coloration, may be well seen in those cases where the brain has been extensively injured, as by sabre wounds. Many such cases are related by our army surgeons. In cases of *hernia cerebri*, also, not only does the protruded portion become softened, with red particles of blood intermixed in it, but in acute cases, which terminate fatally in a few days after the injury, "the medullary structure, intervening between the base of the protruded part and the *anterior cornu* of the lateral ventricle, had entirely lost its natural structure, and become soft and pulpy. Around this disorganized mass, and extending across the *corpus callosum* into the medullary substance forming the roof of the opposite ventricle, the brain had undergone a change from its natural color to a grayish-blue white, while it still retained its natural consistency" (STANLEY). It was remarked, also, in this case, during the last three days of life, that a very considerable quantity of fluid constantly oozed from the centre of the protrusion, whence it trickled down the cheek in a continued stream. Such cases show the acute and rapid nature of the softening process as an inflammatory phenomenon, but without any obvious discoloration. In fact, most of the phenomena connected with the inflammatory process in the nervous substance are characterized by the apparent want of increase of the red part of the blood.

*Suppuration.*—In the three forms of suppuration of the brain which are subsequently described, in by far the greater number of cases no trace of redness can be seen in any part of the brain; and probably this want of vascularity may account for the rare development of pus-corpuscles in the fluid of what appears to be abscesses, and which otherwise looks like pus. Pus is not always present in undeniable abscesses of the brain, but molecular granules, exudation-corpuscles, and pyoid bodies, evidently the result of the exudation-process, are generally the sole objects which the microscope can detect (LEBERT, BENNETT). Exudation in the softened state of the brain-substance, resembling pus, may be thus—(1.) Collected into the form of an abscess; (2.) It may be infiltrated into the substance of the brain; or (3.) It may be detected on the convolutions, in the shape of a ragged ulcer, varying in size from a fourpenny-piece, or less, to that of half-a-crown. It is in this latter form and



site of the suppurative process that well-formed pus-corpuscles can most frequently be detected.

*In abscess of the brain* the surrounding substance is generally of the natural color, except in a very few cases in which it succeeds to apoplectic effusion, when the walls of the cavities are dyed by the previously extravasated blood. Dr. Baillie says, when the abscess is of large size, the weight of the pus breaks down the neighboring parts, and they look simply as if they had been destroyed, or very much injured by the pressure; and also when the abscesses are small there is an ulcerated appearance of the cavity in which the pus is contained. In other cases the usual membrane of an abscess forms. This membrane is at first extremely delicate and easily torn; but as the patient continues to live, the membrane lining the abscess becomes of greater consistency, and even of considerable density, so that in some cases it is fibrous, fibro-cartilaginous, and even ossified, thus laying the basis for the formation of bony tumors of the brain.

Inflammatory *ramollissement* of the brain is looked upon as an essentially fatal disease, and the patient seldom survives the formation of an abscess; but it is apprehended that the pus may be occasionally absorbed, and that the opposite walls may unite by granulations, and leave an areolar-tissue cicatrix. Nevertheless, there is some evidence, also, that inflammatory ramollissement is capable of being cured. The post-mortem evidence of this fact is the disappearance of one or more layers of the cortical substance, probably by absorption, while the pia mater adheres to this part of the brain. The evidence of the cure of ramollissement in the gray matter of the *corpora striata* and other central parts is the presence of a number of "holes," resembling Parmesan cheese, of a red color when there has been transudation from the bloodvessels, and of a fawn color in other cases. The part is atrophied and softened; while the holes may be filled with a limpid fluid, sometimes lined with a membrane (Dr. Simms, *Med. and Chir. Trans.*, vol. xix, p. 413).

*Gangrene.*—There is also evidence of the brain-tissue becoming gangrenous, as a result of the inflammatory process, equally free from the red coloration. Mr. Stanley has shown, in his cases of hernia of the brain, that where portions of the brain have *sloughed away*, they have *granulated* again, and have passed into a state of gangrene, often exhaling an exceedingly fetid odor. It is probable, however, that death of the brain-tissue, as a result of idiopathic inflammation in the form of what is ordinarily known as gangrene, does not usually occur. When the softening is truly gangrenous, it is generally after wounds and injuries, and does not differ from gangrene in other parts; the debris is "of a grayish, brownish, or greenish color, sanious and very fetid" (JOHN HUNTER, JOHN BELL, and others).

*White softening* or *atrophic softening*, and due to interruption of the circulation, is the result of imperfect nutrition, owing to deficient supply of blood, and is in most instances dependent on mechanical obstruction or degeneration of the cerebral arteries. There is undoubtedly a softening which results from obliteration of the

cerebral arteries, consequent on coagulation of the blood in them, or on obstruction from clots of fibrin (CARSWELL, HASSE, KIRKES, SANKEY). But Mr. Paget has shown that such softening is more probably the result of a fatty degeneration of the brain-tissue, as a result of the deprivation of its nutrient material.

Thus softening of the cerebral substance, although characterized by some distinctive phenomena during life, cannot be regarded as an affection *sui generis*, apart from inflammation as now understood (vol. i, p. 82), of which it is simply the result.

The two hemispheres of the brain suffer from ramollissement with nearly equal frequency; and ramollissement of the cerebellum is much more rare than of the brain. But it is not determined whether the gray or the white matter is the more liable to inflammation. Bennett has been led to consider the white as the more frequently softened, while Gluge is of opinion that it is more frequently the gray; but it must be remembered that the gray matter is naturally specifically lighter than the white, and probably also from its structure more amenable to the softening process.

*Induration.*—Instead of being softened, the texture of the brain is not unfrequently indurated, as a result of the inflammatory process, and is associated, also, with an absence of red color. It acquires the consistence of the white of egg boiled hard. M. Dance gives the case of a patient who received a blow on the head about seven months before his death. He afterwards suffered from epistaxis and severe and frequent paroxysms of headache. At length he fell down in walking from the bath, and died convulsed in about a quarter of an hour. On inspecting the brain the convolutions were flattened; there was very little blood, and no serous fluid in the encephalon; but all the substance of the brain resembled white of egg boiled hard. Its weight and density were considerable, and it yielded and recovered its form like an elastic body. There was no trace of a red vessel; but the cortical substance was paler, and the medullary substance whiter than usual (*Répertoire Général d'Anatomie et de Physiologie*, 1828).

*Causes.*—Inflammation of the substance of the brain may be caused by any of the morbid poisons associated with the *specific* or *constitutional* diseases. Many cases also result from mechanical injuries; others from the excitement of insanity and uncontrolled moral feelings. In some instances encephalitis has followed the suppression of a cutaneous eruption, or the accidental inclusion of a nerve by a ligature applied to a bloodvessel (LALLEMAND, quoted by SIEVEKING). In others, caries of the bones of the cranium, and especially of the petrous portion of the temporal bone caused by *otitis*. Intemperance in alcoholic fluids also is a frequent cause of this, as well as of every other disease of the brain. The disease is sometimes brought about idiopathically, by exposure to the sun's rays in very hot summer days, especially in tropical climates. As a secondary disease, encephalitis is produced by cancer, tubercle, and by every other structural disease incidental to the brain textures.

Encephalitis occurs at every age: in childhood during the tendency to hydrocephalus; in adult age from the action of morbid

poisons, and from mechanical and moral accidents; and in old age from the natural decay of the frame. Ramollissement of the brain has occurred at the following ages in a given number of cases: In a few cases from birth to fifteen; 39 cases occurred from fifteen to forty; 54 cases from forty to sixty-five; and 60 cases from sixty-five to eighty-seven. The frequency of this disease, therefore, increases with age. Men are supposed to suffer in a larger proportion than women, probably from their greater exposure to the exciting causes.

The Symptoms of *encephalitis* have considerable latitude. In twelve severe cases related by M. Paroisse, and resulting from wounds, he states that the symptoms were nearly the same in all, and were as follow: The men all stated that after the sabre wound they had felt no other inconvenience than local pain of the injured part, and that for two or three days afterwards they had all been able to march five or six leagues a day. On the third day, however, they had all been seized with fever, which terminated on the evening of the fourth day; but from that period they had suffered little, always preserved a good appetite, and prayed not to be put on a low diet. About the seventeenth day they became downcast and dejected, owing, probably, to many sloughs being detached, and much suppuration taking place about this time. On the following day they first lost the sense of smell, and then the senses of sight and taste. With these symptoms, but without fever or convulsions, they fell into an easy sleep; and, as if they had no further strength to contend with the disorder, they died between the nineteenth and twenty-second days from the infliction of the wound.

The symptoms which have been related by Paroisse agree entirely with those observed by John Hunter. He observed that trifling wounds of the membranes of the brain were often followed by severe and extensive inflammation of those tissues, and by very dangerous symptoms; but if the injury had been great, so as to have excised a portion not only of the arachnoid, but also of the substance of the brain, that the symptoms which followed were comparatively slight—a circumstance which he attributed to the brain, in the latter case, having room for expansion; and he therefore suggested the propriety of extensively incising the arachnoid and pia mater in all cases in which the dura mater alone had been wounded.

In idiopathic inflammation the case may be acute or chronic. The first stage of the acute form is generally of short duration, and in so far the attack may be said to be sudden; and if there have been preliminary symptoms of ill health, the symptoms have generally been headache or long-continued derangement of the digestive organs. *The general symptoms* of the first stage are but feebly marked (unless *meningitis* predominates). There is usually, in the first instance, some heat of head and of surface generally. The face is pale, and the pulse low and irregular. The breathing is variable and sighing. There is slight feverish oppression, with headache and vomiting. *The nervous symptoms* are generally highly marked, and so are those of diminished functional activity in cases

where the cerebral affection predominates as distinct from the meningeal. They consist of—

1. *Mental Phenomena*.—The patient is sullen, and his faculties become obscured. There is confusion of thought rather than delirium, which occurs only in a mild form when the patient dozes.

2. *Sensorial Phenomena*.—Unless meningitis is present, there is no hyperæsthesia; but there is deep-seated, violent, oppressive pain, frequently described as shooting from the centre to the vertex, the temples, eyes, or ears; and it is out of all proportion to the intensity of the febrile phenomena; and while the febrile phenomena subside (as they very generally do in the course of twelve or twenty-four hours) the local pain does not diminish.

The second stage may be ushered in by convulsions; or, after the first stage has lasted two or three days, the pulse usually becomes rapid and weak. The cerebral nervous functions become inactive, anæsthesia, paralysis, and coma commonly follow two or three convulsive paroxysms, and death follows after the first stage has lasted two or three days.

When the signs of meningitis coexist and are unusually severe, the pain deep-seated, and followed, after twelve or twenty hours, by convulsions and coma, there is commonly meningo-cerebritis of considerable extent.

When the cerebritis is local or partial, the symptoms are those which are usually regarded as indicating “inflammatory softening,” “red softening,” as commonly called, or “acute ramollissement.” Premonitory symptoms are common, and consist of—

1. *Mental Symptoms*—namely, some loss of intellectual vigor, failure of memory, confusion of ideas, irritability of temper, and a consciousness of weakness—these symptoms becoming gradually persistent.

2. *Sensorial Symptoms*, such as pain of the head, dull, deep-seated, fixed, and protracted; tingling or numbness in one limb or side; imperfection of the special senses; dimness of sight; dulness of hearing.

3. *Motorial Symptoms*, as evinced in the loss of power of one limb or side, the most important of all the premonitory symptoms.

There is more or less fever in proportion to meningitis: there is heat of head, vomiting, and general malaise. The signs of meningitis, on the other hand, may be feebly marked; but there may be convulsions, followed by coma, partial paralysis, with rigidity, returning more or less rapidly, and ending fatally in a day or two, or from two to three weeks. Thus there may be the symptoms of meningitis, or of cerebritis, of both together, or alternately; and, occurring with such premonitory symptoms as have been indicated, they leave little doubt of the existence of a partial cerebritis.

Although we have seen that the anatomical conditions of the brain-substance generally in acute *ramollissement* are by no means associated with any appearance of much red blood, yet the clinical history of the disease seems closely to resemble that of cerebral hemorrhage, and it is very often impossible to tell whether the physician has to deal with a case of *apoplexy*, as commonly under-

stood, or a case of *cerebral softening*. The premonitory symptoms peculiar to softening appear to be absent in a half, or more than a half, of these cases (ROSTAN, DURAND FARDEL). In some instances, however, the premonitory symptoms just noticed afford strong probability of softening, and are of much value: the absence of them, however, cannot be regarded as equivalent to the absence of softening (REYNOLDS).

The attack itself may be gradual or sudden. Thus, after the progressive development, during some hours or days, of such premonitory symptoms as have been mentioned, the patient gradually becomes apoplectic, or he may at once appear to become so suddenly and instantaneously, without the premonitory symptoms. In the latter case, however, the attack is due to congestion; it gradually passes away, and the patient recovers Intelligence for a time, but the confirmed symptoms of softening remain as follows:

1. *Mental Symptoms*.—Transient excitement or mild delirium may precede the abolition of Perception; and when this does occur it is highly characteristic. Coma is frequently developed abruptly, and is often of the following peculiar character: The patient lies still, as if in a profound sleep, but immediately gives the hand or puts out the tongue, if told to do so, Intelligence remaining intact. The loss of Perception and Volition, however, is not recovered from. Dulness and obscuration of Thought and Perception prevail often to a marked degree.

2. *The Sensorial Symptoms* are not so well marked as the motorial. Hyperæsthesia has erroneously been considered pathognomonic of ramollissement, perhaps because it is more common in softening than in any other apoplectic disease. Numbness and a sensation of cold are not at all unfrequent.

3. *The Motorial Symptoms* are of two kinds,—namely, paralysis, and spasmodic contractions of muscles. The face-muscles act unequally, producing deviation of the features, sometimes very slight, at other times highly marked. Speech is almost constantly impaired, and after slight recovery it continues to be so. Paralysis is commonly limited to one side, sometimes to one limb, but in rare cases it is general. The spasmodic contractions are either of a tonic or of a clonic kind, rigidity or occasional spasm being found in either the paralyzed or non-paralyzed limbs,—most commonly in the former.

The physician, however, will not derive much information from the mere recognition of the presence of single symptoms: it is by a close observance of their combinations that exact diagnosis will be insured. The following combination of symptoms are those which may with most probability be referred to softening:

(1.) Imperfect coma, partial loss of Perception and Volition, with rigidity of the limbs; (2.) Perfect coma without rigidity; (3.) Paralysis without loss of consciousness; (4.) Paralysis with hyperæsthesia; (5.) Rigidity, coming on after the return of Perception and Volition.

The after-symptoms of softening are also strikingly different from those of apoplexy. The morbid phenomena do not suddenly disap-



pear, nor is there the gradual improvement which takes place after apoplexy. Enfeeblement of the mental powers most commonly persists, and the motorial phenomena remain. Slight apoplectic-like seizures occur, convulsive movements and rigidity increase, and some little febrile excitement becomes developed, which in severe cases generally assumes a typhoid type, with brown tongue and rapid pulse. From such a condition recovery is rare.

The duration of life in *ramollissement* of the brain is various; but in 109 cases the disease terminated within the periods indicated in the following table:

DURATION OF LIFE IN CASES OF SOFTENING OF THE BRAIN.

1 died in 12 hours.	2 died in 12 days.	1 died in 35 days.
1 " 15 "	8 " 18 "	1 " 36 "
1 " 24 "	3 " 15 "	1 " 47 "
1 " 32 "	1 " 16 "	1 " 49 "
5 " 2 days.	2 " 17 "	1 " 60 "
9 " 3 "	4 " 18 "	1 " 65 "
5 " 4 "	5 " 20 "	1 " 68 "
4 " 5 "	8 " 21 "	1 " 190 "
7 " 6 "	1 " 22 "	1 " 220 "
8 " 7 "	1 " 23 "	1 " 5 months.
8 " 8 "	1 " 25 "	2 " 6 "
8 " 9 "	1 " 29 "	1 " 1 year.
5 " 10 "	4 " 30 "	2 " 3 years.
4 " 11 "		

It thus appears that *ramollissement* of the brain is more frequently an acute than a chronic disease, the greater number dying before the twelfth day, while at the end of a month only 16 cases out of the 109 were living.

In the thirteen cases which have been collected of *ramollissement* of the *cerebellum*, the impairment of Intellect was trifling, while motion was greatly affected in all except one doubtful case. In ten cases there was palsy, with or without contraction of the muscles of the opposite side of the body; in two others, convulsive actions of both sides of the body; and in the last case observed by Rostan the palsy was on the same side. In this case the disease depended on an exostosis of the petrous portion of the temporal bone. In no instance is it said that any sexual desire troubled the patient.

The symptoms of abscess of the brain are likewise extremely obscure. In a case treated for disease of the nose, the man made no complaint of his head, and was able to sit up in bed, and to assist himself in every way. He died suddenly in the night. An abscess of considerable size was found in the left hemisphere above the ventricle. In other cases, pain, delirium, coma, palsy, and sometimes convulsions, were the symptoms observed. The convulsions were observed most frequently to occur when the abscess formed in the *tuberculum annulare*, or in the *medulla oblongata*, or so close to these structures that the growth of pus would affect their functions simply by its pressure (Baillie, *Morbid Anatomy*, p. 457).

Of eighty instances of abscess of the brain, collected by Professor

Lebert, twenty-two of them, or above a quarter, were cases of scattered abscesses in various parts of the brain; the remaining fifty-eight were cases in which solitary abscesses were found in some part of the encephalon, distributed as follows:

Left hemisphere, . . . in 23 cases.	Cerebellum, . . . in 12 cases.
Right " . . . in 18 "	Pituitary body, . . in 2 "
<i>Corpora striata</i> , . . . in 2 "	<i>Medulla oblongata</i> , . in 1 case.

In the case of multiple abscesses there were never more than five, sometimes three, or two only.

The abscesses generally occupy the white substance, and only affect the gray matter by extension. In form the abscesses are generally oval, and vary in size from a pea to the size of a hen's egg, or larger; so that one entire hemisphere has been found converted into a pouch filled with pus. The pus is generally of a greenish color, of considerable density, and rarely containing blood. Sometimes it is very fetid. It is generally granular, not containing many well-formed corpuscles; and the older the abscess the more the pus-corpuscles are found to be degenerating. Pus is generally found infiltrating the cerebral tissue surrounding the abscess; and in the zone beyond, the tissue is softened, while if the process be recent, a zone of vascular redness surrounds the whole. Connective tissue ultimately grows in a condensed form round the site of abscess, and closes the whole with walls of considerable thickness (1 to 4 *millim.*). Often these walls are very vascular. There does not seem to be any evidence that these encysted abscesses are ever cured.

The lateral ventricles are the parts into which the abscesses most frequently burst. Thickening of the *ependyma* follows, and scattered spots of inflammatory softening may be found in the neighboring tissue. In some cases the abscess makes its way outwards through the petrous portion of the temporal bone or the aural passages.

The most frequent cause of cerebral abscesses is internal *otitis*—a lesion which often results from *scarlet fever*, *typhus fever*, *small-pox*, *measles*, or *scrofula*. They also occur as the sequelæ of inflammation of distant parts, as of *pneumonia*, *pericarditis*, *enteritis*. They may also occur as so-called metastatic abscesses, and as a result of traumatic injury.

The latent character of brain-abscess is important in diagnosis. Sudden headache is the symptom which most frequently excites attention; and it is generally accompanied by febrile symptoms and vomiting. The patients become heavy, morose, and may be delirious, with contraction of the pupils and photophobia. Difficult articulation, numbness, formication, and convulsive attacks may supervene. While the Intellect may suffer comparatively little, sensibility suffers more frequently; and headache, generally at first diffused, is more or less intense, and subsequently becomes unilateral. Coma occurs frequently, is often temporary, and paralysis occurs in about one-half the cases. The paralysis is generally

local; but it may assume the form of general muscular debility. The duration of such cases fluctuates from two or three weeks to two months (LEBERT, SIEVEKING, in *Med.-Chir. Review*, 1857, p. 526).

Schott has analyzed forty cases of abscess of the brain. He finds *otitis*, *pyæmia*, and injuries, to be the most frequent causes. In cases of *otitis* the abscess formed mostly in an imperceptible manner; and in cases of injury the symptoms were very transitory. Encephalitis was present in acute cases (*New Syden. Soc. Year-Book*, 1862, p. 79).

Syphilis is by no means an infrequent cause of circumscribed encephalitis, which terminates in the formation of gummatous tumors in the brain; or it induces internal periostitis, with inflammation of the membranes, with or without *hæmatoma* of the *dura mater*. There is generally in such cases syphilitic lesions in the liver and in the inguinal glands. (See under "Syphilis," vol. i, page 703, *et seq.*)

The symptoms of *hydatids* of the brain are often very obscure. The slowness with which they form probably often causes the brain to become accustomed to their presence, and consequently they do not give rise to any very prominent symptom. Cruveilhier gives a plate of a hydatid occupying the internal surface of the right hemisphere, immediately above the *corpus callosum*, and which caused no cerebral symptoms. Dr. Baillie gives a case in which a serous cyst as large as a gooseberry pressed on the optic nerves at their junction, and yet the pupils were not dilated nor the eyesight impaired till within a day or two of the patient's death. In other cases they cause severe headache, palsy, loss of sight, or of other senses, and also absorption of the bones of the cranium, followed by coma and death.

**Diagnosis.**—The great difficulty in the diagnosis of acute ramollissement is to distinguish it from apoplexy. In most cases it is not possible to make the distinction. "On the other hand," writes Dr. Maclachlan, "there are numerous cases of acute softening which can scarcely be mistaken for cerebral hemorrhage. In such a combination of symptoms as the following—and I do not group them artificially, but as I have repeatedly observed them—the probabilities are, that the case is one of acute softening, and not sanguineous apoplexy: After several days' suffering from headache, giddiness, drowsiness, dulness of comprehension, tingling or numbness in the toes or fingers, followed by sudden hemiplegia, without loss of consciousness, the probabilities are, that the symptoms are due to softening instead of hemorrhage. If to these symptoms succeed pains in the palsied limbs, and diminution or exaltation of the cutaneous sensation, while the symptoms maintain a variable rather than a fixed character, whether the palsied limbs are contracted or relaxed, or alternately contracted, the chances are still greater that the case is one of softening; and the diagnosis may be considered as established should the paralytic symptoms, and with them the associated stupor, preserve this vacillating, impulsive peculiarity, there being periods of amelioration followed by increasing coma and increasing palsy" (*l. c.*, p. 193).

**Prognosis.**—The prognosis in every case of encephalitis is grave; but, as far as we can judge, even acute cases recover, and live for many years afterwards, notwithstanding the unfavorable opinion expressed by Rostan. The changes seen in the brain after death in cases which have recovered have already been noticed (p. 297, *ante*).

**Treatment.**—In diffuse inflammation of the brain arising from mechanical injuries there can be no doubt that bleeding and antiphlogistic treatment generally are most beneficial, when employed with a wise discretion and at an early period. When, however, inflammation occurs during the progress of a *zymotic* or *constitutional* disease, it is necessary that such measures be employed with the greatest caution, and in the majority of such instances they are better omitted altogether; for we find in many cases of typhus fever, in which the brain is probably partially impaired in consistence, that the patient recovers under a stimulant treatment.

In acute idiopathic *ramollissement* of the brain the treatment can hardly be said to be yet determined; but there is good reason to believe that general bleeding is only to be practised with benefit when the disease is associated with congestion of the brain. Most advantage is to be derived from the use of tonics, and of a nutritive diet. As a general rule, *cerebritis* does not admit of so copious depletory measures as *meningitis*. In acute cases, where the premonitory symptoms of congestion are obvious and urgent, bleeding may be carried to a considerable extent, consistent with the nature of the case and the individual; and after a reasonable quantity of blood has been taken without producing nausea or fainting, the bleeding may be again repeated till some decided impression is made. In many cases, on the other hand, cupping, or bleeding by means of leeches to the temples or back of the ears, may be more useful than venesection. Such cases are indicated by the signs of determination of blood to the head, heat of the scalp, suffusion of the eyes, redness of the face, with or without inordinate action of the carotids. Such cases are also more likely to be benefited by the cautious repetition of this treatment than by general bloodletting, which can very seldom be required in softening of the brain occurring in persons turned of fifty (MACLACHLAN).

Even when the pulse may seem to warrant bleeding, and at a time when bleeding may seem safe and proper, it is advisable, before having recourse to this measure, carefully to examine the state of the heart, the state of the arteries at the wrist, and the condition of the corneæ. By such an examination, assisted by the previous history of the case, the condition of the cerebral arteries will best be indicated, and the readiest clue gained to the probable nature of the cerebral affection. If the heart's action and sounds are feeble, or if signs of valvular insufficiency are present, it is probable that the softening is anæmic, atrophic—a consequence of partial inanition, and not of partial cerebritis. This conclusion is still more probable if the radial arteries are rigid, and if the *arcus senilis* is fully developed. To bleed under such circumstances is unwarrantable.

In severe seizures resembling apoplexy the bowels should be opened by an enema of castor oil and turpentine. In less urgent cases, where there has not been loss of consciousness, or where it has been restored and the patient is able to swallow, a purgative of colocynth and calomel, with or without croton oil, may be substituted for the enema. Perfect rest in the recumbent posture must be enjoined, with the head slightly elevated, and all constrictures of dress removed from the neck. The scalp should be kept cool by rags dipped in cold vinegar and water, or iced water. If much reaction supervene, leeches may be applied to the head; and should the pulse be weak, twenty or thirty drops of the *spiritus ammoniæ aromaticus* may be administered. The catheter may require to be used; and the bladder must always be examined, in case the urine may be retained. Nourishment must be given in small and oft-repeated quantities.

If sanguineous effusion has actually happened, *calomel*, to the extent of permitting the mouth to be made slightly tender, is believed by not a few to encourage the absorption of the fluid. It can only, however, be given in healthy subjects, free from gouty or renal disease; and Dr. Maclachlan recommends that it may be given in the form of *three* or *four* grains of blue pill and two of James's powder every evening for ten days or a fortnight, the effects being carefully watched, so that the gums should not be more than touched.

To allay the pains in the palsied limbs *opiates* are of great value. They procure sleep and moderate tetanic rigidity of the flexor muscles. *Muriate of morphia* may be given in doses of one-sixth to one-fourth of a grain at bedtime; and the doses may after a time be increased, if necessary; and may be combined with extract of colocynth or croton oil, to counteract the tendency to constipation (MACLACHLAN, *l. c.*, pp. 195, 196).

In chronic cases local bleedings from the temples, or from behind the ears, combined with moderate purgatives every three or four days, are demanded in most cases, with a blister applied now and again to the nape of the neck; and as much as possible the currents of blood must be diverted from the brain. Headache and stupor are generally relieved by full feculent evacuations. The diet ought to be strictly that of a vegetarian, and as little stimulant as possible (WOOD).

In the more chronic, and although ultimately fatal, forms of the disease, life is evidently prolonged by mild tonics, attention to the bowels, and by a liberal and nutritious diet, with such a graduated allowance of alcoholic beverages as the case may require. Beyond this the medical treatment of ramollissement of the brain is still a problem, with only a few unsure data to guide us for its solution.

#### ATROPHY AND HYPERTROPHY OF THE BRAIN.

*Atrophy of the brain*, or diminution of its substance, without induration or softening, is usually congenital, or the consequence of



some severe hydrocephalic disease, or of old age, or of long-standing exhausting diseases, especially in children, serum being effused in the space between the brain and its coverings, in order to supply the deficiency of bulk. Sometimes also one side may be more atrophied than another; the membranes appearing to be greatly shrivelled after the fluids escape; the convolutions are thin, and the sulci wide. The sufferers are generally idiotic, and possess but little use of their limbs. Andral gives a singular case, in which the patient, a girl, though an idiot, was able to do little errands in the neighboring villages, and lived to an early adult age, yet, when examined after death, she was found to have no trace of cerebellum.

*Hypertrophy of the brain* is usually connected with hydrocephalus, or is probably caused by some inflammatory action. Those persons who thus suffer seldom possess much power of Intellect, but their faculties generally are less impaired than in cases of atrophy.

## SECTION II.—CEPHALIC DISEASES, CHARACTERIZED BY A GROUP OF SYMPTOMS COMMONLY CALLED APOPLECTIC.

The literal meaning of the term *apoplexy* conveys the idea of a *sudden stroke*, and it has been usual to confine the term to the results produced by extravasations of blood into the nervous tissue of the brain, a portion of which is thus destroyed. More comprehensive pathological doctrines teach us to give a wider signification to the term. It is now used to characterize a group of symptoms irrespectively of the anatomical conditions upon which they may depend. These symptoms consist of,—(1.) Premonitory warnings, extending over variable periods (seconds, weeks, months, or years), marked by sundry derangements of the nervous functions, such as loss of Memory, dulness of Sensation, or diminished power; (2.) The individual is more or less suddenly deprived of Volition and Perception in their relation to Sensation and Motion. Consciousness is thus more or less lost, and paralysis is more or less complete. The patient may fall to the ground completely insensible, or he may only stagger and cling to some object for support. The respiration and circulation may be unaffected, or the former may be stertorous and the latter labored. Some group of muscles, a side of the body, or the whole body is paralyzed, flaccid, motionless; or it may be rigid with tonic, or convulsed with clonic spasm. From this state the patient may never recover. Life becomes gradually extinguished, or the sufferer may recover partially or entirely; in the former case leaving some mental, motorial, or sensorial faculty impaired for weeks, or for the whole of after-life. The essential phenomena of an apoplectic seizure consist in the severance of the brain-functions, namely, Volition and Perception, from Motion and Sensation: the other symptoms that occur are additional phenomena, depending on secondary changes subsequently induced in the part, or its vicinity, which has been the primary seat of lesion.

The essential phenomena of the *apoplectic state* are found to be due to a variety of local lesions, or complex morbid states, and not

to any constant lesion. One or more of the following local lesions or complex morbid states may induce the apoplectic condition, namely,—(1.) *Congestion of the brain*, or what is commonly called *determination of blood to the head*,—*congestive apoplexy*. (2.) *Hemorrhage*, or *extravasation* (a.) *into the substance of the hemispheres or cerebellum*, (b.) *into the ventricles*, or (c.) *into the arachnoid cavity*. One or other of these lesions (under 2.) constitutes *apoplexy* in the common acceptation of the term,—*sanguineous apoplexy* or *cerebral hemorrhage*. (3.) *Sudden serous effusion in large quantity* is equally efficient in bringing about the apoplectic state commonly called *serous apoplexy*. (4.) *Local cerebritis*, or *softening of the brain*, produces, as already shown, symptoms, in the first instance, of an apoplectic kind. So also do (5.) *Tumors of the brain*, or *meningitis*; (6.) *Tuberculous meningitis*; (7.) *The progress of various specific and constitutional diseases from blood-poisoning*; (8.) *Anæmia*, as in the *hydrocephaloid disease of children*, *disease of the heart*, and *vascular obstructions*.

To the phenomena produced by the first three of these conditions only has it been common or usual to apply the term—

#### APOPLEXY.

LATIN, *Apoplexia*; FRENCH, *Apoplexie*; GERMAN, *Apoplexie*—Syn., *Hirnschlag*; ITALIAN, *Apoplessia*.

**Definition.**—*A disease essentially characterized by the sudden loss, more or less complete, of Volition, Perception, Sensation, and Motion, depending on sudden pressure upon the brain (the tissue of which may be morbid), originating within the cranium.*

**Pathology.**—This disease was well known in the Greek and Roman schools of medicine, and is of too frequent occurrence, and of too striking a character, to have escaped observation even in the rudest ages of society. Patients have died with undoubted apoplectic symptoms when nothing has been found but congestion of the vessels of the scalp, of the membranes of the brain, and of the brain itself, but without the extravasation of a particle of blood. More generally, however, a greater or less quantity of blood has been effused either into the cavity of the arachnoid, into the substance of the brain, or into some of the ventricular cavities. Thus it is that the lesions found in cases which die of undoubted apoplectic symptoms vary much. Sometimes the evidently congested state of the brain during life leaves no trace visible after death. Such cases have been described as “*nervous apoplexy*” (SANDRAS). Although much stress is laid upon the fact that “*a sudden pressure upon the brain is necessary to produce the apoplectic state*,” yet it is difficult in all cases to account for the proximate cause of the disease by such an explanation; for when the quantity of blood extravasated has not been larger, for instance, than a barley-corn, it is difficult to account for all the phenomena by mere pressure. The ideas or theories which have thus been formed to account for the apoplectic state may be shortly stated as follows: (1.) The result of sudden

pressure, effected by causes within the cranium ; (2.) From a peculiar morbid state of the nervous matter of the brain, and which predisposes to the extravasation of blood (DRS. ROBERT WILLIAMS, WOOD, SIEVEKING)—an apoplectic orgasm, as it has been called by some, but which is probably of the nature of softening (ROCHOUX); (3.) A morbid condition of nerve-matter and minute bloodvessels, also probably of the blood itself (BOUILLAUD, PAGET), and in connection with chronic renal disease (BRIGHT, BURROWS, CHRISTISON, KIRKES).

When the quantity of blood extravasated is small, the disease is seldom fatal from the first attack ; but the rapidity of the fatal issue appears to bear some relation to the vicinity of the hemorrhage to the *medulla oblongata* ; and the effusion of blood into the ventricles is also generally most rapidly fatal.

In examining apoplectic cases it is therefore not unusual to find a cavity scarcely bigger than a barley-corn in the substance of the brain, the evidence of the primary attack, and containing a clot of blood variously changed. If the blood be effused among the membranes, it may be altogether absorbed, and not a trace of disease be found. In severe cases still greater quantities of blood are effused ; and if the apoplexy destroy the patient in a few minutes or a few hours, the quantity of blood effused will sometimes fill the whole cavity of the arachnoid, or extensively rupture the substance of the brain, forming a cavity as large as a nut or an egg, or even lay the ventricles into one cavity.

It is rare that sanguineous effusion occupies both cerebral lobes, or the whole extent of the membranes of the brain, although such instances are occasionally seen. More commonly it is limited to the substance of one hemisphere, or to the membranes covering it, or to the cavity of a ventricle. When the membranes of the brain are affected, the more immediate seat of the hemorrhage is usually that portion covering the convexity of the brain. This varies, however, so that the portion covering the base, or that investing the cerebellum, or, indeed, any other part, may be its seat.

The superficial membranes of the brain are not the only membranes of that organ which are the seat of apoplectic effusion. Hemorrhage may take place from the membrane lining the ventricles, and which sometimes bleeds so profusely as not only to fill the lateral ventricles, but even to enlarge their cavities. As death in these severe cases is usually sudden, the walls of the ventricles are generally healthy ; but in some very few instances the *septum lucidum* has been found ruptured, and the ventricles have freely communicated. The smaller ventricles are in a very few instances also the seat of apoplectic effusion. Dr. Abercrombie gives a case in which the third and fourth ventricles were filled with blood. The patient was not at first insensible, but gradually became so, and died in a few hours.

The appearance of the blood effused into the membranes of the brain varies according to the time which elapses before the patient dies, thus affording opportunity for its examination. If that event takes place in a few hours after the attack, the blood is still fluid, or is found in black clots, while the membranes, except being infil-

trated with blood, are as yet healthy. The substance of the brain, likewise, has no other appearance of disease than that of being flattened, from the presence of the extravasated blood. If the patient, however, survives a few days, the membranes show marks of inflammatory action. They are injected, thickened, and although dry and pitchy-like in the immediate neighborhood of the clot, have yet some serum effused in other parts of the space they inclose. The convolutions of the affected part of the brain are likewise now not only flattened but softened. Thus various processes immediately commence in the blood after it is extravasated, as well as an interchange of processes between the blood and the surrounding parts. The most obvious of these phenomena are the formation of a coagulum or clot; its solution, and the formation of blood-crystals; the gradual absorption of the more fluid constituents; the formation of an organized membrane round the clot; continuous absorption of the exudation; induration of the surrounding cerebral parts; contraction of the cavity, and ultimately the formation of a cicatrix. Those changes are effected with various degrees of rapidity, depending on the site and extent of the extravasations, the healthy state of the nervous texture, and of the patient constitutionally.

When effusion has taken place into the substance of the brain, if the patient has died in the fit, or shortly after, the hemorrhagic cavity is found filled with half-coagulated blood, its walls irregularly softened, and dyed to the extent of some lines deep with the coloring matter of the blood; and a small stream of water directed upon this part at once removes the extravasated blood, and also a layer of softened cerebral matter. Again, if the patient has survived a week, the blood is found coagulated, and the serum set free; but the presence of the clot has caused inflammation, so that the walls of the cavity are not only discolored, but more decidedly softened, and are softer in proportion as they are nearer the clot. If life be prolonged till the fifteenth day, the serosity is absorbed, but the walls of the cavity are still of a deep red. About the seventeenth day Virchow has discovered blood-crystals, or hæmatoid crystals, in the cavity. These blood-crystals were first discovered by Sir Everard Home, and have been more recently described, and their nature explained, by Funke, Kunde, Lehmann, Beale, Parkes, and Sieveking. It appears that these crystals do not form from clotted blood until the blood-corpuscles have become ruptured by endosmosis. Their contents then escape and crystallize as the solution gradually becomes concentrated (BEALE). Thus another guide to the age of the clot exists in the presence of these crystals. About the thirtieth day, if the patient lives so long, the clot is isolated, and a membrane forms, at first muciform, fragile, intermixed with particles of cerebral matter, and also with some of the coloring matter of the clot. By degrees this membrane becomes more consistent, the clot diminishes, and some serum is probably secreted by the new membrane surrounding it. The cyst has also been found fully formed, organized, and nearly empty, by the thirteenth day (MACINTYRE); and by the seventeenth day after extravasation it has been found to contain sanguineous fluid (MOULIN, SIEVEKING). The cerebral walls

surrounding the cyst, previously softened, now become indurated, and are stained yellow from the usual changes which the extravasated blood with which they are penetrated undergoes—a color, however, which they ultimately lose. The cavity thus formed may be filled at length with serum only; or, the serum being absorbed, the membranous cyst may ossify, and may be thus converted into a bony tumor. At other times the opposite sides of the cavity unite by a kind of areolar membrane, which thus forms a species of cicatrix, but possessing so little power of conducting nervous influence that the patient seldom recovers from his palsy. Such is a short outline of the effects of hemorrhage into the substance of the brain. The size of an apoplectic cavity varies from a barley-corn to that of an egg, and their number is as variable as their extent. Sometimes we find but one, sometimes two, and in a very few instances three or more cavities. When many apoplectic cavities exist in the brain, it is rare to find them all in the same state. Some are old and almost obliterated, others are fresher, and others again quite recent, their different stages marking a distinct and different period of attack. An account of a most interesting case of this nature is given by Dr. Fuller, in which there were *six* clots, each of a different date, and in different stages of discoloration, and corresponding to each of *six* well-marked apoplectic seizures in the course of nine months (*Diseases of the Chest*, p. 602).

Some of the most exact data we possess regarding the pathology of apoplectic seizures are to be found in the *Transactions of the Pathological Society of London*. Up till 1857, lesions of the brain connected with apoplectic seizures have been shown at the Society in *fourteen* cases. The position of the blood effused in these cases was as follows: (1.) Superficial meningeal effusions, five cases; (2.) Within the substance of the hemispheres or the *central ganglia*, four cases; (3.) In the *pons Varolii*, two cases; (4.) In the *pons Varolii* and *crura*, one case; (5.) In the *pons Varolii* and *cerebellum*, one case; (6.) In the *pons crura*, and *cerebellum*, one case.

*Superficial* or *ventricular* extravasation occurred in five cases; that is, the blood lay in a more or less coagulated mass under the arachnoid over the hemispheres, and was most abundant at the base and about the roots of the nerves. In two of the cases there was some evidence of previous *meningeal* disturbance. This evidence consisted of adhesion of the cerebral arachnoid and pia mater to the cerebral convolutions along the anterior margin of the middle line of the hemisphere: the history of the other case recorded the occurrence of two previous apoplectic seizures. In it post-mortem evidence of previous irritation also existed, in the adhesion of the *dura mater* to the calvarium.

The conditions as to general health of those in whom these attacks occurred were as follow: One was convalescent from a uterine disease, the nature of which is not stated; another had chronic bronchitis and asthma; in a third intemperance was predominant; and in the fourth there had been two previous attacks of apoplexy. In three of these cases the condition of the heart is recorded as being larger than natural—in one of them weighing



11½ ounces, in another 15 ounces, where the hypertrophy was general. In the case of the repeated attacks the heart was fatty, as well as the coronary arteries and those of the pia mater. The kidneys also were fatty. In all of them the attack was sudden—followed in one by instantaneous death, in the others by insensibility, collapse, and blanched surface. In one case the pupils were dilated; in another, where the *septum lucidum*, *corpora striata* and *optic thalami* were broken down, there were paralysis and rigid contraction of the right arm. Death followed in thirty-four hours; another died in twenty-two hours; one on the third day, and another on the fourth. In none of these cases is there noticed any lesion of the brain-substance itself. In all, the hemorrhage appeared to proceed from the tomentose or vascular surface of the pia mater. In this variety of meningeal hemorrhage the vessels of the pia mater become inordinately injected, and the effusion consisted of blood, or merely blood-serum (DR. WILLIAMS). While this effusion of blood or serum lies on the convoluted surface generally, it occasionally, as in this instance, is effused into the ventricles, so that the brain was compressed both from the peripheral or pericranial surface and from the ventricular or internal surface. There was no connection of these cases with external injury, the most common cause of extravasation between the pia mater and brain. They all appear to have been the result of morbid states of the vessels of the pia mater. Cases of this kind are recorded by Craigie in the *Edinburgh Medical and Surgical Journal*, vol. xviii, p. 487; also by Morgagni, *Epist.* iii, 2 and 4, quoted by him from Valsalva, *Epist.* xi, 19 (see Craigie's *Pathological Anatomy*, p. 730).

*Extravasation into the Substance of the Hemispheres or Central Ganglia.*—In four cases some part of the cerebral substance contained the extravasated blood, more or less free, or surrounded by a cyst. In two of the cases the clot was surrounded by a cyst, in one of which, death taking place thirteen days after the seizure, the cyst was found nearly empty, disintegration and absorption having thus early taken place (MACINTYRE). These cysts seem to be formed of fibrilloid tissue like condensed fibrine, more or less hyaline, or stained with coloring matter of the blood; and in one case, where death occurred twelve weeks after the fit, blood-crystals were found in abundance in those parts of the wall of the cyst, and most abundantly on the yellow parts. In this case, at the end of twelve weeks, the cyst was filled with slightly turbid yellow fluid, a small portion of coagulum only remaining (BRISTOWE). In the case where the extravasation occurred into the *corpus striatum* it protruded into the lateral ventricle of the right side, and nearly filled it, and the nervous substance around was broken down and soft. Death occurred in half an hour (GIBB). The position of the clot in the fourth case was above the lateral ventricles, separated from the longitudinal fissure only by the gray matter. Some ecchymosis existed in the neighborhood, and there was yellow discoloration on the surface of the left *corpus striatum*, where two cysts existed with fibrous walls, the remains of old extravasations (OGLE).

The conditions as to general health of the patients are not stated.

One case occurred during convalescence from an attack of *delirium tremens*, after having been apparently benefited by a dose of morphia amounting to one grain of the *acetate*. There was extensive cardiac, pulmonary, and gastric disease in this case, and the patient lived thirteen days after the seizure. A bloated, florid look were the only circumstances to indicate the general state of the case where the extravasation occurred in the hemisphere above the lateral ventricle. In three of the cases the heart is specially noted as fatty, its weight in one instance being thirteen ounces, in which case, also, the liver was enormously enlarged, containing sugar. In all the cases the arteries at the base of the brain were diseased. The symptoms were generally those of a fit, in one case like syncope, when the extravasation was into the ventricles and *corpora striata*, death following in half an hour, preceded by a feeble pulse, the pupils being slightly dilated. In the other case, in which there was post-mortem evidence of previous extravasation by the remains of cysts on the surface of the left *corpus striatum*, together with a thickened and opaque arachnoid with subarachnoid fluid, a fit occurred with paralysis of right side, a feeble and quick pulse, followed by convulsions, contracted pupils, and incoherent speech, breathing becoming stertorous, death following by coma on the sixteenth day.

*Extravasation into the Pons Varolii and other parts.*—Five cases are noted in which the extravasation took place into the *pons Varolii*. In two of the instances the *pons* alone was implicated. In one, the *pons* together with the *crura cerebri*; in another, the *pons* together with the *cerebellum*; and in a third, the *pons* together with the *crura* and *cerebellum* were implicated. Of the cases where the *pons* alone was implicated, a clot the size of a pea occupied its centre. It occurred in a lunatic, taking his walk out of doors in his usual health. Sudden paralysis indicated the seizure, and death followed in twenty-four hours. In the other case (age thirty-four) the *pons* contained a clot as large as a filbert-shell, seated in the middle line, the blood having burst through into the fourth ventricle. Surrounding the clot the nerve-tissue was softened and shreddy. The seizure was sudden, with right hemiplegia and anæsthesia. Paralysis of the left side remained, with feeble circulation, and death followed in five days. In the instance in which the *crura* as well as the *pons* was affected, the blood was partially coagulated, breaking up the nervous matter, and distending the parts into a bag. In the instance in which the *crura* and *cerebellum* as well as the *pons* were implicated, the clot was similarly inclosed in a bag of nervous matter. In the other instance the lesions in the *pons* and *cerebellum* were confined to the left side.

These cases were distinguished by rapid and extensively increasing difficulty of breathing—in two amounting to *stertor*—and by contraction of the pupil; features placid; convulsions of pectoral muscles; coma becoming more and more perfect; and death followed in from four to twelve hours. In three of the cases extensive disease of the vessels, both large and small, especially towards the brain, is noted.

These cases have been related, from the supposed rarity of the lesion as to its seat. And although the greater number of the cases are deplorably meagre in the details recorded in the Society's *Transactions*, yet they are exceedingly interesting on the whole, when thus classified, establishing as they do several facts, not accurately determined before, relating to the pathology of cerebral apoplexy or cerebral hemorrhage. They confirm the observation that, when the extravasation is extensive, although superficial, the result is rapidly fatal by the coma, resulting from compression over a large surface; that when effusion is sudden and extensive in *certain parts* of the brain, such as in the *corpora striata*, or *optic thalami*, or the *pons Varolii*, the result is rapid death; and generally they confirm the belief that the more nearly the extravasations approach the *medulla oblongata*, the more rapidly fatal is the result. The symptoms which were associated with irritation of the parts connected with the *pons Varolii* were peculiarly well marked in three instances. So delicate are the sensibilities of these parts in the exercise of their functions, that it has been taught that blood is rarely found effused in them as a lesion after death, because simple vascular injection of such parts was considered sufficient to disturb the functions of the brain so much as to produce the fatal result before such changes can be effected (Craigie's *Prac. of Physic*, p. 309, vol. ii).

The cases are much too few to found any general conclusions upon relative to the numerical frequency of lesion in any particular part of the brain, associated with the apoplectic seizure.

It is well established (notwithstanding statements to the contrary, founded on experiments of a certain kind), that the brain-substance is liable to compression from vascular injection generally, and increased turgidity of its vessels. There are also parts of the brain so constituted anatomically that hemorrhage more readily occurs in them than in other parts. The anatomical constitution which favors this consists in the increased provision for the transit of bloodvessels. For instance, through the white perforated spot at the commencement of the fissure of Sylvius, the sylvian or middle artery of the cerebrum sends its numerous branches of various size into the substance of the brain. These, in the first instance, penetrate the *corpus striatum*, which lies immediately over this *anterior perforated spot*. The *corpus striatum*, and the parts at its level, from their anatomical position and relations, are thus the most vascular parts of the whole brain; and most pathologists agree in considering these to be the parts most liable in the substance of that organ to effusion of blood, more especially when the vascular system of the brain is overloaded (BONETUS, MORGAGNI, ROCHOUX, ANDRAL, CRAIGIE. Opposed to this belief were, HOWSHIP, LERMINIER, SERRES, TACHERON). A summary of the facts recorded may be stated as follows:

Site of Effusion.	Andral.	* Tach- eron.	† Serrea.	‡ Ler- minier.	Path. Soc. of London.	Total.
Corpora striata, . . . . .	61	8	—	1	1	66
Thalami optici, . . . . .	85	2	—	2	—	89
Portions of hemisphere above cen- trum ovale, . . . . .	27	9	2	8	8	44
Lateral lobes of cerebellum, . . . . .	16	—	—	—	—	16
Before corpora striata, . . . . .	10	—	—	—	—	10
Mesocephalon, . . . . .	9	1	2	—	—	12
Spinal cord, . . . . .	8	—	—	—	—	8
Posterior lobes, . . . . .	7	2	2	—	—	11
Middle lobe of cerebellum, . . . . .	5	—	—	—	—	5
Peduncle of brain, . . . . .	8	—	—	—	—	8
Peduncle of cerebellum, . . . . .	1	—	—	—	—	1
Corpora olivaria, . . . . .	1	—	—	—	—	1
Pituitary gland, . . . . .	1	—	—	—	—	1
Pons Varolii, . . . . .	—	2	8	—	5	10
Meningeal hemorrhage, . . . . .	—	—	—	—	5	5
Level of corpora striata and into them,	202	—	—	—	—	202
Total, . . . . .	386	19	9	6	14	444

The result of these observations shows that the comparative liability of parts of the brain to extravasations of blood may be stated in the following order:

- (1.) *Corpus striatum*, *opticus thalamus*, and hemisphere at the level of these parts; (2.) The *corpus striatum* alone; (3.) The hemisphere above the *centrum ovale*; (4.) The *thalamus opticus* alone; (5.) The lateral lobes of *cerebellum*; (6.) The *mesocephalon*; (7.) Posterior lobe of *cerebrum*;  
{ (8.) Before the *corp. striat.*; } (10.) { Middle lobe of *cerebellum*; }  
{ (9.) *Pons Varolii*; } { Meningeal hemorrhage; }  
(11.) Peduncles and olivary body.

Much more extensive records, however, are required to determine these points accurately; and Rokitansky does not consider as yet that we can state such a result in other than general terms. While the ages of those whose cases are recorded varied from thirty-three to sixty-nine, the average age was fifty-four.

*Circumstances under which the lesion occurred.*—In two of the cases the arteries are stated to have been healthy. In three of the cases no mention is made in regard to them; but in three there was hypertrophy of the heart, amounting in one case to fifteen ounces. In all the others (nine in number) the arteries were in a diseased condition; and in seven cases there was evidence of concurrent disease in other organs.

Thus far these cases rather tend to confirm the observation of Paget in 1850; namely, that sudden death from apoplexy is most

\* *Recherches Anatom. Pathog.*, par C. F. Tacheron, tome xiii, Paris, 1823, Ordre xiv, An. 81.  
† *Annuaire Med.-Chir.*, p. 324, sect. xi. ‡ *Ibid.*, p. 213.

generally associated with fatty degeneration of the minute cerebral bloodvessels; while, at the same time, the associated morbid states of other organs are such as to lead to the belief of a more general morbid state, and perhaps more especially of the nervous matter—an apoplectic orgasm which predisposes to the extravasation of blood from diseased bloodvessels—the belief now most generally entertained regarding the nature of the apoplectic lesion.

Another interesting feature in the pathology of the lesions demonstrated to the Society is the organization of a wall round the extravasation, and the future changes of the blood-clot, as shown by its examination at various periods after the extravasation. Cysts existed in two of the cases, in one of which, on the thirteenth day after the seizure, the cyst was found nearly empty, the absorption having thus early taken place (MACINTYRE). On the other hand, again, at the end of twelve weeks, a portion of coagulum still remained inclosed in a cyst, and blood-crystals existed in abundance on its walls (BRISTOWE). Even at a still longer interval bloody fluid has been found in a cyst otherwise nearly empty, as recorded by a French physician of the name of Moulin, who mentions such a morbid state existing seventeen years after the extravasation (SIEVEKING). The age of the clot may thus, in some measure, be indicated by the cyst, the condition of its contents, and especially as to blood-crystals. Virchow has recorded their existence in the cavity by the seventeenth day; and, as shown by Beale, they do not form from clotted blood until the blood-corpuscles become ruptured by endosmosis. The contents of the blood-cells then escape and crystallize, as the solution gradually becomes concentrated.

**Symptoms.**—Whatever may be the pathological doctrines taught regarding the morbid state of the cerebral parts in apoplectic states, we are able practically during life to do little more than merely to recognize the apoplectic state itself; the diagnosis between the congestive, the hemorrhagic, and the serous or merely morbid nervous state, can only be arrived at approximatively by a careful comparison of symptoms closely observed, such as are detailed in the treatise of Dr. Russell Reynolds already noticed, and from which the following statements are condensed:

**I. Symptoms of Apoplexy from Congestion.**—The face, scalp, and conjunctivæ are increased in vascularity; the skin generally is of a dusky venous hue, and the surface is warm. There is fulness of the jugular veins, with increased pulsation in the carotids. The tongue is foul, and nausea prevails, with constipated bowels. Respiration and the pulse are both labored, and the extremities are cold. Such are some of the general symptoms which indicate the approach of an attack of the congestive form of apoplexy.

The symptoms peculiar to the brain itself are,—

1. *Mental.*—The activity and power of the Intellect are diminished. General confusion of thought prevails, with deficient memory. Any attempt at mental exercise increases the expression of these signs; so does the recumbent position and emotional disturbance. Sleepiness, also, with labored respiration, is common, especially after meals; and there is a general tendency to inaction of body



as well as of mind—a “not-to-be-disturbed” sort of desire is experienced. Such mental phenomena, however, are not permanent; and while there is a readily induced state of general confusion, there is no persistent, special, or permanent loss of power of Intellect.

2. *Sensorial*.—The senses generally are obtuse. The hearing is dull; and heavy rumbling noises are constant auditory illusions. The sight is dim, or *amaurosis* is complete, and often black or variously colored spots are seen floating in the field of vision, or other spectral illusions are more or less constant. The patient may see only half of an object, or halves of objects of different colors. Vertigo is also present, with a sense of fulness and oppression in the head; numbness and weight of the limbs; dull and heavy *cephalalgia*. These symptoms, however, are only of occasional occurrence, and change their localities. While the absence of pain is not now considered of much diagnostic value, yet the occurrence of severe acute pain is generally indicative of something more than congestion.

3. *Motorial Symptoms*.—Little jerkings of the muscles, and irregular or sluggish movements of the eyeballs, are occasional. These precursory symptoms having generally been more intense for a few minutes or hours, an attack takes place, distinctly apoplectic. The seizure commonly occurs during some muscular exertion, such as lifting a heavy weight, pulling on a pair of boots, blowing the nose, straining at defecation, or the like; or even upon a simple change of posture, such as stooping, or suddenly assuming the erect attitude.

The special nervous symptoms of the attack in the *congestive form* are,—

1. *Mental*.—Some evidence of the existence of Perception may generally be obtained by loud noises, speaking to the person by name, or pinching him. If, however, Perception is quite extinct for a few seconds or minutes, it soon again partially returns, and there is confusion of Thought, with little Volition as to the direction either of Thought or movement.

2. *Sensorial*.—Except during the first few moments of the attack, when sensation generally is gone, the changes are slight. Sensation,—indicated at least by reflex action (the limbs being withdrawn if pinched),—is generally present.

3. *Motorial*.—There is more or less paralysis of all the limbs, to a slight degree, and for a short time. It is very rare to have either hemiplegia or paraplegia. Short or involuntary evacuations do not occur unless there have been some convulsions. There is no rigidity of the limbs, but clonic spasms are not unfrequent. Generally, and in the course of a few minutes, the symptoms begin to abate rapidly, and they rarely last even for an hour. With the return of consciousness paralysis disappears, and sensibility rarely remains deficient.

The manner of appearance and proportion between the three groups of nervous symptoms is of great diagnostic value. As indicative of *congestion*, rather than of *hemorrhage* or *softening* any

one of the following combinations of groups of symptoms are of importance to be noticed :

1. *The simultaneous development of the three groups of nervous symptoms. There being either—*

2. *Distinct loss of Perception, profound coma, and general paralysis, without rigidity or convulsion.*

3. *Imperfect loss of Perception, with general paralysis.*

4. *General paralysis, incomplete in degree, and sensation unimpaired, or but little affected ; or—*

5. *Paralysis complete in degree, but without stertor or rigidity.*

II. **Symptoms of Apoplexy from Hemorrhage.**—*A. Into the cerebral substance of the hemispheres.* Very different statements are made as to whether or not premonitory symptoms are present ; and the practical point in diagnosis which such discrepancy of statements has taught is, “*that the non-existence of precursory symptoms in a given case is in favor of the belief that hemorrhage rather than congestion is the cause of the lesion or softening.*”

The attack is generally sudden, and rapid in its development. Sometimes after a few hours there may be sudden aggravation of the symptoms, generally due to a renewed extravasation. The patient, if standing, generally falls instantaneously, as if knocked down. It is the nervous symptoms, however, which are of the most importance.

1. *Mental.*—Loss of Consciousness (of Volition and Perception) is commonly complete at the outset. For a few seconds at least the patient is utterly deprived of intellectual power, which in slight cases partially returns in a few minutes. In severe cases, however, Perception does not return till after some hours, and with vague ideas of things ; expressions of Thought are confused, amounting to delirium ; and *after the first few minutes or half hour has passed, the degree of intellectual obscuration may be taken as an approximate measure of the amount of extravasation*, although it is to be remembered that there are some exceptional and rare cases on record of hemorrhagic apoplexy in which the mental faculties were very slightly, if at all, impaired. After some days the Intellectual powers are often entirely restored ; but in many cases confusion of Thought and partial loss of Memory remain. If the case does not terminate fatally, the well-marked character of the recovery is a strong presumption that the symptoms resulted from hemorrhage, and were not due to softening of the cerebral substance.

2. *Sensorial.*—Sensibility is usually less commonly affected, and less intensely, than mobility. When cutaneous anæsthesia is complete, although the surface so affected may be limited in extent, the occurrence indicates severity of lesion. In slight cases there is generally only numbness and tingling of the tips of the fingers. Evidence of Sensation may be obtained when there is no proof of distinct Volition. During the profound coma of the attack at the commencement, the dilated pupil and the half-opened eye indicate that the retina has lost its impressibility ; and if hearing and smell are similarly affected, the persistence of such symptoms are signs of evil omen.

3. *Motorial*.—*Paralysis* is present in the immense majority of cases, its characteristic form being *hemiplegia*; but sometimes it is general, the proportion of cases being as .84 to .16 per cent.; and when the paralysis is general, the hemorrhage is rarely limited to the substance of the hemispheres. During profound stupor the deviation of the face indicates paralysis on one side. In less severe cases the condition of the limbs as to Volition is the guide. The tongue commonly deviates to the paralyzed side; and any extreme movement of the face, such as crying or laughing, renders the inequality of action more apparent. The *orbicularis oculi* generally escapes paralysis, or is less affected than the other facial muscles. The loss of motion is commonly absolute at first, especially in the arm, which is generally more profoundly affected than the leg, the one being more completely paralyzed than the other. Stertor, with involuntary defecation and micturition, are common. *Involuntary contractions* of a tonic or clonic kind are extremely rare from hemorrhage limited to the cerebral substance.

The more common combinations of symptoms by which the existence of cerebral hemorrhage limited to the medullary substance might be inferred are—

1. *Profound coma, with hemiplegia on one side, of marked intensity, and without rigidity.*

2. *Paralysis of both sides, but one more profoundly affected than the other—a rare occurrence in limited hemorrhage.*

3. *Slight coma, but paralysis hemiplegic and complete.*

*B. Hemorrhage into the Ventricles* cannot in some cases be distinguished from arachnoid extravasation, or in others from effusion into the cerebral substance only, especially when in the vicinity of the ventricles. The cases, however, which are less doubtful are marked by the following characteristics, in addition to the general signs of apoplexy:

1. *The Mental State*.—Coma is very profoundly marked at the commencement, and remains of equal intensity; or the patient, after partially recovering from a slight seizure, is again suddenly plunged into profound coma, from which there is no recovery. This second attack is presumed to indicate the rupture of the hemorrhage either into the ventricles or the arachnoid cavity, from its original site of extravasation in the medullary substance of the brain near the ventricles or near the surface.

2. *Motorial Symptoms*.—*Paralysis* is complete in degree, and is developed simultaneously on both sides; or after having been hemiplegic for a short time it becomes general; when the coma of the second attack above noticed comes on, stertorous breathing is strongly marked. Involuntary evacuations follow. The pupils remain dilated. Deglutition is dangerous and difficult. When the paralysis is general and the coma profound, it is almost a sure sign that hemorrhage has taken place to a considerable extent into the ventricles. *Rigidity* or *tonic contraction* of the muscles is present in many cases of hemorrhage, and in nineteen out of twenty-six cases occurs in the paralyzed limbs; and in about four out of twenty-six cases may be seen in those of the healthy side (Dr.

RUSSELL REYNOLDS). Its presence is a sign of extensive hemorrhage, with laceration of the brain. The most frequent combination of symptoms indicative of hemorrhage into the ventricles may be shortly stated to be *profound coma, with general paralysis and rigidity*.

*C. Arachnoid Hemorrhage* occurs when the extravasation bursts through the pia mater and arachnoid into the space between the membranes, and such cannot be distinguished from the *ventricular extravasation* just noticed. If, however, the extravasation is immediately subarachnoid at first, and of limited extent, it may be approximately diagnosed,—*First*, By the nature of the premonitory symptoms having partaken of meningeal inflammation, such as by severe pain in the head, with impaired Intelligence and power of movement. *Second*, The attack is less sudden than in cases of congestion or of central hemorrhage, and the symptoms are progressively developed.

The following are the combinations of symptoms which indicate the occurrence of subarachnoid hemorrhage:

(1.) *Complete and profound coma without paralysis; or with general paralysis very slightly developed in intensity.* (2.) *Complete loss of consciousness without paralysis; but combined with rigidity or clonic contractions of the limbs.* (3.) *Paralysis of hemiplegic distribution as regards the limbs, but without deviation of the features, the muscles of the face not being implicated.* (4.) *An apoplectic attack without anæsthesia.* (5.) *Imperfectly developed coma with general paralysis.* (6.) *An apoplectic attack, of which the symptoms are somewhat interchangeable or remittent.*

**Causes.**—Among the most frequent causes of apoplexy, especially in some constitutions, is an intemperate use of fermented liquors—a class of substances which powerfully excites, mainly by inducing paralysis of functions, which allows the evidence of excitement to appear as the prominent phenomena (ANSTIE). Alcohol also acts specifically on the heart and arteries, causing not only temporary energetic action of those parts, but also organic alterations in their structure (vol. i, p. 772 *et seq.*). In the latter case the powers of the heart are often permanently augmented, while the coats of the arteries, thickened, thinned, or ulcerated, have their elasticity destroyed, and thus the tendency to hemorrhage in the brain is increased. The excessive use of narcotics, as opium or tobacco, is also supposed to predispose to congestion of the brain, and consequently to cerebral hemorrhage.

The following conclusions have now been arrived at from a comprehensive view of numerous cases: (1.) That in by far the greater number of cases cerebral hemorrhage is due to degenerations of the cerebral arteries as its predisposing cause. In the smaller arteries these consist of fatty metamorphosis, or simple atrophy, with the various forms of consecutive dilatation; while in the larger arteries of the base there is *arteritis*, issuing in ossification, or fatty degeneration, or passive calcification. (2.) A not entirely rare cause are true aneurisms of the large cerebral arteries. (3.) Hypertrophy of the left ventricle will only favor cerebral hemor-

rhage when it permanently increases the normal tension of the aortal system: but this is not the case in compensating hypertrophy of valvular disease of the heart. (4.) In about one-seventh of all cases of apoplexy, neither predisposing diseases of the heart nor of the vessels could be demonstrated (EULENBERG, in Virchow's *Archiv*, vol. xxiv, p. 329; and *Syden. Soc. Year-Book* for 1862, p. 81).

The connection of apoplexy with advanced disease of the kidney, has occasionally attracted the attention of pathologists; and the late Dr. S. Kirkes recently brought forward some facts in proof of the frequency with which the kidneys are found diseased in fatal cases of apoplexy; and which, to a certain extent, explain the connection between the renal and cerebral affections (*Med. Times and Gazette*, 1835, p. 515). In all the cases except one the heart was found enlarged,—an enlargement due to the prolonged disease of the kidney,—and generally in the form of hypertrophy of the left ventricle, without valvular disease. The primary disease is that of the kidney; then the heart and then the bloodvessels become diseased in consequence of the primary affection. The disease of the bloodvessels is chiefly in the form of the well-known yellowish-white thickening and deposit within the coats of the bloodvessels; and Dr. Kirkes believed that the disease of the vessels is in a great measure the result of the continual over-distension and straining to which the arteries are subjected by the unwonted energy with which an hypertrophied heart propels the blood along them: the full force of the hypertrophied ventricle, independent of valvular disease, will be exercised upon the arterial current.

Extremes of temperature are likewise powerful predisponents to apoplexy. In summer the fluids of the body tend to produce turgidity of the vessels in some constitutions, and the tone of the capillaries is impaired; while in winter the cold drives the blood from the periphery of the body to its central organs, and consequently to the brain. Sudden and great vicissitudes of the weather, as they rapidly exhaust the nervous power, are more frequently fatal than the uniform continuance of its extremes.

The powerful effects of *moral causes* in producing this fatal disorder are well known. *Mechanical obstruction* is also a frequent occasion of apoplexy. If an obstacle, for example, be opposed to the course of the blood, as when the valves of the heart are diseased, the blood accumulates in the capillary system generally, and consequently in the brain; or errant clots of fibrin choke up the minute cerebral vessels.

[Thrombosis and Embolism are destined to take a prominent place in cerebral pathology, and particularly in softening of the brain, which, it is believed, will be found to be much less frequently a primary affection, depending upon an inflammatory process, than has been generally supposed. That both autochthonous and migratory clots are capable of producing apoplectic symptoms, by the occlusion of one of the cerebral arteries, or the capillaries, and that, too, when the brain-substance is apparently sound, there is no doubt. There is always, as a consequence, paralysis of motion in the side opposite to the obstructed vessel; in many



cases the general sensibility is unimpaired, sometimes lessened, but rarely, if ever, completely absent, and never perverted. The loss of consciousness is slighter, and is sooner gotten over than after cerebral hemorrhage; and the mind is less affected, sometimes not at all. Sight and hearing may be disordered, and aphasia sometimes happens. Death may follow closely upon the apoplectic attack, or the serious symptoms may amend, to reappear at the end of a few days or even weeks, when they are fatal; or, the cerebral symptoms may remain for awhile stationary, and death be caused by the concurrent disorder,—cardiac disease, gangrene of the extremities, Bright's disease, &c. The middle artery of the brain would seem to be the most common seat of emboli, but they have been found in the anterior, basilar, and vertebral, and serious apoplectic phenomena have followed carotidean emboli. Hasse gives two cases where the clot extended into the common carotid, and Dr. Markham records a case (*On Diseases of the Heart*), in a woman of fifty, of plugging up of the innominate, right common carotid, left internal carotid, and middle cerebral arteries, with apoplectic symptoms, hemiplegia, and death. Dr. Abercrombie mentions an instance of obstruction of the basilar artery. On this subject the excellent thesis of Dr. Lancereaux,\* and the recent essay of MM. Prevost and Cotard,† may be read with advantage.]

Apoplexy is still more common when the aorta is diseased, the force of the heart, unchecked by the elasticity of that vessel, acting directly on the brain, so that its vessels often give way from this cause. *Mechanical violence*, also, often produces apoplectic effusion. Thus, concussion of the brain, however slight, always produces more or less congestion of that organ; and, if severe, effusion may take place below the *dura mater*, or between the membranes, or into the substance of the brain, which may be extensively ruptured.

Apoplexy has been known to occur even in childhood. Billard gives the case of a child three days old that died apoplectic from effusion into the left hemisphere and about the lateral parts of the *corpora striata*. Serres saw a similar case in a child three months old. Apoplexy, however, is extremely rare till puberty, and only a few cases are met with before twenty. It is not unfrequent between thirty and fifty, while after fifty it is one of the most frequent causes of death. There are many circumstances which favor the disposition to apoplexy in old age. At that period the capillary system becomes impaired in most organs, and thus the veins are filled with a greater quantity of blood, or they become congested. The cerebral arteries also are often diseased; the heart has frequently acquired an abnormal power, driving the blood with great violence, and with an increased momentum, towards the brain, while the lungs have their functions so impaired that the blood is only imperfectly oxygenated; and all these are causes of congestion, and of tendency to rupture of the vessels of the brain.

Both sexes are liable to this affection and in nearly equal propor-

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\* [De la Thrombose et de l'Embolie Cerebrales, considérées principalement dans leurs rapports avec le Ramollissement du Cerveau. Par E. Lancereaux. Paris, 1862.]

† [Etudes Physiologiques et Pathologiques sur le Ramollissement Cérébral. Par MM. J. L. Prevost et J. Cotard. Paris, 1867.]

tion. Those most liable to attack are the florid in complexion, of short-necked conformation, with prominent eyes, broad chests, and protuberant bellies, and sometimes enormously fat, especially if high livers, sedentary, and indolent. Many thin persons, with spare, long necks, however, frequently die from apoplexy; but it is probable, that in these cases the heart or large vessels have been diseased.

The act of digestion, or rather fulness of the stomach, appears to predispose to apoplexy. Numbers are attacked after dinner. Sleep, also, associated with a temporary congestion of the vessels of the brain, is another predisposing circumstance. Thus, of 176 cases examined by Gendrin, 97 had been attacked during sleep. In the sleep of a healthy person the brain is anæmic (DURHAM).

Many diseases, or conditions of the body, predispose to apoplexy, as mania, epilepsy, suppressed hemorrhoids, amenorrhœa, and especially the "*turn or change of life*."

[**Diagnosis.**—The differential diagnosis between cerebral hemorrhage and cerebral softening, with or without a clot, may be generally made out by the suddenness and rapidity of the paralysis, in softening without loss of consciousness as a rule, though this sometimes happens, but is of short duration. A clot, when it exists, causes some confusion and slowness of mind, diminished sensibility, and greatly impaired, or total loss of, motor power, but not loss of consciousness, or coma. Whenever the coma is sudden and complete, hemorrhage may be affirmed, and, usually, of considerable amount (RECAMIER, TODD, TROUSSEAU).

When coagula form in the large bloodvessels supplying the brain, the symptoms may not be distinguishable from those of cerebral hemorrhage (DICKINSON, *St. George's Hospital Reports*, vol. i).]

**Prognosis.**—Apoplexy is always a grave disease, and the more grave in proportion, generally, as the respiration is stertorous and the deglutition difficult. Each succeeding attack is more dangerous than the former. The practitioner should also be guarded in his prognosis till after the first week or ten days, lest inflammation should come on, or a fresh attack destroy the patient. Popular opinion (and it is useful in pronouncing an opinion to know what are popular beliefs) supposes the patient to suffer three apoplectic attacks, the first being mild, the second followed by paralysis, while the third is fatal. It is only in a few instances that this number is exceeded.

In the congestive form of apoplexy, if active and judicious measures of treatment are employed, the recovery may be rapid and complete; but if this is not the case, there is a liability to a more aggravated form, from which nothing is more variable than the time of recovery. In a very few instances the patient is restored in a few days, or in a few weeks, or in a few months, but more commonly the lesions of motion are permanent, or nearly so. In general, however, some slight improvement takes place even in the worst cases, so that the patient recovers some use, first of his leg, and then, perhaps, of his arm, so that he is able to walk with a

“straight leg” and a dragging foot. The use of his arm returns more slowly and more imperfectly.

[According to Trousseau, the prognosis is unfavorable when the arm regains power more rapidly and more completely than the leg, this unusual fact occasionally happening. Several such cases are mentioned by Trousseau (*Clin. Méd. de l'Hôtel-Dieu*, t. ii, 1865), and by the late Dr. Todd (*Clin. Lect. on Ner. Dis.*); and Dr. Bazire, the translator of Trousseau, relates one, in the practice of Dr. Ramskill, of a gentleman who, after an attack of hemiplegia, regained complete use of his affected arm, whilst his leg remained paralyzed, but in a few months all the symptoms worsened, and he soon died.

Another sign of prognostic value is insisted upon by Dr. Trousseau, as the result of his experience; it is where, after cerebral hemorrhage, the fingers of the paralyzed hand continue firmly flexed into the palm through contraction of the flexors; the use of the hand is permanently lost, and no hope of improvement should be held out. This statement is perhaps too absolute. Where this deformity of the hand has existed for any time atrophy and degeneration of the extensor digital muscles are very likely to have happened, and any treatment is most generally of but little or no avail.]

This recovery is often preceded and accompanied by very severe pains, especially of the upper extremity, marking the still irritated state of the brain. The limb, however, uniformly wastes, and its vital powers are so impaired that, if inflamed, the inflammation seldom terminates by resolution, but has a great tendency to gangrene, while cicatrization is slow and difficult.

The commonest occurrence is the perfect recovery of the mental faculty, and the progressive but much more gradual return of motion and sensibility. Paralysis of motion sometimes persists in groups of muscles, such as those of the tongue, the forearm, or hand. Sensibility is generally first restored, then the motion of the lower extremity, then the arm, and lastly that of the forearm and hand. Discordant opinions are entertained relative to the influence of the electric structures on the paralyzed muscles. The late Dr. Marshall Hall was of opinion that the muscles of the non-paralyzed limb were the more irritable. The late Dr. R. B. Todd entertained precisely the reverse opinion. These were the highest authorities of their day in this country. Duchenne draws attention to two conditions of paralysis after apoplexy—namely, one directly dependent on the central lesion, the other the mere result of inaction for some time. In the former case there is contraction of the muscle as the result of increased spinal action, uncontrolled by the central ganglia of the brain—in the latter form the muscles are flaccid. Active tonic contraction of the muscles, distinct from simple shortening of the flexors, he considers as indicating inflammatory action in the walls of the cyst; but this distinction is one which requires corroboration or contradiction by subsequent observation (Dr. RUSSELL REYNOLDS).

No doubt the principal adverse circumstances attending recovery from apoplexy are, that although the patient appears to be doing well the first few days after the attack, yet towards the close of the first week the brain, irritated by the presence of the clot, inflames

and softens, and thus induces another and a fatal attack of apoplexy. Should the patient, however, survive this dangerous period, he may continue to live many months, or years, according to his age; but he is generally at length cut off by a fresh attack of apoplexy, or his brain ultimately inflames and softens, and he dies in a typhoid state, as already described.

Although it is the general rule that the patient, on recovering from the attack, has the good fortune to recover all the faculties of his mind, yet his Memory is not unfrequently impaired, often to such a degree that he has forgotten all dates, the names of his friends, or even the names of things. Broussonet, Professor of Medicine at Montpellier, had entirely lost the remembrance of all substantive nouns, and another case is on record in which the patient lost the recollection of all his adjectives. In some instances the power of association is also so destroyed that although many remember both names and things, they are unable to connect the thing with the proper word, so that they call that which is cold, hot; or speak of night when they mean day; or call a coffee-pot a wash-hand basin. Others, again, have forgotten how to read, and the power thus lost either returns suddenly, or they are obliged to learn *de novo*.

The attention generally is very greatly impaired, and the patient is no longer able to transact business; or if he begins a sentence, is unable to finish it, or he repeats the same idea over and over again. The passions also are little under control, for while some weep like children, others laugh immoderately, and all are easily terrified, or otherwise easily influenced.

All these circumstances must be remembered in giving a prognosis regarding an apoplectic patient.

**Treatment.**—The patient, if seen during the fit, may be bled if the tendency to death is by *coma*, and if the pulse be full, or hard, or thrilling; if the vessels of the neck be congested, the heat of the scalp increased, and if the face be full and turgid. The state of the heart ought to be examined into first. If its action be vigorous, its sounds normal, and the heat of the skin preserved, bloodletting is still more required. Slow and deep respiratory movements, with stertor, add greatly to the necessity of immediate venesection; and the beneficial action of the remedy is shown by the pulse becoming fuller, stronger, and more regular; and the bleeding may be allowed to proceed till an obvious impression of this kind is made, or until the pulse begins to flag. Large bleedings are to be avoided. Ten ounces will generally be sufficient, if taken at the first outset of the attack; but if the pulse does not improve, and other symptoms remain unrelieved, sixteen to twenty ounces may be taken. The indications for bloodletting being thus strongly pronounced, “we ought not to hesitate to open a vein, regardless of the age of our patient” (MACLACHLAN). The bleeding ought to be permitted to flow from a large opening, in order to relieve the congestion, to check, if possible, a further effusion of blood, and to divert its active flow from the head. The quantity taken should be proportioned to the degree of stertor, and to the powers of the patient. The head and shoulders should be raised

while the blood is flowing. But, on the contrary, if the pulse be small and slow, feeble, or almost imperceptible, the skin cold and clammy, with a tendency to death by syncope, then no advantage is to be gained by the abstraction of blood at this time and in this way. Bloodletting is therefore contraindicated under the following circumstances: (a) anæmia, (b) aortic valvular disease, (c) in cases commencing with syncope.

If the bloodletting is not followed by some degree of consciousness, it may be inferred that the amount of blood effused is considerable, and that the patient in all probability will not recover. Still, perhaps, an additional chance may be given by applying to the head cold cloths, or crushed ice in a bladder, leeches to the temples, and mustard cataplasms to the feet; also by placing a drop or two of croton oil on the tongue, and by throwing up a cathartic enema of castor oil or other purgative. At one time every case of apoplexy was treated by bloodletting; and statistics prove, of such indiscriminate practice, that the more freely the blood was taken away the greater was the mortality (COPMAN). Some Physicians are opposed to any considerable bleeding during the fit, considering that the bony structure which contains the brain removes all atmospheric pressure so entirely as to cause that organ at all times to contain an equal quantity of blood. A space within the head for a very sensible expansion and contraction at each pulsation of the heart is opposed to such a belief, while post-mortem examination shows the brain to contain very different quantities of blood, being sometimes gorged and sometimes blanched of that fluid. These facts distinctly show that some arrangement exists for regulating the quantity of blood sent to the brain; and we ought, therefore, in a disease of this moment, to follow the dictates of a long experience, rather than the conclusions of a fallacious reasoning.

When emetics are now spoken of, it is generally with the view of condemning them. The physiological phenomena which attend their action increase the determination of blood to the head, as is now well known; and the action of vomiting may be fatal in such cases. After the patient has in some degree revived, and the congestion removed, some time for the absorption of the blood effused should be allowed before deciding upon the future treatment. Any very large depletion after that point is gained would rather facilitate extravasation than prevent it. A few hours, then, having elapsed, the conduct of the practitioner should be guided by the occurrence or not in the patient of *pain of the head*, which may be taken as a measure of the fulness of the brain, and its tendency to inflammation. If, therefore, there be pain in the head, ten to twelve leeches should be applied from time to time till that symptom is entirely relieved; or, supposing the pulse to be full and strong, and the patient free from headache, yet, under these circumstances, leeches should be applied to the head, to subdue that reaction which so generally takes place from the fourth to the seventh day.

The further treatment of the case is by moderately purging the



patient, both as a means of relieving the head and of improving the secretions of the alimentary canal, which are often black and fetid. Active purgatives generally do good. Five grains of *calomel*, with a drachm of *compound jalap powder*, given as soon as the patient can swallow, and followed up by *black draught*, or by an ounce of *sulphate of magnesia* with *camphor mixture* every four or six hours, and continued, according to its effects, for a greater or less length of time, are the best means we have for promoting recovery, and for preventing a relapse. These prescriptions are recommended on the supposition that the attack has been associated with simple plethora. In many cases, however, it is a consequence of hypertrophy of the heart, without valvular disease. And in such cases less blood should be taken, and eight to ten minims of *digitalis* may be added to each dose of the purgative medicine. If the power of swallowing is in abeyance, then three or four drops of croton oil should be put on the back part of the tongue, and stimulating enemata thrown up the rectum. The following are recommended by Dr. Tanner:

1. *Enema of Turpentine and Castor Oil*.—R. Olei Ricini, Olei Terebinthinæ, āā ℥iss.; Tincturæ Asafœtidæ, ℥ij; Decocti Avenæ, ℥xij; misce, fiat enema. To be thrown up the rectum by means of a long stomach-pump tube.

2. *Croton Oil Enema*.—R. Olei Ricini, Olei Terebinthinæ, āā ℥j; Olei Crotonis, ℥vj; Decocti Avenæ, ℥iv; misce, fiat enema.

Although turpentine is objected to by some, on account of the intoxicating effects which it is sometimes apt to produce, it is nevertheless an efficient remedy where torpor and insensibility exist. It is of great importance to empty the rectum and lower bowel. In cases where the ability to swallow is lost, a drop or two of croton oil with five grains of calomel, rubbed up with fresh butter, may be laid on the back of the tongue.

If the intestines are distended by gases, an enema of castor oil and rue may be given:

3. *Castor Oil and Rue Enema*.—R. Confectionis Rutæ, ℥j; Olei Ricini, ℥j; Tincturæ Asafœtidæ, ℥ij; Decocti Avenæ, ℥vij; misce (*Practice of Medicine*, 4th edition, p. 653).

[With a better knowledge of the nature of apoplectiform phenomena there has come the conviction that general bloodletting should not be used as a matter of course,—once a treatment unfortunately too common. The late Dr. Todd wrote: “Bear in mind that in a large number of cases,—probably the majority,—there is in reality no cerebral congestion, and that the hemorrhage is not of a kind likely to be stopped by taking away blood. If you find your patient has been of intemperate habits, is laboring under organic disease of the heart and arteries, is of gouty or rheumatic constitution, then hesitate much before you deplete by bleeding” (*Clinical Lectures on Nervous Diseases*).

Of 159 cases of apoplexy analyzed by Mr. E. Copeland (*A Collection of Cases of Apoplexy*, London, 1845), of which 129 were bled, 51 recovered [from the attack] and 78 died; of 85 bled generally and copiously, 28 recovered, and 57 died. Of the 26 not bled, 18 recovered, and 8 died.

The writer's views and experience coincide, to a great extent, with those who deprecate an active and spoliative treatment in cerebral hemorrhage. Dr. Trousseau says: "I not only abstain from all energetic remedies when the symptoms of cerebral hemorrhage are slight, but I refrain from doing so in very grave cases; in fact, in all cases of apoplexy. If I do not have recourse to bloodletting, purgatives, or revulsives, it is because experience has taught me that patients do better without them; for I do not see how those methods of treatment can be of any use, since the hemorrhage is an accomplished fact when we are called upon to note its symptoms. What influence can be exerted on a foreign body in the shape of extravasated blood, by bleeding from a vein of the arm, or of the foot, or from the jugular, or by dividing an artery, or by cupping, or leeching? Of what use are purgatives or revulsives? These means, it is said, empty the vessels, and thus facilitate the absorption of extravasated blood; and that they antagonize the cerebral congestion, which, according to the practitioners who recommend them, precedes, accompanies, or follows, the extravasation of blood, and lessens the amount, or the risk of a second attack. With regard to the first point we may well doubt whether there is any difference between cerebral and other hemorrhages; how can we act more powerfully on effused blood in the brain than on extravasation under the skin? As to the second point, that bloodletting is required to arrest the *molimen hæmorrhagicum*, it is very doubtful. The part played by congestion seems to me to have been much exaggerated, and I do not think that the necessity, nay, more, the usefulness of the measure, have been proved. Do we know well the organic conditions under whose influence cerebral hemorrhage is produced? Is not congestion rather an effect than a cause of the sanguine extravasation? What influence then can bloodletting exert on this sequential hyperæmia, when it can have none on the foreign body, the clot? Far from being useful, I believe bloodletting to be injurious, and favors, instead of hindering, congestion. Apoplectic phenomena are in some cases more allied to syncope than to congestion, and then bleeding is contraindicated. What treatment do I adopt in cases of apoplexy? Instead of bleeding my patients, and putting them on low diet, and keeping them in bed, I save their blood, recommend them to get out of bed, and sit up, and I feed them. I am convinced that I get better results than when I treated them more actively,—bled them, starved them, and kept them in bed. Now, in cerebral hemorrhages of a certain amount, there is nearly always, about twenty-four hours after the attack, some febrile reaction; the pulse is hard and frequent, the face flushed, the respiration quickened and laborious, and the skin hot. I used to bleed here, but I am satisfied that it did harm, and that without bloodletting the febrile symptoms cease sooner. Very recently, one of my colleagues, Professor Monnet, has stated, that for a long time he has given up active treatment in apoplexy, and that instead of using lowering means, he feeds his patients, and gives them wine. Since I have adopted the plan of keeping up the strength of my patients by giving them food in moderation, I find that the unpleasant symptoms disappear more rapidly than when my treatment was more active" (*Clinique Médicale*, t. ii, 2ième ed., 1865).

It should, too, be borne in mind that cerebral hemorrhage commonly happens in connection with tissue-changes in most of the organs of the body, as well as in the bloodvessels, and that usually it is secondary or intercurrent, and not primary or essential. The morbid systemic condition is to be considered both with reference to prognosis and treatment.]

All apprehension of a relapse being at an end, the patient is in general most willing to believe that the palsy which may remain is a mere local disease, and to submit to any treatment for its removal. The ancients applied the actual cautery to the extremities, to the coronal suture, or to the occiput, but without any beneficial success. The moderns have had recourse to blisters, to friction, to electricity, and to strychnine; but every attempt to act locally on the muscular system is prejudicial so long as any central irritation exists. (See "Paralysis.") Active and passive exercise of the muscles are remedies highly beneficial.

**Dietetic Treatment.**—The diet of the patient should be low, till all apprehension of a relapse is past, and limited to milk, boiled vegetables, light puddings, and fish. At no subsequent period ought he to indulge in a full animal diet, or to drink undiluted wines. At the same time, too lowering a regimen is to be avoided, as thereby the irritability of the system and the heart's action generally is increased. All the causes of the disease already fully referred to should be avoided, counteracted, or overcome.

#### HÆMATOMA OF THE DURA MATER

**Definition.**—*Sanguineous flattened masses composed of fine layers of fibrin spread to a greater or less extent over the dura mater, accompanied by small extravasations which are converted into pigment. By repetition of the process numerous layers come to be deposited one upon the other. Numerous and large bloodvessels form in these layers; and from these vessels renewals of the hemorrhage occur. The disease is chronic, and terminates, after continued cephalic suffering, generally suddenly, with symptoms of apoplexy.*

**Pathology.**—These tumors of bloody formation seem to occur in connection with more or less inflammation of the *dura mater*. The effusion of blood between the *dura mater* and the arachnoid is not in most cases a primary occurrence, but the productive results of inflammation first occur; and the new growth is developed into a fibrous membrane traversed by a copious network of new-formed bloodvessels. It is from the rupture of these vessels that extravasations proceed (VIRCHOW, WEBER). The lesion is sometimes described as due to *intrameningeal* apoplexy, with false membranes on the *dura mater*; but the false membranes, which are the result of the chronic inflammation, precede the apoplectic phenomena. The *hæmatoma* often attains a considerable size. It may be from four to five inches long by two and a half inches broad and one-half to three-quarters of an inch thick. It is generally of a flattened circular form, with a central elevation. The long diameter is parallel to the falciform process. The tumor generally occurs on one side only; or, if bilateral, one is more developed than the other. The affection appears to occur only in the adult, and generally after the age of fifty. In recent cases very fine layers of fibrin are found to a greater or less extent spread over the surface of the *dura mater*. By repetition of the inflammatory process, numerous layers of fibrin

become deposited one upon the other; and much more numerous and larger bloodvessels form in these layers than are to be met with in the *dura mater* itself. From these new-formed vessels the hemorrhage proceeds which gives rise to the formation of the *hæmatoma*, and its cystic inclosure is formed by the extravasation taking place between the layers of the false membrane (VIRCHOW).

**Symptoms** may extend over several months, and consist in general weakening of the Memory and of the Intelligence; the occurrence of giddiness; and of continuously intermittent general or local pain in the head. At a later period an aggravation of all these phenomena occurs, with transitory losses of consciousness from the momentary arrest of cerebral circulation. Somnolence and apathy prevail, with weakness, and generally one-sided paralysis of the extremities, which may soon disappear.

**Treatment**—Is more or less expectant. The newly-formed membranes tend to undergo retrograde change, and thus finally disappear. To effect this end is therefore the object of any rational treatment, which must be based on the special history of the individual case, especially as to the previous existence or not of syphilis.

### SECTION III.—GENERAL DISEASES OF THE NERVOUS SYSTEM CHARACTERIZED BY EXALTED, PERVERTED, OR SUSPENDED FUNCTIONAL ACTIVITY.

This group may be illustrated by the diseases known as *chorea*, *epilepsy*, *hydrocephalus*, *insolatio*, and *insanity*.

#### CHOREA—SYN., ST. VITUS'S DANCE.

LATIN, *Chorea*; FRENCH, *Chorée*; GERMAN, *Veitstanz*—SYN., *Chorea*; ITALIAN, *Corea*.

**Definition.**—*An irregular convulsive action of the voluntary muscles, of a clonic kind, especially of the face and extremities, they being either entirely withdrawn from the control of Volition, or but little under the direction of the Will.*

**Pathology.**—The history of this disease is a sad picture of superstition. As late as the close of the fifteenth century it does not appear to have been studied by physicians, but was supposed to depend on supernatural causes or “demoniacal possession.” In Germany it was said for two centuries to have been epidemic, and the patients, probably many of them maniacs, were wont to join in frantic dances; and as late as 1673 they went in procession to the church of some favorite saint, of whom St. John, St. Guy, and St. Vitus were the most reputed. Hence the name of St. Vitus's Dance, by which the disease is sometimes described. As physical remedies were supposed to be unavailing in such a disorder, the priests said masses, sung hymns, and sought by such means to exorcise the foul fiend.

The morbid appearances of the body which have been observed

in cases of chorea have not as yet thrown much light on its pathology. Sydenham, Cullen, Rostan, Bright, Stoll, Pinel, and others, who have had frequent opportunities of examining cases of this disease, failed to detect any other morbid appearances than those which were commonly seen in other affections of the brain and spinal cord. Accordingly many various pathological views are entertained regarding this disease, which may be classed as follows: (1.) By some pathologists it has been regarded as a disorder entirely functional or dynamic, and independent of organic change. (2.) It is also believed by some, and not without good reason, that the blood, at all events in some cases, is primarily diseased, or becomes so constitutionally, the precise nature of the change being as yet unknown. (3.) Associated with some other diseases, whose pathology is better known, it has been regarded either as a concomitant feature, or as a necessary consequence of their previous existence: such, for instance, as *rheumatism*, and *diseases of the heart*.

Much evidence has been brought forward in favor of the humoral or rheumatic character of the disorder. Dr. Copland has the merit of having been the first to indicate the complication of chorea with that class of diseases (*London Medical Repository*, vol. xv). His views have been subsequently confirmed by Drs. Prichard and Roeser, and more recently by the elaborate researches of Dr. Begbie and Dr. See [and Henri Roger]. Numerous instances have also been adduced by Andral, Bouillaud, Bright, Mackintosh, Watson, R. B. Todd, Kirkes, and others, in which diseased conditions of the heart and pericardium have been attended with, or have given rise to, spasmodic diseases of the nature of *chorea*, *paralysis*, *mania*, or *dementia*; and the evidence of these writers is amply sufficient to prove that a considerable number of individuals affected with *chorea* have suffered from *cardiac* or *synovial rheumatism* previously. Dr. Sidney Ringer has observed one or two cases of chorea with considerable elevation of the temperature, but without any of the ordinary evidences of *rheumatism*. He believes such cases may tend to show that even in those in whom there has been no evidence of previous rheumatism, it nevertheless probably existed, but was latent in respect of all the symptoms except elevation of the temperature. But it is unquestionable that all have not so suffered; and, indeed, the history of the majority of the cases clearly shows that chorea has a more intimate connection with mental disease, such as imbecility, or even insanity, than with perhaps any other morbid state. The presumed blood-condition, similar to that which exists in *rheumatism*, can only thus be regarded as one of the many occasional causes, the real essence of the disease being a perverted nervous function, as Dr. Reynolds writes, and with whose remarks I am pleased to find the views here stated agree.

Cardiac affections are apt to supervene in cases of chorea, differing in kind as well as in degree, namely,—(1.) Rheumatic endocarditis, or pericarditis, resulting in organic change; (2.) Functional derangement and cardiac murmurs, due to an impoverished condition of the blood (ROMBERG); (3.) A chronic affection of the heart itself, “evidenced by the existence of a systolic murmur at the left



apex, which cannot be referred to inflammation or organic change of the mitral valve, which has not the usual accompaniments of a hæmic murmur, but which does seem plausibly ascribable to disordered action of the muscular apparatus connected with the valve" (WALSHE).

[Dr. Henri Roger, who has carefully studied, with large opportunities, the relationship of chorea, rheumatism, and heart disease, believes it is at its period of decline that articular rheumatism in young persons has most tendency to be complicated with chorea; and this complication is more common in those vague and mild cases, so apt to be called "growing pains."

There is an antagonism between the violence of the phenomena of the two affections. Very acute and general articular rheumatism is, as a rule, associated, either from the beginning or during its course, with cardiac disorder, and not with chorea; and chorea, where it does happen in such cases, is partial, not severe, and of short duration. The milder forms, on the contrary, are usually complicated with the most obstinate and violent attacks of chorea.

In those cases where the two disorders, both liable to relapse, alternate or are coexistent, the intensity of the phenomena of each is in inverse ratio to the intensity of either one (*Archives Gén. de Méd.*, vol. i, 1867).]

As in the case of most diseases expressed during life merely by perverted functional activity, morbid anatomy is often at fault. Our means and appliances for the accurate appreciation of nervous lesions especially are but rude compared with the fine and delicate textures with which we have to deal. When patients suffering from chorea have died, and the brain has been carefully examined, the most experienced observers have failed to detect any lesions by which the occurrence of the symptoms could be explained. This negative evidence no doubt points to some morbid condition of the blood as an essential element in chorea. Rostan had once an opportunity of examining a woman upwards of fifty, and who, from her childhood, had labored under chorea of the whole of the left side of the body, and of which the limbs were atrophied. "I expected to find," he says, "atrophy of the right side of the brain, but there was nothing morbid; at least, after a most careful examination, I could see nothing." Dr. Bright has given one case which he had an opportunity of examining, and which gives equally negative results. It was that of a young woman aged seventeen, who had formerly labored under this disease. She had been free from it for four years, when she formed an attachment, was forsaken, was attacked with chorea, and died. The attack was of great severity; she tossed herself about in all directions, bit her tongue, and was with difficulty in any degree controlled. On examination, there was a slight effusion into the arachnoid cavity, more *puncta cruenta* than usual, and five or six bony plates opposite the *cauda equina*—phenomena common in many diseases of the brain or cord, and of course incapable of having any pathological significance assigned to them in relation to chorea. It is equally impossible to fix upon any other organ or part of the body in which anything like constant

structural lesions have been observed, susceptible of being associated in explaining the nature of chorea. The structures which most obviously manifest disordered action during life are the nerves and muscles; and for the following reasons we are led to believe that they are maintained in their disturbed and excited state by some morbid condition of the central parts of the brain, and not of the spinal cord, either directly or by reflex action.

1. *Clonic spasm*, of the incessantly repeated character peculiar to chorea, is not a phenomenon of persistent spinal irritation; while *tonic spasm* is a mark of such a condition (REYNOLDS).

2. The movements can generally be in some measure controlled by the Will, unless they are very severe; and even then they are so controlled to some extent (REYNOLDS).

3. The spasmodic contractions cease during sleep, whereas phenomena of an excito-motor character are increased by the removal of volition. Fixing the attention also to some other object likewise diminishes the intensity of choreic movement (REYNOLDS).

4. The special occasions of increase or of induction of choreic movements are the attempts at volitional action and emotional changes (REYNOLDS).

5. The phenomena of chorea during life, in accordance with the views expressed by Drs. R. B. Todd and Carpenter, which are now very generally received, tend to refer the exciting cause of the disease to changes going on in the central ganglia of the brain, such changes being expressed in a healthy state through "volition, perception, or emotion, or the balancing and co-ordinating of movements."

6. Experiments on living animals, and observations in morbid anatomy, tend to prove that injury to the *optic thalami* is productive of considerable disturbance to the movements of the body.

7. An opportunity was afforded me, when Pathologist to the Glasgow Royal Infirmary, of examining carefully a case of chorea, which terminated fatally after a most violent attack, the acute symptoms lasting ten days. The result of the examination showed some decided changes in the *corpora striata* and *optic thalami*, sufficiently indicated by the following observations:

"The specific gravity of the *corpora striata* and *thalami optici* was different on the two sides of the brain: those on the right side were of the specific gravity of 1.025, those on the left side of 1.031; and this difference appeared from the hydrostatic experiments, as well as from those made with the gravimeter, confirming in some measure the accuracy of the general result.

"The vascularity, also, of these central parts of the brain, when compared with the gray matter of the spinal cord, which was healthy, was so well marked as to leave no doubt of its abnormal increase.

"Microscopic examination confirmed the existence of increased vascularity, for numerous capillary vessels, in unusual abundance, existed in every section examined. Some of these were irregularly dilated, as in a varicose condition, and were filled to a greater or less extent with the red corpuscles of the blood. The amount of granular substance in these parts of the brain, on both sides, appeared to be greater in proportion to

the fibrous substance than in the same parts of healthy brain with which I compared them."—("Contributions to Pathology," *Glasgow Medical Journal*, No. I, 1853.)

The late Dr. R. B. Todd remarked, that "further observations on this subject are greatly needed, and will no doubt throw great light on the pathology of chorea and other allied affections."

Dr. Walshe, the Emeritus Professor of Medicine of University College, was the first to call attention to a not unfrequent occurrence in the course of chorea; namely, a high specific gravity of the urine. The high density of the urine is most marked where the choreic movements are most active; and no doubt it indicates increased waste of tissue, consequent on the disturbed state of the muscles and nerves (*Lancet*, Jan. 27, 1849, p. 85). In the acute case of chorea so carefully recorded by Dr. Walshe, four phases were observed in the characters of the urinary discharge, namely: During the first five days it was "*febrile*;" that is, of high specific gravity, deep brownish-gold color, strong urinous odor, and depositing lithates in abundance. *Second*. There came a period during which a great excess of urea gave a special character to the fluid, while alternating improvement and recrudescence marked the course of the chorea. This superabundance of urea is, in the present state of our knowledge, referred to the muscular waste entailed by the constant convulsive movements. *Next*, there appeared oxalates in the urine passed on the twenty-sixth day, on which day the improvement in the case was so marked that the child might have been considered convalescent. *Subsequently*, an abundant precipitation of phosphates took place, the indubitable result of previous nervous waste.

These observations, originally made by Dr. Walshe, were subsequently confirmed by the late Dr. Todd, and by Dr. Bence Jones.

In a case recorded in his most interesting *Clinical Lectures on Paralysis, Disease of the Brain, and other Affections of the Nervous System*, where the urine was carefully examined from day to day, by Dr. Todd, the density of the urine was shown never to have fallen below 1.019, and frequently reached 1.030, and once was found as high as 1.035. As the patient improved in health, the urine fell in specific gravity, but was never below 1.019. Lithate of ammonia was nearly always present, and oxalate of lime was frequently found mixed with it. An excess of urea was also frequently present. In another case he records the specific gravity of the urine as ranging between 1.030 and 1.040, and afterwards falling to 1.020 and 1.022. Generally speaking, he found the density of the urine highest in those cases in which the movements were most general and most active, and falling steadily with their diminution, and with the restoration of a greater controlling power on the part of the patient.

**Symptoms.**—Chorea principally consists in singular and involuntary movements of one or more limbs, which prevent the patient from being able to lay hold with certainty of any given thing, or to carry that object, be it a spoon or a glass, with any certainty to

his mouth, or to any other place. These symptoms are developed gradually, reach a certain point of intensity, remain at such a point for a variable period, and it is often a long time before they subside, and all traces of them disappear. Premonitory symptoms are neither frequent nor characteristic; but a certain susceptibility to nervous disturbance, and irascibility of temper, are not uncommon. General ill-health is not unfrequent, arising from various causes, as delay of menstruation, and constitutional morbid states, such as rheumatism, or the existence of some zymotic disease. The commencement of the symptoms is often at first insidious, but more commonly gradual, and sometimes sudden. They consist at first simply of restlessness, or of hurried and somewhat clumsy movements. The left side and the upper limbs are frequently affected first; but subsequently the whole body is involved. The lower limbs are generally as much affected as the upper, and the patient can with difficulty walk in a straight line, or, if he does, it is always by a series of movements which tend towards the object, counteracted by another series which altogether diverge from it,—his feet turning in and out, upwards and downwards, in every possible direction. The muscles of the face and neck are sometimes seized with this species of convulsion, when the head is not only tossed about, and the mouth contorted into the most singular grimaces, but it may require two or three persons to feed him—one or more to hold him, and another to watch the proper moment to pop the food into his mouth. Sometimes the motor nerves of the fifth pair are affected, and then the jaw closes with a loud snap, or the articulation of voice is affected, or the effort of swallowing difficult.

Thus the essential phenomena of chorea are motorial, consisting of spasmodic involuntary contractions of the muscles, which have been thus classified by Dr. Reynolds: (1.) *Clonic Spasms*; of great frequency, unattended by pain, resembling the restless movements of a child who has been irritated or put out of temper. Such spasms occur independently of any attempt at voluntary movements, and are in slight cases almost unobserved. (2.) The patient is agitated by all sorts of odd motions, and has often a vacancy of countenance which gives him a fatuous appearance. These symptoms are constant during the day, but during sleep they generally cease altogether. [Of 158 cases observed by Sée, there were but 6 exceptions, and in these the muscular disorder was at its height.] They affect both sides, as a rule, and in a very few cases one side only. The patient is then said to labor under *hemichorea*. The child's health is generally good; his pulse natural; and his bowels, though occasionally constipated, are by no means uniformly so, but for the most part act regularly. The spasms are generally increased by Emotion, and, while they persist during the day, disappear during sleep. The heart acts regularly, probably owing to the anæmic state generally associated with chorea; [and palpitations are complained of, with increased cardiac impulse. According to Sée, temporary hypertrophy and dilatation are sometimes

met with.] Dr. Addison describes a bellows murmur, often mitral, but sometimes aortic, and probably due to the same cause.

**Causes.**—The disease frequently attacks children otherwise in good health, and without any obvious cause. When any cause is assigned, it is usually terror. Somebody has pretended to cut off the child's head, and perhaps has drawn the back of a knife across the throat; or a person dressed in a white sheet has personated an apparition. The symptoms have been known to follow the fright in a day or two, at other times about three or even six weeks have elapsed before the disease became manifest. The causes producing this affection, however, are generally referred to mental impressions. A woman in the fourth month of her pregnancy had a frightfully disgusting object thrown at her bosom. She continued for two months in a state of extreme nervous illness from this circumstance, but recovered, and went her full time, remarking, however, that the child was extraordinarily lively in the womb, and that she was often overcome with the sensations it produced. At birth, the child (a girl) displayed the writhing motions of chorea, and continued to suffer throughout life. When she was about thirty years of age she had the appearance of an elderly child, with a head remarkably small, and a mind hardly removed from complete idiocy (MAYO).

Chorea is limited, or nearly so, to early life, and is rarely seen after twenty. Dr. Heberden states it to be most frequent between the ages of ten and fourteen, and also that it is more common in the female than in the male, three-fourths of the patients under his care having been females. Dr. Rufz regards the ages from five years to fifteen as the most liable to chorea; and that girls are three times more frequently sufferers than boys. Dr. Todd's experience shows that chorea generally occurs between nine and fifteen years of age. It is really a disease of childhood; and although symptoms somewhat resembling those of chorea are sometimes seen at the adult period of life, and at more advanced ages, still such cases are exceptional; and it may be questioned whether they are due to exactly the same morbid condition as that which gives rise to the ordinary clonic convulsions of early life. [Dr. H. Roger has published a case of chorea in a woman aged 83, and Dr. Max Simon another at an advanced age (*Bul. de Thér.*, 1854), and Graves one in a male of seventy years.] The probable influence of the rheumatic constitutional state, or of some other unknown constitutional diathetic condition, has been already noticed.

[The occurrence of chorea in pregnancy—*chorea gravidarum*—should not be overlooked, and it in nowise differs from infantile chorea, either in the convulsive movements, or variable degree of intensity. An attack of chorea may happen for the first time during gestation, or a woman who has suffered when a child from an attack, and been cured, may have a re-seizure on becoming pregnant. Hysterical or epileptiform fits may complicate chorea gravidarum (ROMBERG, MOSLER). Hecker, of Munich, relates an instance of a woman of thirty-two years of age, who, towards the end of her fourth pregnancy, was attacked with a choreic rotary motion of the head, which lasted for thirteen days; for one year and a half



she was free from any nervous trouble, when becoming again pregnant, during the latter half, she was attacked with chorea limited to the upper part of her body, her head going from side to side like a pendulum. These symptoms lasted for nine days, and disappeared, to reappear at delivery, and again cease when the uterine contractions became strong.

The chorea of pregnancy is nearly always bilateral; it generally happens between 17 and 24 years of age, in about two-thirds of the collected cases in primipara, and usually during the first four months. It predisposes to miscarriage, or to premature delivery.]

**Prognosis.**—The recovery of the patient, with very few exceptions, may be always prognosticated. The disease will in general gradually decline, with complete removal of the spasms. The mean residence in hospital for cases of chorea has been found to be thirty-one days (RUFZ). Those cases only are apt to terminate fatally which occur during an attack of rheumatism or pericarditis, or when the disease assumes an intensely acute form, the patient losing rest at night and becoming exhausted; then emaciation progresses rapidly, and death occurs in from nine to twenty days. (See account of such a case by Dr. Wm. Weir, in *Glasgow Medical Journal*, No. I, 1853.) Dr. Walshe's experience leads him to believe that when the disease is slowly ushered in, it is more obstinate and enduring than when it is suddenly developed. An irregularly remittent course he regards as an inherent quality of the disease (*Lancet*, l. c.).

**Treatment.**—The indications of cure are, (1.) To remove, if possible, all morbid states of the body which may tend to aggravate the disease, such as *constipation*, *anæmia*, *amenorrhæa*, *worms*; (2.) By well-regulated purgative medicines, to subdue any cerebral congestion; (3.) To sustain the strength and improve the vigor of the nervous system by tonic and stimulant medicines, by food, and by the cold bath.

The particular tonic is not of much moment. Dr. Wood recommends the powder of the black snake-root (*cimicifuga*), in doses of from half a drachm to a drachm, or from one to two fluid ounces of a decoction; or from one to two drachms of a saturated tincture should be given three or four times a day, and continued for several weeks, the dose being gradually increased till it produces headache, vertigo, or disordered vision. The sulphate of zinc has also had the credit ascribed to it of curing a large number of cases, beginning with a grain in the form of a pill, three times a day, and increasing the dose till it reaches seven or eight grains daily. [The *oxide of zinc*, *ammoniated copper*, or the two combined, are favorite remedies; and the sulphate of manganese is favorably spoken of.] The preparations of iron are also frequently resorted to with benefit.

Dr. Walshe did not find any of these remedies at all useful in the acute case he has described. Of all the remedies he tried, the "extract of *cannabis Indica* was followed by the most satisfactory results. It exercised a sedative influence on the muscular action in a marked degree, and that immediately. *Nitrate of silver* he also found to have no mean influence in aiding the cure. The *Indian hemp* was given in doses of one-fourth of a grain of the extract thrice daily. The dose was subsequently increased to half a grain, and at

the same time one-grain doses of *nitrate of silver* were administered, and a draught containing eight drops of *dilute nitric acid* (*Lancet*, *l. c.*). Dr. Corrigan had also previously used *Indian hemp* with much success in chorea (*Med. Times and Gazette*, 1845, p. 29; also *Dublin Hosp. Gazette*). [It has also been successfully used in this disorder by Dr. Storer, of Boston.] The student is recommended to consult a valuable paper "On the Uses of Indian Hemp in Nervous Diseases," by Professor Russell Reynolds, in Beale's *Archives*.

Of other classes of stimuli, *camphor* in five-grain doses has acquired much reputation, especially after the discharges have become healthy by the action of purgatives. Many young women, also, who attribute the attack to fright, frequently get well from the simple administration of the *spirit of nitrous ether* in one fluid drachm doses three times a day, combined with the officinal *camphor mixture*. The catalogue of remedies proposed is endless. In many instances, however, the above medicines are continued for weeks, without any manifest improvement. In such cases the cold bath, or the cold shower bath, is an excellent adjuvant; and, unless the patient is suffering from some structural disease, the case uniformly yields to this conjoined treatment; great care and attention being bestowed on regulation of the diet, which should be light, nutritious, and easily digested.

[Dr. J. W. Ogle, has reported several cases of chorea successfully treated by the tincture of calabar bean (3j to f3j of rectified spirits of wine), beginning with 20 minims three times a day, and increasing by 10 minims a dose, to f3j. Sulphate of aniline, one grain to one grain and a half three times a day, has been used with alleged success by Dr. James Turnbull, of Liverpool, and others. Trousseau relies mainly upon the sulphate of strychnia, and Romberg, Avery, Dr. T. K. Chambers, and C. B. Radcliffe, strongly recommend arsenic. The latter gives it hypodermically. Bromide of potassium has been successful in the chorea of pregnancy (GUBLER, DUMONT, JACCOUD). Chloroform has been used to control the convulsive movements when violent and incessant so as to prevent sleep; and Dr. A. Clark, of New York, in a case of aggravated chorea, gave whiskey in large doses with good effect (*Med. Times*, 1862). The spasms have been quieted by spraying the upper part of the spine with ether, from Richardson's apparatus (LUBELSKI). Chapman's ice-bag has been applied with good result.

A chief difficulty in the treatment of chorea, as well as other nervous disorders, has been too great and exclusive reliance upon specifics. Hence the fifty remedies we have for the disease, all backed by high authorities; and the number is continually being added to. The rational treatment of the affection has been generally ignored. There is no doubt that a certain number of choreic cases get better under simply hygienic treatment,—better food, moral discipline, and a change of surroundings. In chorea there are depressed nervous energy and lessened systemic vitality. Anæmia is constantly present. A general tonic and restorative treatment is required, and under it the patient rapidly mends, and soon gets well. Nourishing food, regular exercise in the open air, warm clothing, occasional alkaline, or sulphur, baths, with cod-liver oil, iron, the hypophosphites, and arsenic, will generally succeed in effecting a permanent cure. The tonic effect of general electrization may be tried, and is often fol-

lowed by good results. Appropriate gymnastic exercises, first proposed by Récamier, and for some years in constant use at the Hôpital des Enfants Malades, Paris,\* act, not only by invigorating the system, but by special influence upon the affected muscles, and enable the patient to regain his lost control over them. Carrying articles which require careful handling, fix the attention, and call up the will, and slow walking in measured or cadenced step, and skipping the rope, should also be practised.]

## HYSTERIA.

**LATIN**, *Hysteria*; **FRENCH**, *Hystérie*; **GERMAN**, *Hysterie*; **ITALIAN**, *Isterismo*.

**Definition.**—*A complex morbid condition of all the cerebral functions, of a chronic kind, probably associated with some morbid state of the emotional or sensori-motor centres, and presenting every variety of alteration, so that the phenomena of hysteria simulate or mimic the phenomena of almost every other disease, while the most common and characteristic features of the affection are certain motorial changes of a convulsive nature, and usually of paroxysmal occurrence.*

**Pathology.**—Three theories have been entertained relative to the nature of this disease, and to the primary seat of the affection: (1.) Some, with the ancients, refer it to a morbid condition of the uterus. (2.) Others consider it exclusively due to a morbid state of the cerebral structures. (3.) A third class refer the phenomena to a morbid excitability of the whole nervous system, which renders it liable to be thrown into disorder by causes insufficient materially to disturb its action in health,—thereby implying paralysis of some nervous centres. Post-mortem examinations of the bodies of those who have died from other diseases while suffering from hysteria have yielded negative results.

**Symptoms.**—The forms and degrees of hysteria are so numerous that the difficulty of describing this disorder is very great. The modifications of age, temperament, states of nervous sensibility, physical and moral education, and grades of society, so influence its aspect that it is only possible to give a mere general outline. It is usually divided into three forms: *first*, that which is characterized by what is termed the "*globus hystericus*," in which the sensation of a ball rising in the throat, or a feeling of suffocation, is experienced by the patient, but without convulsions; *second*, its paroxysmal form, or that in which the *globus hystericus* occurs with convulsions; and *third*, those irregular and anomalous phenomena which often manifest themselves during the intervals of severe attacks.

The milder forms are those which terminate without the formation of the paroxysm. They commonly begin with pains in the epigastrium; in the left side, or in some other part of the abdomen; or the patient is unusually nervous, her feelings excited or depressed. These symptoms having existed for a longer or shorter period, the patient experiences the sensation of a ball, the "*globus*

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\* [*Du traitement de la Chorée par le gymnastique*, par le Dr. Blache, Mém. de l'Acad. de Médecine, t. xix, Paris, 1855. *Rapport de M. Bouvier sur ce travail*, Bul. de l'Acad. de Méd., t. xx, Paris, 1854-55.]

*hystericus*," rising apparently from the lower portion of the abdomen, and proceeding upwards with various convolutions to the stomach, thence to the throat, and causing sometimes an intense sense of suffocation. At this point the slighter forms frequently cease, but are followed by headache, stiffness of the neck, general weariness, a profuse discharge of a light-colored limpid urine, and by great flatulence, the abdomen becoming almost instantaneously distended.

When hysteria assumes a paroxysmal form or "fit," it may be preceded by the pains and mental feelings which have been described; but not unfrequently the attack is sudden, and is often caused by some transitory occurrence. In such a case the patient bursts out into a fit of immoderate laughter or crying, the *globus hystericus* begins to form and to rise, and no sooner reaches the throat than she falls to the ground, apparently unconscious and violently convulsed. The fit is now said to be formed, and while in general the convulsions are easily controlled, yet,—not only in the strong and plethoric, but sometimes also in delicate-looking, slight-made girls,—many persons are sometimes necessary to restrain the patient, who writhes her body to and fro, agitates her limbs in various directions, and beats her breast repeatedly with her arm and hand. During the fit the patient also often knocks her head against the bed or floor, tears her hair, screams, shrieks, laughs, cries, or sobs alternately. The respiration is slow, and is rendered still more laborious by spasms about the pharynx and glottis, so that the patient often grasps her neck and throat, or rubs or strikes the epigastrium and side with her hand. During this struggle she may bite her own arms or those of the bystanders, and will sometimes move round the room while lying on her back, by means of the muscles of the dorsal region. The abdomen is often singularly distended with flatus; but in other cases the muscles of that region are tense and irregularly contracted. The pulse is in some cases increased by the violence of the exertion, but in others its beat is natural. The veins of the neck are distended, the carotids beating with more than usual violence. The face is flushed, and "the head is generally thrown back, so that the throat projects; the eyelids are closed, but tremulous; the nostrils distended; the jaws often firmly clenched; there is no *distortion* of the countenance, and the cheeks are at rest," unless when giving expression to some of the above-mentioned phenomena. The temperature of the extremities is often lower at the commencement than natural, so as to cause a momentary shivering; but, as the paroxysm forms, the heat is usually restored, and sometimes increased. The phenomena attending the subsidence of the paroxysm are very various; sometimes a flood of tears, a fit of laughter, or an exclamation, is followed by a great flow of limpid urine, after which the recovery is generally rapid and complete. In other cases the action of the stomach becomes inverted, and the sympathizing attendant, perhaps watching the patient with the tenderest care, receives its whole contents over her person, after which the patient may lapse, most unconcernedly, into a profound sleep. In others, again, the fit only partially passes

off, and the patient lies, to a certain extent, sensible of what is passing about her, perhaps jaw-locked, the secretion of urine suspended, unable to talk, and often obliged to be fed. The fit having entirely subsided, the patient lies exhausted and unwilling to be disturbed, and although more or less conscious of what has passed, she wishes to be thought ignorant of all that has taken place. A want of consciousness may exist when the fit assumes a severe or epileptic form, but this is not a common symptom of the purely hysterical convulsion. In some few cases the patient appears to be delirious, and makes the most extraordinary noises, such as barking or howling like a dog. The duration of the fit is very various, from a few minutes to two, three, or more hours. These fits readily recur, and no sooner is one fit ended than the patient suffers from another; and in this manner the whole attack may last twelve, twenty-four, or even forty-eight hours. In general the intervals are much longer, and not subject to any general law of recurrence, except that they are more common about the period of menstruation.

In the interval between fits, the symptoms are extremely anomalous and irregular, and more strange and difficult to describe than even those of the paroxysm. Some have their senses so acutely alive that although the window and bed-curtains may be drawn, still they are pained with light, and the slightest noise distresses them. In some again, the sense of touch is so exquisite that they can scarcely bear the weight of the bedclothes; and to others odors are similarly intolerable. Besides this extreme acuteness of the senses, others suffer pains under or in the *mammæ*, known to surgeons as the "hysterical breast," lumbar pains, pains in the hip-joint, headache fixed to one spot, *clavus hystericus*, and palpitation. Pain in the region of the spine is also frequent, and often so intense and so exquisitely increased by pressure that it has often been mistaken for ulceration of the intervertebral cartilages. The late Sir B. Brodie recorded that he had seen numerous instances of young ladies condemned to the horizontal posture, and to the torture of issues and setons for successive years, whom air, exercise, and cheerful occupation would have cured in a few weeks. As to painful affections of the joints, it has been stated by the same high authority, that at least four-fifths of the females among the higher classes who are supposed to labor under diseases of the joints are suffering from hysteria, and from nothing else. The morbid sensibility is chiefly in the integuments, as in the case of the hip-joint, and if they are slightly pinched or drawn from the subjacent parts, the patient complains more than when the head of the femur is pressed against the acetabulum. There is likewise no wasting of the *glutei* muscles, nor flattening of the *nates*, nor painful starting of the limbs. In some instances the patient becomes paraplegic, and is unable to walk, while others suffer temporarily from *hemiplegia*.

It is the extreme acuteness and exquisite sensibility of the senses in hysteria which has led those less skilled in female arts to believe in the instances of animal magnetism and mesmerism they



seemed to exhibit, which formerly attracted so much public attention. A most interesting account of the vagaries of hysteria may be read in Dr. Watson's Thirty-eighth Lecture, *On the Practice of Physic*.

**Diagnosis.**—The best diagnostic guide may be obtained by classifying the symptoms according to Dr. Reynold's plan, as followed in the description of the previous diseases. There is to be noticed—

1. *The Mental State.*—Volition is deficient and misdirected. The Emotions and Ideas exhibit excessive activity; and to the combination of these two conditions is to be attributed many of the peculiar and characteristic features of the disease. *Assertions* by the patient are being constantly made—such as that she cannot control her Thoughts, Emotions, Expressions, or general voluntary movements; or that she cannot move this or that limb, that she cannot open her eyes, that she cannot stand or walk; and if she makes the attempt under such impressions, she certainly fails; and she may simulate the real inability so completely, and so well, that it seems almost incredible that nothing but defective Will is the real source of the failure. If, however, some strong Motive, Emotion, or Sensation, come into operation, she may for a moment forget her condition, clap the hitherto moveless hands together, open the closed eyelids, and, with the rapidity and energy of robust health, run across a room or up a staircase with her *quasi*-palsied limbs. It does not appear that with all this there is any intention on the part of the patient to deceive any one, more than herself. It is truly a morbid mental condition on her part, and she doubtless believes in the real nature of her symptoms. Often, also, a species of delirium prevails, in which nonsensical sentences are pronounced in an excited manner. Uncontrolled sobbing, sighing, and laughing, are alternately produced, or accompany each other. Somnambulism, ecstasy, or hysteric coma (which is rare), may prevail. Often the expression of the face is insane. A listless, abstracted, vacant look pervades the countenance, as if the individual cared nothing for the things of this world. Combined with this condition there is restlessness and impatience of temper, with monosyllabic talking.

2. *Sensorial.*—The pain of hysteria, which may be anywhere, but most commonly in the head and mammary region, is always described as “intense,” “horrible,” or “agonizing,” and is increased when the attention is directed towards it, but lessens when the attention is withdrawn. The patient generally shrieks when the skin is touched.

3. *Motorial* phenomena, when voluntary, are performed sluggishly and imperfectly. The other phenomena in connection with the motor power are exhibited in convulsions or paralysis. It is the convulsive paroxysms of hysteria which may be mistaken for epilepsy. When they occur, it is almost always at the period of puberty in young females. They frequently attend the menstrual period, and are preceded by the premonitory phenomena of hysteria already described, and which reappear towards the close of the convulsions. There is probably never complete loss of Sensibility

and Perception. The spasmodic movements are general. The face undergoes little alteration. There is commonly a contractile movement of the eyelids. The patient appears to see, and there is no marked change of the pupil. Foaming at the mouth or a bitten tongue is rare. The attacks are sometimes of considerable duration, and the respiratory movements become very disorderly. After the paroxysm has passed, there is no marked stupor, but merely general exhaustion; and loss of Consciousness appears to be very seldom complete, and never occurs at the outset of the attack. "The patient," writes Dr. Watson, "is often able to repeat (although she may not always choose to confess it) what has been said by the bystanders during the period when she seems insensible. This is a point of distinction well worth remembering, for more reasons than one. It not only helps the diagnosis, when the fact comes out, but it suggests certain cautions to ourselves. We must take care not to say anything by the bedside of an hysterical patient which we do not wish her to hear; and we may take advantage of her apparent unconsciousness, and pretend to believe in it, and speak of certain modes of treatment which she will not much approve of, but the very mention of which may serve to bring her out of the fit."

The less-expressed forms of hysteria cannot be confounded with the less-expressed forms of epilepsy. The non-convulsive form of epilepsy is exclusively expressed through disordered sensorial states, such as by vertigo and a suspension (however brief and transitory) of the mental powers. The non-convulsive forms of hysteria, on the other hand, are chiefly expressed in derangement of the organic functions of the thorax and abdomen (FOVILLE, WATSON).

It is often difficult to distinguish between the many painful affections of the joints which arise from hysteria and the formidable diseases of these parts which they simulate, mimic, or copy; and many mistakes have been made fatal to health and even to life. The character, however, of the patient, her time of life, her general good health, the intermitting nature of the pain and its following the course of the nerve, enable us generally to determine with much accuracy between these different classes of disease. The most common mistake, however, is that of considering the pains under the mammæ as pleurisy or disease of the liver, or the abdominal pain as peritonitis or enteritis, thereby leading to an abuse of bleeding, blistering, and the administration of mercury. The state of the pulse, however, the general good health of the patient ("for," writes Dr. Wood, "one of the most striking circumstances connected with the disease is the general integrity of the nutritive process—the patient continues plump and rosy"), and most commonly the existence of some uterine irritation, furnish sufficient grounds for diagnosis between these different diseases.

Nausea, eructations, borborygmi or tympanitis, palpitation of the heart with syncopal feelings, frequent micturition of clear pale urine, are characteristics of the hysterical state. A certain constitution is also characteristic of the sufferer from hysteria. The external conformation of the features of the face is often of itself sufficient to indicate the existing tendency. The "*facies hysterica*"

may be recognized by the remarkable depth and prominent fulness of the upper lip, which is more or less thick. There is also a fulness of the eye, with a tendency to drooping of the upper eyelids.

**Causes.**—The remote causes of this affection are rather moral than physical; and in a young person predisposed to the disease almost any mental emotion will excite it, as anger, disappointment, jealousy, protracted expectation, the loss of a husband, a friend, or a child; indeed, all that brings the Passions, Emotions, or Affections of the mind into play is a cause of this disease; and many women cannot go to church, or witness a tragic representation, without suffering from hysterical paroxysms.

This disease almost exclusively attacks females between the ages of fifteen and thirty, or during that period of a woman's life when the generative functions are fully developed and in their greatest vigor. Those most liable are the unmarried or continent, and those that labor under amenorrhœa or menorrhagia. The married woman often suffers just after conception, or before parturition, or subsequently as a consequence of protracted suckling. The barren woman, however, is more liable, and probably from her mind being acted upon by a greater number of exciting causes, such as disappointment in the prospect of being a mother. Taking classes of women, the higher classes, from their artificial modes of life, are greater sufferers than the lower orders. But although this is a disease almost peculiar to woman, it is not entirely so, but occasionally affects the male sex under conditions of mingled debility and excitement. Shakspeare makes Lear exclaim, when Gloster relates the cause of his being put in the stocks—

“ Oh, how this mother swells up toward my heart!  
*Hysterica passio!*—down, thou climbing sorrow,  
 Thy element 's below !”

The predisposition to the disease, however, is most manifest in that peculiar condition of the nervous system for which we have no more precise or definite expression than *nervous irritability* or *mobility*—a condition which is more common in women and children than in men, and more common in all persons when in a state of weakness than when in the full enjoyment of muscular strength. In women the affection is more common about the menstrual periods, and immediately after parturition, than at other times; more common likewise among those in whom the monthly discharge is habitually *excessive* or *altered* (as in *leucorrhœa*), or suddenly *suppressed*, or more gradually obstructed (as in the different forms of *amenorrhœa*). In this condition of mobility both Sensations and Emotions are intensely felt, and their agency on the body is stronger and more lasting than usual; continued voluntary efforts of Mind, and steady or sustained exertions of the voluntary muscles, are difficult or impossible; the muscular motions are usually also rapid and irregular.

**Prognosis.**—The ultimate result of these cases, though often long and tedious, is always favorable. “In nine hundred and ninety-

nine cases out of a thousand, hysteria is attended with no ultimate peril either to mind or body" (WATSON).

**Treatment.**—The treatment may be divided into what should be done during the fit, and into what should be done afterwards.

When the patient falls in a fit of hysteria, the first thing to be done is to loosen everything tight about her person. The window should be opened, and the cold air allowed to blow over her. She should then be laid in the horizontal posture on a bed, or on the floor, as a means of rendering the circulation through the brain more equal, and to enable us the more readily to control the convulsive movements of her body. This being done, many modes of further proceeding may be followed. Some recommend, in plethoric cases, that the patient should be bled—a remedy certainly in many instances manifestly improper, and in all of doubtful efficacy. When the jaw is locked, Dr. Wood recommends that an enema, consisting of the yolk of an egg beat up with two drachms of *asa-fetida*, with half a pint of water added, may be administered; or, still better, an enema of turpentine, in which half an ounce of turpentine is similarly mixed with the yolk of an egg, and half a pint of water added. These remedies, in some instances, he adds, instantly remove the affection, but in other cases not for some hours. Another remedy is to fill the mouth with salt: "You generally see them come round if you fill the mouth with salt." The remedy, however, which supersedes all others, and is unquestionably the best, is a good drenching with cold water. "I believe there is more virtue in cold water than in any other single remedy" (WATSON). If the patient lie on the bed, the head should be drawn over its side, and a large quantity of water poured on it from a considerable height out of a pail, jug, or other large vessel, and directly over the mouth and nose of the patient, so as to stop her breathing and compel her to open her mouth. This practice is generally introduced into hospitals, and until it was adopted it was not unusual to see three or four patients in hysteria in the same ward, and at the same time. Under this practice, however, an hysterical case is rare, and the fit seldom occurs twice in the same person, and never becomes epidemic.

[Large doses of morphia and sulphuric ether, or chloroform, or chloric ether, or of some preparation of valerian, or of bromide of potassium, are among the best means of controlling a seizure.]

After the paroxysm is over, if the patient complains of continued headache, a few leeches to the temples may be necessary, especially if the urine be small in quantity and high-colored; but in all other cases leeches, blistering, or cupping, should be avoided, as tending rather to aggravate than to control the disease. The next object is to regulate the bowels by such remedies as may be necessary, and at the same time to support and tranquillize the patient by stimulants, such as *ether* or *asa-fetida*, combined with *hyoscyamus* in the form of tincture, the *syrup of poppies*, or small doses of *morphia*. The state of the uterine functions is next to be inquired into. If leucorrhœa

be present, or the menstruation be profuse, the *mineral acids*, or the *bitartrate of potass*, will be found most efficacious, by restoring a more healthy state of the deranged organs.

The urine is often suppressed for a time after an attack of hysteria; but unless the bladder be sensibly, and perhaps painfully distended, no attempt should be made to draw the urine off. Something more should be hazarded to avoid this necessity, for the catheter once passed, that operation will require to be performed morning and night, perhaps for several months.

### CATALEPSY.

LATIN, *Catalepsis*; FRENCH, *Catalepsie*; GERMAN, *Catalepsie*; ITALIAN, *Catalepsia*.

**Definition.**—*A sudden suppression of Consciousness; but instead of falling down convulsed, as in hysteria, the patients maintain the position in which they were when the attack commenced. The limbs and trunk persist in a state of balanced muscular contractions; the same expression of countenance which may chance to be at the moment of seizure is preserved. If sitting, the patient continues to sit; if standing, he continues to stand; and if occupied in any mechanical employment, he continues fixed in one attitude; and if he is under the influence of any Passion, the countenance retains its expression.*

**Pathology.**—This is an extremely rare form of nervous disease, apparently intermediate between hysteria and epilepsy; but probably more allied to hysteria. It affects the two sexes nearly with equal frequency. Dr. Reynolds has observed the cataleptic state ensue in cases of chronic ramollissement of the brain, and in tubercular meningitis; and Dr. Laycock compares the condition to the state presented by the so-called "*brown study*." The combination of fixed attitude and of unvarying expression gives to the patient the air of a statue rather than that of a living being, and he appears as if suddenly changed to stone. The most remarkable circumstance, however, in this disease is, that the attitude of the body and position of the limbs admit of being changed almost into as many new forms as a painter's lay figure, and the new position, however inconvenient, and almost volitionally impossible, is preserved till again changed, or until the paroxysm has subsided.

Besides this singular state, Consciousness is suspended, and the patient neither receives any impression from external objects, nor retains any recollection of what has happened during the fit.

In this respect the disease approaches in character to epilepsy. The organic functions of life, however, continue to be performed, though feebly. The pulse and respiration are regular, only the former is smaller and the latter less frequent than in health. The color of the countenance is either pale or undergoes no change. The fit may last a few minutes or a few hours, and is said to have continued in some cases three or four days. The patient at length awakes as from sleep, and generally with a deep sigh, when all the functions of the body are suddenly restored. The attack is



generally sudden, and without any previous symptoms, but it is sometimes preceded by headache, stiffness of the neck, or some obvious torpor of the mind or body. The return of the paroxysm is very uncertain, but the disease seldom subsides with the first attack. The following case, given by Dr. Gooch, will best exemplify this affection:

A lady who labored habitually under melancholy, a few days after parturition was seized with catalepsy, and presented the following appearances: She was lying in bed motionless, and apparently senseless. It was thought the pupils of her eyes were dilated, and some apprehensions were entertained of effusion on the brain; but on examining them closely it was found they readily contracted when the light fell upon them. Her eyes were open, but there was no rising of the chest, no movement of the nostril, no appearance of respiration. The only signs of life were warmth and a pulse which was 120, and weak. Her fæces and urine had been voided in bed. In attempting to rouse her from this senseless state the trunk of the body was lifted up and placed so far back as to form an obtuse angle with the lower extremities, and in this posture, with nothing to support her, she continued sitting for many minutes. One arm was now raised, and then the other, and in the posture they were placed they remained. It was a curious sight to see her sitting up staring lifelessly, her arms outstretched, yet without any visible signs of animation. She was very thin and pallid, and looked like a corpse that had been propped up and stiffened in that attitude. She was now taken out of bed and placed upright, and attempts were made to rouse her by calling loudly in her ears, but in vain; she stood up, indeed, but as inanimate as a statue. The slightest push put her off her balance, and she made no exertion to regain it, and would have fallen had she not been caught. She went into this state three times; the first lasted fourteen hours, the second twelve hours, and the third nine hours, with waking intervals of three days after the first fit, and of one day after the second; after this time the disease assumed the ordinary form of *melancholia*. It might be supposed that symptoms such as these were feigned; but there are cases beyond suspicion of this kind; and another very interesting case is related by Dr. George Buchanan in *The Glasgow Medical Journal* for July, 1857. It is an instance of this singular affection occurring in the male sex.

[Catalepsy is not easily feigned, but it would seem that a person may voluntarily throw himself into it. An instance is given by St. Augustine of a presbyter named Restitutis who, when he pleased, could lie like a corpse, and beyond the influence of sensation, by having a noise made like some one crying. He was insensible to pinching and pricking, and fire caused no pain, except from the after-sore. The body was motionless, without any appearance of respiration, as a corpse. He said that he could hear people's voices, if they spoke distinctly, as if they were a long way off.

In feigned catalepsy the peculiar wax-like yielding resistance of a cataleptic muscle is wanting, and shows the imposture. Dr. Marx having noticed that really cataleptic limbs slowly yield to the force of gravity and fall by their own weight, attached a heavy body to the extended hand of

a malingerer; it was borne up without moving, and the fraud subsequently acknowledged.]

**Prognosis.**—The affection is generally innocent; but as it is apt to be associated with cerebral disease, which may end in cerebritis, apoplexy, or insanity, and also with serious organic disease of the viscera (REYNOLDS, WOOD), it behooves the physician to be guarded in predicting results, especially in our ignorance of the nature of this disease. [It is more apt to be the precursor of epilepsy, and is sometimes followed by melancholia. In both the natural and in the induced disorder somnambulism is frequently a complication.]

**[Treatment.]**—No constant line of treatment can be stated. The individual case must be judged of upon its own merits, and prescribed for according to the principles which have guided the dictates of treatment in the allied nervous affections.

[Of the treatment of catalepsy, Dr. T. K. Chambers remarks: "I would strongly urge upon all who have charge of these and similar semi-mental, semi-corporeal manifestations, to try to acquire (surely it is to be acquired by trying) the habit of command. Let them exercise it in the direction of supplying the deficient will, not of paralyzing it; of demesmerizing instead of mesmerizing their patients, and it is astonishing how much pharmacopœial medication will be saved to both parties" (Reynolds's *System of Medicine*, vol. ii).]

## EPILEPSY.

**LATIN**, *Epilepsia*; **FRENCH**, *Epilepsie*; **GERMAN**, *Epilepsie*—Syn., *Fallende Krankheit*, [*Fallsucht*]; **ITALIAN**, *Epilessia*.

**Definition.**—A complex nervous state, in which, as a rule, a sudden and complete loss of consciousness prevails, associated with convulsions, as if tonic at first, but which subsequently become clonic, and ultimately impede the respiratory process. The attack, lasting from two to twenty minutes, is followed by some exhaustion and sleep. The expression of the epileptic state varies from the most severe paroxysm to simple vertigo, a momentary suspension of consciousness, a fixity of gaze, a totter of step, and a confusion which appears and disappears almost instantaneously, and which only the patient can recognize.

**Pathology.**—This disease has been known from the earliest antiquity, and is remarkable as being that malady which, even beyond insanity, was made the foundation of the doctrine of possession by evil spirits, alike in the Jewish, Grecian, and Roman philosophy. The interest and importance which attaches to this disease cannot be better expressed than has been done by Dr. Watson. He writes that it "is scarcely less terrible to witness, when it occurs in its severer forms, than tetanus or hydrophobia; but it is not attended with the same urgent and immediate peril to life. Yet it is, upon the whole, productive of even more distress and misery, and is liable to terminate in even worse than death;—a disease not painful probably in itself; seldom immediately fatal; often recovered from altogether; yet apt in many cases to end in fatuity or insanity; and

carrying perpetual anxiety and dismay into those families which it has once visited" (Watson, Lect. xxxv). Again, fully impressed with the responsibility entailed on the physician in the diagnosis between hysteria and epilepsy, and the necessity of its being certain and accurate in either case, he says, "It is a dreadful announcement to have to make to a father or a mother that their child is epileptic" (Lect. xxxviii).

[The proportion of true epilepsy to other diseases of the nervous system is about 7 per cent. (REYNOLDS.)]

It has been affirmed, that in fifteen out of twenty cases, in which the brains of epileptic patients have been examined, the structure of that organ has been in every respect healthy. Even when the patient has died during the paroxysm, the brain has in many instances been found congested only. Epilepsy has been, therefore, regarded as a functional disease, the particular seat of lesion not being determined. But although epilepsy may exist without any obvious disease of the brain or of its membranes, it must be admitted that the brain and its membranes are occasionally found in every state of disease to which those parts are liable. Thus, the membranes may be inflamed, thickened, or ossified, and with every variety of exudation; or the substance of the brain may be indurated or softened—the seat of abscess, of cancer, of tubercle, or of other structural disease. Any such structural disease is then considered to give rise to the epileptiform attack. Dr. Sieveking, whose researches into the nature of this disease have been most prolonged and laborious, once showed me an interesting old German work, by T. and C. Wenzel, in which the epileptic state was shown to have been invariably found associated with a morbid state of the *pituitary* body in the *sella turcica*—a spot of the encephalic region very rarely examined in post-mortem examinations. The tendency of modern pathology seems to be to connect the epileptic seizure with a variety of pathologico-anatomical lesions of a variable and inconstant kind, such as have been mentioned. There are also cases referred to causes of an eccentric nature, such as to uterine or ovarian disease, which are thus said to act upon the brain through the medium of the nervous system, in some way as yet unknown. Dr. Todd developed a theory of the disease, suggested by the occasional occurrence of epilepsy with renal affections. He held that the peculiar features of an epileptic seizure are due to the gradual accumulation of a morbid material in the blood, until it reaches such an amount as to operate upon the brain, as it were, in an explosive manner. In other words, the influence of this morbid matter, when in sufficient quantity, excites a highly polarized state of the brain, or of certain parts of it, and these discharge their nervous power upon certain other parts of the cerebro-spinal centre, in such a way as to give rise to the phenomena of the fit. This theory assumes that the essential derangement of health in epilepsy consists in the generation of a morbid matter which infects the blood, and it supposes that this morbid matter has a special affinity for the brain, or

for certain parts of it, just as *strychnine* exercises a special affinity for the spinal cord. According to this theory the disease ought to have found a nosological place amongst the *constitutional diseases*. But, to give a definite character to such a humoral theory, it were necessary to discover some morbid matter in the blood in every case of epilepsy.

“This desideratum has, as yet, been only partially obtained. The clue to a discovery of this kind was first given by the observations of Prevost and Dumas upon the effect of excision of the kidneys. These observers found that the removal of the kidneys always led to an accumulation of a considerable quantity of urea in the blood, followed by convulsions and coma—an epileptic state. After this, clinical observations by practical physicians showed that disease of the kidney was apt to be followed by attacks of convulsions and coma, when the excretion of urine fell in quantity to a very low amount; and it was found that, in such cases, a considerable quantity of urea was present in the blood. A connection was clearly thus established between the presence of urea in the blood, defective renal action, and the epileptic condition; but whether the active poison is urea cannot yet be decided. Frerichs, indeed, has lately affirmed that it is carbonate of ammonia, a product of the decomposition of urea. But even this is still *sub judice*. All that we really know is, that in certain states of diseased kidney, when the excretion falls below a certain point, urea will accumulate in the blood, and epileptic seizures will ensue; and, should the patient die, we find no brain-lesion to explain the phenomena; but we find unequivocal evidence of diseased kidney” (“Clinical Lectures,” by Dr. R. B. Todd, *Med. Times and Gazette*, Aug. 5, 1854).

In the present state of our knowledge, therefore, and knowing that there are a great many cases in which the epileptic phenomena have recurred during a long period, and in which post-mortem examination reveals no lesion with which symptoms can be connected, it is then better to consider epilepsy as an intrinsic disease of the brain, because the most constant and marked groups of phenomena are referable to the functions of the central parts; while the loss of Consciousness, associated with excessive mobility, leads one to regard those parts of the brain in the immediate vicinity of the *sella turcica* and basilar portion of the occipital region—for example, the *central ganglia* or *medulla oblongata*—as parts where, in future, morbid anatomy may yet discover a lesion. On the other hand, epilepsy in very many cases must be regarded as “a disease of the whole man, and not of any one organ or system of organs alone;” and if lesions of a more or less constant kind be ever found in the brain, they would merely stand in the same relation to epilepsy that morbid states of the kidney may be regarded in relation to the whole phenomena of Bright’s disease, regarded as a *constitutional disease*. Epilepsy, therefore, might with fair reason be regarded in this respect as a *constitutional disease*, with intervals of apparent freedom, and with times at which the disease culminates in the characteristic paroxysm; and in order fully to appreciate the nature of epilepsy, as Dr. Sieveking justly remarks, a careful study of the general condition of each patient is necessary, and especially

of the phenomena which may show themselves in the free intervals. Attention must not be limited to the paroxysm alone.

[In the greater number of, if not in all, cases of true idiopathic epilepsy, no coarse or appreciable material lesion can be found. The functional disturbances which have their expression in the type-symptoms of the disorder follow, no doubt, certain molecular tissue-changes, which, however, elude all our present means of investigation. Wholeness of function is kept up by interstitial movements of repair and waste, and loss of balance between these brings on functional trouble. Now the nutrition changes in epilepsy may be connected with a general cachectic condition, like gout, scrofula, &c., and have their remote origin in the blood; but when once set up, they fasten on the medulla oblongata and upper segment of the spinal cord, establishing an excessive and perverted readiness of action. The first step of disordered movement in this disease would seem to be the bringing on of arterial-spasm, and the consequent production of cerebro-spinal anæmia. The excessive paleness of the face, loss of consciousness, contraction of the muscles of the chest-walls and larynx, &c., at the beginning of the fit, all point to this condition. Many of the subsequent symptoms are the result of the state of the blood, which becomes venous, from the arrest of breathing hindering the proper changes in it, and causes the phenomena of asphyxia.

It is very probable that the sympathetic system is deeply, if not primarily, implicated, but there is as yet no substantive proof of this hypothesis.]

**Symptoms.**—Epilepsy may be grave or slight. The attack often occurs without any previous warning; so much so that Georget estimates that in 95 cases out of 100 there are no premonitory symptoms. These warnings are known by the term "*aura*." They comprise all the premonitory symptoms which may prognosticate the approach of a fit. Dr. Sieveking has noted such "warnings" in 48 out of 104 cases—a little more than 46 per cent. Many patients, however, on the approach of the fit, have vertigo or headache; some swelling of the veins, or throbbing of the arteries of the head; while others, again, have ocular spectra, or affections of the other senses. Dr. Gregory used to mention, in his lectures, the case of an officer whose paroxysm was always preceded by the spectre of a little old woman dressed in a blue cloak, who issued, as he imagined, from the corner of the room, and knocked him down with her stick. Dr. Fothergill attended a Quaker who always fancied he saw his garb covered with spangles before he fell into the fit. These ocular spectra are very numerous; but the most common are flashes of light, tadpoles, flies, colored areolæ around the flame of the candle, black dogs, and white horses. Others have hallucinations of hearing, as the ringing of bells, or the roaring of the sea; while others, again, are annoyed by the smell of disagreeable odors, or by the sensation of unpleasant tastes. When the sense of touch is the seat of the hallucination the term "*aura epileptica*" is used to express it. In these cases the patient has often the sensation of a fluid creeping from the fingers or toes upwards towards the trunk; others feel as though a spider or other insect were crawling over the skin. Dr. Elliotson speaks of a patient that had two *auræ*, each of which ran



along the dorsum of each foot, ascended up the front of the legs and thighs to the trunk, where they broke into five streams, all of which again met at the epigastrium, and, having reached this point, he fell into the fit. The late Dr. John Thomson, of Edinburgh, relates an instance of an epileptic “*aura*” commencing in an old cicatrix in the side; and Dr. Watson mentions the warning sensation as originating in the thumb of one of his patients, which presently became twisted inwards; and he could sometimes prevent the complete expression of the fit by tying his handkerchief tightly round the throat. Esquirol relates the case of a woman, in which the *prodrome* consisted in the patient turning round for a considerable time; and another of a man, who ran with all his might, till at length he fell down, overpowered by the disease.

Although these sensations may be subjective, and experienced only in the skin, and not following the course of any particular nerve, yet their subjective origin may “be due to some injury done to, or some morbid impression made upon, an *afferent* nerve,” as well as to some morbid condition of the brain itself.

Regarding the frequency of the occurrence of individual symptoms, Dr. Sieveking met with headache in 56 out of 104 cases, or in a ratio of 53.8 per cent.; and the pathological import of the symptom varies much according to the period at which it is met with. It may be connected with the fit etiologically; or it may be a consequence of the attack; or it may be a casual coincidence (*On Epilepsy and Epileptiform Seizures*, second edition, p. 54).

[According to Reynolds (*l. c.*, p. 264), the relative frequency of the different classes of premonitory symptoms are:

Mental and Emotional, . . . . .	11.1 per cent.
Sensational, . . . . .	19.8 “
Motorial, . . . . .	8.6 “
Vascular and Secretory, . . . . .	8.7 “ ]

In the *adult*, whether the warning symptoms be or be not present, the attack usually commences by the patient uttering a cry, losing on the instant all Consciousness, and falling down in convulsions, his mouth covered with foam.[\*] The convulsions vary from the most trifling and transitory convulsive movement to the most frightful, terrific, and long-continued struggles. In mild cases only one limb is convulsed; in others only the face, the lip, or the eye. Esquirol gives the case of a lady whose fits were so slight that although often seized on horseback, she never fell off. In a few seconds she recovered, and resumed the conversation by finishing the sentence she was expressing. In this case, however, the epileptic cry and the convulsed eye denoted the true nature of the attack. Another lady, advanced in life, suffered from fits so slight that she preserved her seat in the chair; and except for the occurrence of some slight convulsive motions about the mouth, followed by a

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\* [The immediate precursory symptom of an attack is a momentary corpse-like paleness of the face at the time of the fall, which is immediately followed by the flushed face (DELASIAUVE, TROUSSEAU, G. JOHNSON).]

short sleep, the attack would have passed unnoticed. Attacks so mild often occur many times in the day, last about five minutes, and appear for a time to leave no feeling of ill-health behind.

In severe forms of epilepsy the convulsions are more formidable; the hair stands on end, the forehead is wrinkled, and the brow is knit. If the eyelid be opened, the eye is seen to be injected, sometimes convulsively agitated, at other times in a state of strabismus, and sometimes fixed; more commonly the eyelid is quivering, and half-open, so as to show the white of the lower portion of the conjunctivæ. The face is red, or livid and swollen, the teeth generally clenched, and the lips covered with foam; sometimes, however, the mouth is open and the tongue thrust forward; and should the masseter muscles now act spasmodically, it may be bitten through, or otherwise much injured, and the foam consequently be mixed with blood. The force with which the jaw closes is so great that teeth have been known to be broken and the jaw luxated. The limbs, also, are violently convulsed, thrown about in every direction, and with such power that it often requires three or four persons to prevent the patient seriously hurting himself. In these convulsions, also, the hands are strongly clenched, and the body is often arched backwards (*opisthotonos*); when, on the muscles relaxing, the patient may fall to the ground with great force. While the limbs and trunk are thus powerfully agitated, the muscles of the chest are often spasmodically fixed, so as hardly to permit the acts of respiration.

The functions of organic life are also implicated in this scene of tumult. The pulse is generally frequent, sometimes hard and intermittent, and at other times scarcely perceptible, although the heart's beats are strong and tumultuous. The respiration is stertorous, the stomach and bowels troubled with horborygmi, the skin bathed in sweat, while the urine, semen, or fæces are occasionally emitted. Blood sometimes flows from the eyes, ears, or nose, frightfully expressive of the violence of the attack.

When the paroxysm has reached its crisis the muscles relax, the convulsions subside, the respiration becomes more free, the pulse more regular, and the countenance more natural; and at length the patient falls into a heavy sleep, from which he awakes sometimes in good health, but more often shaken, exhausted, and suffering from severe headache, which lasts some hours or even days. In neither case, however, has he the slightest consciousness or remembrance of what has passed. In other instances the termination of one paroxysm is but the beginning of another, and the succession is occasionally so continued that the attack, with short intermissions, may last twenty-four or forty-eight hours, or even longer.

When *children*, from teething or other causes, are seized with epilepsy, the attack is often preceded by a spasmodic affection of the larynx, causing the whooping or crowing sound so well known to every practitioner; but it may, and often does, take place without any warning. In the former case the child perhaps is in his best health, but on awakening is seized with the characteristic whoop, often accompanied by a spasmodic flexion of the thumb

against the palm; or the fingers are clenched, or the toes bent. These symptoms may recur a varied number of times, till at length, with or without this warning, the eye is seen staring, fixed, or convulsed; the face and extremities pale or livid; the hand clenched, the body rigid, and the head and trunk curved backwards. The fit is now formed; and if we examine the *fontanelle* we find it distended and pulsating. These symptoms generally last only a few minutes, when a strong expiration takes place; a fit of crying succeeds, and the child, much exhausted, recovers its Consciousness, and after a short interval generally falls asleep. These convulsions seldom occur during the early periods of lactation, nor until the commencement of the period of dentition, and rarely after three years of age.

The duration of the paroxysm in children seldom lasts more than a few minutes. In the adult it often does not exceed that period; but in many cases it lasts half an hour to two hours; while in others the greater part of the day passes before the paroxysm terminates.

It seldom happens that the paroxysm occurs but once. In the mildest case in the child it is commonly repeated three or four times in the course of the first three or four years of childhood, while in other cases it will occur three or four times in the day; and in severe cases the child is hardly out of one fit before it falls into another, till at length they gradually subside. In the adult the frequency of the fit varies extremely in different patients. In some instances there is an interval of several years; at others it returns annually, or every six months, or mensually, weekly, or even daily; while others will have twenty or thirty fits in the course of the same day. The period of the day at which the attack takes place is also very varied, for it may occur during the day, at night when asleep, or in the morning when just awakening.

**Causes.**—When epilepsy is the result of a powerful original tendency, it often occurs without any apparent cause, and when the patient is in his best health. The effects of moral causes in its production are so well known that Raphael has introduced into his picture of the Transfiguration a boy falling into an epileptic fit. Fright is considered a very common cause. Dr. Webster says that one of the worst cases he had ever seen was that of a young female who was frightened by seeing a young man dressed in a white sheet, personating a "ghost." Besides moral causes, errors in diet, excess of any kind, blows on the head, every structural or functional disease of the brain, and especially insanity; or any severe disease, as fever, or small-pox, are all powerful remote causes. Amongst soldiers in the Guards, in the experience of Dr. Graham Balfour, the fits, with one exception, seem to be brought on by gorging the stomach, usually with beer. He became so satisfied of this that, when turned out in the middle of the night to a man in a "fit," the excitation of vomiting generally relieved the paroxysm. In children the irritation of teething is the most common cause; and, indeed, in France epilepsy is often termed "*mal des enfans*." Puberty is the next most frequent period at which it

occurs ; and its frequency as a primary disease decreases from that time till fifty, when it again increases, from the tendency the brain now has to insanity and to structural disease.

[The influence of age in the production of epilepsy is strongly marked as is shown by the following table of Dr. Reynolds of cases collected by himself :

Age at Commencement.	Males.	Females.	Total.
Under 10 years, . . . . .	10	9	19
Between 10 and 20 years, . . . . .	66	40	106
Between 20 and 45 years, . . . . .	25	20	45
Over 45 years, . . . . .	1	1	2
	<hr/> 102	<hr/> 70	<hr/> 172

The chief facts to be noted are: (a) that the period of life embracing the processes of the second dentition and of the establishment of puberty—between 10 and 20 years of age—is the one of greatest frequency of the beginning of epilepsy ; (b) by far the larger number of the group showed their first symptoms of the disease between the ages of 13 and 17, inclusive ; (c) there is comparative immunity from attack between 25 and 35, the greater proportion of cases forming the third group having been seized at or about the age of 40 years.

When there is a marked hereditary taint, the disorder is developed at a somewhat earlier age.

Commencing.	Hereditary.	Non-hereditary.
Under 15 years, . . . . .	88.88	46.15
Above " . . . . .	16.56	58.82

Hereditary epilepsy shows itself at an earlier age among boys than among girls.]

As epilepsy is common in idiots whose heads are deformed, it has been affirmed that mankind become more liable to this disease in proportion as the facial angle approaches to  $70^{\circ}$ . There are many exceptions, however, to this statement. In infancy, and under seven years, epilepsy occurs in nearly equal proportions in both sexes. After puberty, when the distinction of sex is marked, some authors contend that it is more common in males than in females ; Dr. Elliotson thinks in the proportion of 27 to 11 ; Esquirol, however, states that, on comparing the number of epileptics at Bicêtre and at Salpêtrière, the number of women attacked was one-third greater than that of the men. In an analysis of the returns of the Registrar-General, given by Dr. Sieveking, with reference to the mortality from the disease in either sex during the past seven years, it appears that 6729 were males, and 6149 females, giving a relative proportion of 52.26 males to 47.73 females. Dr. Watson also states that he has seen "more epileptic boys and men than girls and women." Dr. Webster is of opinion that the disease is on the increase in this country.

Dr. Sieveking could trace hereditary tendency in 13.4 per cent. of his cases. Dr. Webster believes, from a combination of his own investigations with those of Esquirol and others, that *one-third* of the cases may be traced to hereditary descent. [Reynolds found only 12 per cent. of epileptics giving a distinct history of epilepsy

in other members of their family, and Delasiauve about the same number.] There is no doubt that a tendency to the disease is frequently hereditary. It may pass from parent to child; or it may skip over a generation or two, and appear in the grandchild or great-grandchild. The scrofulous diathesis is also a strong predisposing cause of epilepsy.

Of other exciting causes "there are certain vices," writes Dr. Watson, "which are justly considered as influential in aggravating, and even in creating, a disposition to epilepsy. Debauchery of all kinds, the habitual indulgence in intoxicating liquors; and, above all, the most powerful predisposing cause of any, not congenital, is *masturbation*—a vice which it is painful and difficult even to allude to in this manner, and still more difficult to make the subject of inquiry with a patient. But there is much reason to be certain that *many* cases of epilepsy owe their origin to this wretched and degrading habit; and more than one or two patients have voluntarily confessed to me their conviction that they had thus brought upon themselves the epileptic paroxysms for which they sought my advice" (Lect. xxxvi).

Sir Charles Locock attributes the great increase of the disease during late years to the cause last mentioned in the above quotation (Med.-Chir. Society's "Report," *Medical Times and Gazette*, May 23, 1857).

[The ætic influence of excessive venery, or of masturbation, in epilepsy, is undoubtedly overstated. The serious nervous disturbances to which both give rise are of a very different nature. The writer has seen many confirmed masturbators of both sexes without a suspicion of epilepsy. The proof is wanting that epilepsy and this particular infirmity have any special relationship to one another. Dr. Reynolds observes: "The one is a tolerably prevalent disease, the other a very widely distributed vice. There are multitudes of epileptics with regard to whom no such suspicion could ever be entertained; and there are, it is to be feared, much larger multitudes of masturbators who have never become epileptic. When, therefore, we find the two elements combined in the same individual it is necessary to observe some caution in our attempt to interpret their relations" (*l. c.*, p. 257).]

Irregularity and perverted state of the menstrual function, associated with hysteria, is another frequent source of the malady. The repression of eruptions, and especially those about the head, are also to be set down as causes which bring about the development of the disease: so are some of the *constitutional diseases*, such as *rheumatism*.

**Diagnosis.**—An immeasurable responsibility is associated with the diagnosis of such a disease; and, as already seen, the very slightest cue may be all which may be given to distinguish the epileptic state. It is especially to be distinguished from *apoplexy* and *hysteria*; and the following are the classical grounds of diagnosis as given by Dr. Reynolds:

1. *The Mental State* of the epileptic is thus far characteristic. By far the greater number exhibit a deficiency of the powers of the



Will in relation especially to *Thought, Emotion, Sensation, and Mobility*. The mind is inclined to wander in a half-abstracted state, and without energy of purpose. There is little or no power of attention or concentration of Thought, and there is a slowness of apprehension, with defective Memory. The Emotions and their expression are undirected and uncontrolled. The patient can only give unsatisfactory and often totally unmeaning accounts of sensations experienced. Something is felt to be wrong, but the place can hardly be fixed upon; and if the head, thorax, abdomen, or limbs are referred to, the patient is rarely able to express what he has experienced. A "working in my inside" is the comprehensive phrase commonly used to express their indescribable sensations. There is also a characteristic sluggishness and clumsiness of the voluntary movements. The walk and manner of the patient become ungainly. He rolls along rather than walks, stumbling over objects in his way, in an unnecessarily awkward manner. The countenance tends to be dull, expressionless, and morose. These phenomena may be so slight as almost to escape detection; and may in many cases be overcome by a determined effort of Will. Sometimes, on the other hand, they are extremely well marked, and graduate into utter stupidity and dementia with paralysis.

2. The *Motorial* and 3. *Sensorial* phenomena are such as have been described under the head of *symptoms*.

The attacks may be distinguished into two groups, namely: (1.) Those in which the loss of Consciousness is complete, associated with violent spasmodic movements. This group comprehends "*le haut mal*" of the French authors, and the laryngismal and tracheal epilepsy of Dr. Marshall Hall. (2.) Those in which one element predominates much over the other, even to its entire exclusion;—(a.) Attacks in which, loss of Consciousness being complete, there is little or no spasmodic movement. This class includes "*le petit mal*," or "*vertigo epileptiforme*," of the French, and the syncopal attacks of Dr. Marshall Hall; (b.) Attacks in which there is marked general or partial spasms of the muscles, somewhat of a tonic kind, but in which there is no appreciable loss of Consciousness. Such seizures constitute the "abortive" attacks of Dr. Marshall Hall.

One individual afflicted with epilepsy frequently presents every variety of these attacks, while any one form may exist alone; but the essential features of a fully expressed epileptic attack cannot be mistaken. They consist of—(1.) The simultaneous occurrence of the following symptoms: Complete loss of Consciousness, general quaitonic contraction of the muscles, impeded respiration, darkened face and surface generally, with distended jugulars, dilated pupil, distorted features, throbbing carotids; (2.) These phenomena are quickly followed by—persistent loss of Consciousness, clonic violent muscular contraction, laborious respiration, with tracheal gurgling noises; slight return of color in the face and body generally; oscillation of the pupil and eyeball; chewing movements of the jaws, and foaming at the mouth; (3.) The gradual cessation of these symptoms, and the production of another stage, marked by the following characters: Return of Consciousness for a short time, with an

aspect of astonishment, alarm, and suspicion; and then followed by drowsiness or profound coma; occasional semi-voluntary movements, such as change of position, labored slow respirations, with stertor and tracheal rattle, paleness of face, coldness of surface, with perspiration; the pupils often contracted, and the conjunctivæ injected; (4.) After sleep the patient becomes more natural in manner, and feels some headache and general soreness.

In the diagnosis of epilepsy it must be always borne in mind, especially in dealing with soldiers, seamen, prisoners, mendicants, and vagabonds, or others with whom powerful motives often prevail to feign diseases, that epilepsy is perhaps more frequently attempted to be copied than any other affection; and often with wonderful success. The means of detection consist,—(1.) In cross-examination as to the consistency or inconsistency of the accounts of the fits and general description of the attacks. This can only be well done when a perfect knowledge of the symptoms and grounds of diagnosis are familiar to the student. (2.) By observing whether or not a situation (favorable always to the malingerer) is chosen for the seizure. (3.) True epileptics seek retirement, and are frequently hurt by their falls. Feigned epileptics delight to exhibit in public, and rarely sustain any bodily injury. (4.) Let the eyes be closely observed. In true epilepsy they are partly open, with the eyeballs rolling and distorted, the pupils dilated, and not contracting by the stimulus of light. The feigning epileptic prefers to shut his eyes completely; and may occasionally be seen to open them to “take a peep,” so as to ascertain the effect of his exhibition. *His iris always acts on exposure to the light.* (5.) The skin of an impostor generally perspires from his exertions; that of a true epileptic in the paroxysm is generally cold. (6.) An impostor will not readily bite his tongue or void his excrements or urine. (7.) Tests peculiar to beadles and police constables consist in dropping melted wax upon the suspected feigning person, putting some gin into the eyes, pressing the thumb nail with force under that of the supposed impostor—an experiment productive of sudden, excruciating, and harmless pain. (8.) The mere speaking of or proposing some severe remedy in the presence of the patient is sometimes enough to detect imposition. Dr. Watson (from whose *Lectures* these statements have been condensed) specially recommends a very harmless and ingenious device—namely, in the hearing of the would-be patient, gravely to propose to pour *boiling water* on his legs as a remedy, and then to proceed actually to pour *cold water* upon them. Three humorous instances of detection are thus related by him: .

“Dr. Cheyne mentions an instance in which one table was placed upon another, and a soldier who was supposed to be shamming was laid upon the upper one while his paroxysm was on him. The fear of falling from such a height soon stopped his convulsions. Mr. Hutchinson relates the case of a sailor who was suspected to be a cheat, in whom the convulsions were instantly removed by blowing some fine Scotch snuff up his nostrils, through a quill. This brought on another kind of fit—namely, a fit of *sneezing*—which lasted nearly half an hour; and there was no return of

the epilepsy so long as Mr. Hutchinson remained in that ship. He tried the same expedient in cases of *real* epilepsy, but never could produce any similar effects, although the patients were not snuff-takers. There was a beggar in Paris who often fell into epileptic fits in the streets. One day some compassionate spectators, fearing that he might injure himself in his struggles, got a truss of straw and placed him upon it; but when he was in the height of the paroxysm, and performing remarkably well, they set fire to the straw, and he presently took to his heels" (Lect. xxxvi).

[The sphygmographic characters of the pulse in epilepsy have been studied by Dr. Voisin, and he proposes by their aid the detection of the true from the feigned disorder. Both in the *petit mal* and in full attacks the pulse-curves are large, the line of ascension is high, with well-marked diastole, and these last from thirty minutes to several hours. Similar pulse-traces could not be obtained from persons made to gesticulate violently, walk or run rapidly, or undergo other severe exertions. In cases of feigned epilepsy the pulse-traces bore no resemblance to those in the true disorder. The peculiar sphygmographic characters always lasting for some time after the fits, in suspected malingerers, the pulse-traces should be taken several times during the hour after the end of the attack (*Annales d'Hygiène Publique*, &c., Avril, 1868).

One of the most reliable diacritic signs between true and feigned epilepsy is the initial facial pallor, which cannot be imitated (TROUSSEAU).]

**Prognosis.**—Epileptic convulsions during teething generally subside about the second or third year; children, likewise, first seized between three and four years old, are often cured, or the disease often subsides at puberty, except when hereditary. Epileptics attacked after puberty are generally incurable, and especially when epilepsy is conjoined with insanity. Pregnant women attacked with epilepsy are in great danger. As to the positive certainty of any cure for the disease, a proper feeling of skepticism prevails. In the majority of cases no anatomical lesions exist, even after a long series of years in which the recurrence of the fits have been more or less constant. According to the belief of Dr. Sieveking and of others, a diathesis is necessary to their occurrence, and this may be suppressed or held in check; but it is very doubtful if it can be eradicated. Nevertheless, well-selected remedies have a power in repressing the paroxysm, and often of indefinitely postponing it;—more especially dietetic and regimenal treatment. The duration of the disease before treatment is commenced has an obvious influence over its curability. "It is seldom," writes Dr. Watson, "that any permanent ill effect can be noticed as having been left behind by any one single fit; but, unhappily, this cannot be said of their repetition." "More, probably, depends," he continues, "upon *repetition* of the fits than upon their precise *nature* or *severity*." "Every successive attack strengthens the *habit*, and renders the individual more obnoxious to future seizures; every arrest or postponement of a seizure is so much gain in favor of the patient, not only by avoiding the pain and risk of the isolated paroxysm, but still more by diminishing his future liability to the disease" (SIEVEKING, *l. c.*, p. 212).

Areteus, in describing the symptoms of epilepsy, has not neglected to speak of the baneful influence of this disease on the Intellect, of the Memory being lost, of the Imagination being impaired, and of the functions of the brain being, in many patients, so subverted that they fall into incurable insanity. Esquirol gives the cases of 385 epileptics under his care, in the Hospital Salpêtrière, and he states that four-fifths were more or less insane. The remaining fifth had preserved their reason, but, he adds, "a reason so broken!"

"A single paroxysm often leaves the patient in a worse condition than that in which it found him; but this is not perceptible to an ordinary observer until after the alteration has been rendered apparent by repeated fits and repeated small additions to the permanent injury. The friends of the patient remark that his Memory is enfeebled in proportion to the number of the attacks; that his mental power and intelligence decline. His features even assume by degrees a peculiar character; and too often he sinks into hopeless fatuity, utter imbecility, or confirmed insanity. It is this tendency which renders epilepsy so sad and fearful a disease. . . . Cases do occur in which epileptic persons preserve their faculties to a good old age; but those who are early epileptic do not often attain old age; and whenever the disease comes on, if it repeat itself frequently, it is followed much more often than not by impairment of the Mind, or by some apoplectic or paralytic affection" (Watson, Lecture xxxv).

Such are the phenomena associated with the paroxysms of epilepsy,—a disease not only frightful from the violence of the symptoms, but also from the serious effects it may produce on the moral character, as well as on the physical frame of the unhappy patient. While some may fall into the fire, and may be burnt to death, others fall into the water, and may be drowned, although the pool may be but a few inches deep (Cheyne). Bruises and fractured limbs are also not unfrequent. Many epileptics have a convulsive action or *tic* of the muscles of the face, or their legs waste, and are unable to support the weight of the body. In some instances the leg has been flexed under the thigh,—a contraction which has lasted more than a year; while in others the patient has become paralytic.

**Treatment.**—The treatment divides itself into what is to be done during the paroxysm, and subsequently during the interval.

When adults are laboring under the paroxysm, little, in general, can or ought to be done, except bringing the patient into fresh air, taking off what may be around the neck, and baring the chest, together with the more imperative duty of preventing the patient doing himself any injury. Bleeding, so often had recourse to, is rarely found beneficial, except, however, as regards females, in cases of suppressed menstruation. If, however, the paroxysm be greatly prolonged, the application of cold to the head, and opening the temporal artery, where symptoms of excessive cerebral congestion are obvious, may be of some service, as in cases recognized as plethoric.

It is of great importance to shorten the paroxysm as much as

possible. Dr. Sieveking recommends that a trial be made of the galvanic current during the fit. The conductors should be moistened sponges, so as to insure the passage of the current to the deeper-seated tissues.

The paroxysm over, the probable exciting cause of the paroxysm should be investigated, and if possible removed; the state of the bowels should be particularly inquired into and regulated, and leeches should be applied to the temples if the headache be severe. In women, also, the catamenia, if defective or excessive, should be remedied. These few simple rules are of the first importance, not only as removing the immediate inconveniences incident to the attack, but also as a means of prolonging the interval, and perhaps preventing its future occurrence. In a few instances the patient, by their adoption, is cured, and the prevailing principles of treatment in epilepsy mainly consist in local derivation, or counter-irritation directed to subdue cerebral congestion; and in the use of such tonic remedies as may be indicated by a careful inquiry into the condition of the individual organs, and how their several functions are performed. The intensity of the headache suggests more or less active counter-irritation by *blisters, dry or wet cupping, ointment of tartrate of antimony, setons, or even the actual cautery applied under chloroform*. The most usual remedies employed are *valerian, iron, [the several preparations of] zinc, quinine, musk, opium, asafoetida, the iodide and bromide of potassium, camphor, ether, and the preparations of turpentine*. The *nitrate of silver*, once esteemed a specific in this complaint, has not only failed, but, by occasionally staining the *rete mucosum* of a dingy blue, has often permanently disfigured the patient. [To this list may be added chloroform (MURRAY), the sulphate of copper, ammoniated copper, oxide of silver, chloride of silver, and digitalis.] Of the long catalogue which has been mentioned, each medicine is perhaps useful for a few weeks; but after that period its good effects are, for the most part, lost; so that it would appear to act rather mentally than physically, in removing the cause and altering the morbid tendency.

“Whatever remedies or course of treatment you pursue,” writes the late Dr. Todd, “do not appear to despond, or use any other language to the patient than that of hope. Avoid extravagant promises, as inconsistent with that love of truth which ought to characterize every professional man; but unless you have the strongest evidence against it, do not yourself, nor allow your patient, to abandon hope” (“Clinical Lectures,” *Medical Times*, August 12, 1854).

The employment of purgatives is indicated for the removal of waste matter to act as a derivative from the head, and expel foreign matters or worms from the intestines, and generally to regulate the bowels; and the laxatives most suited to epileptics are *rhubarb, compound colocynth pill, aloes, castor oil, taraxacum, sulphur in combination with magnesia or rhubarb*, and the Pullna bitter water imported from Bohemia, containing, as it does, *sulphates of soda, of lime, of potash, and of magnesia, carbonates of lime and of magnesia,*



*chloride of magnesium, and phosphates of lime* with *free carbonic acid*,—about 200 grains of saline matter in a pint, so that half a tumblerful taken in the morning generally produces a full pultaceous evacuation [or, bitter or common Kissingen water may be used].

The judicious use of an anthelmintic sometimes frees the patient from the disease as well as from a tape-worm or other parasite, which not unfrequently may be the *eccentric* source of the fits. Independently of its anthelmintic properties, however, both Drs. Watson and Sieveking strongly recommend the use of turpentine. It may be given in half-drachm doses every six hours.

The preparations of iron and zinc [and manganese] are the most useful tonics; and for the general principles on which iron may be administered, the reader is referred to the pages on *anæmia*.

[Dr. Trousseau believed that belladonna is the least inefficacious of all the remedies for epilepsy, and from its use not only obtained alleviation and improvement in many cases, but was able to count a certain number of cures (*Clinique Médicale*, t. ii, p. 95). His formula is:

Atropiæ Sulphatis, gr. j; Sp. Vini Gallici, ꝥc. M. One drop of this solution is to be given every day in the morning, if the attacks happen in the daytime; or, in the evening, if during the night; the dose to be increased by one drop for each succeeding month, and to be always taken at the same period of the day. It should not be pushed beyond the first toxical indications. If illy borne the dose should be increased only every second or third or fourth month. When improvement is apparent, the last dose (in quantity) given should be continued for some time, and then gradually lessened, and finally stopped, to be, after an interval, resumed.

“A year is scarcely sufficient to test the influence of belladonna, and, if in the succeeding year some improvement follows, the treatment is to be followed for two, three, and four years” (*l. c.*, p. 95).

Dr. Trousseau, however, in practice seems to have adopted a mixed treatment, giving belladonna in the morning, with the nitrate of silver, the sulphate of copper, and the lactate of zinc, in the evening, alternating these last every ten days.

R. Argenti Nitratis, gr. ij; Pulv. Acaciæ, Aquæ destil., aa q. s. Div. in pil. x. One every night.

R. Cupri Sulphatis, gr. xx; Pulv. Sacchari, ʒj. Div. in pil. xx. Two every night.

R. Zinci Lactatis, ʒj; Pulv. Sacchari, ʒij. Div. in pulv. xx. One every night. Or, the zinc may be made into pills with the conserve of roses.

Within the past ten years the *bromide of potassium* has been largely used in epilepsy, and the testimony to its utility in the disorder is uniform and strong. Dr. C. B. Radcliffe writes: “In the summer of 1858 I began to give this medicine almost promiscuously in cases of epilepsy, and epileptiform disorder, and from that time to this (1864) I have been continually finding fresh reasons for persevering in the practice. . . . The conclusion at which I have arrived is, that bromide of potassium is the only remedy in epilepsy upon which most dependence can be placed” (*Lectures upon Epilepsy, &c.*, Am. Ed., p. 201). The late Dr. Bazire, in a note to his translation of Trousseau’s *Clinical Lectures*, speaks thus of the experience of the physicians of the Hospital for the Epileptic and Paralyzed, London, and of his own, with this remedy: “The results ob-

tained are such as to warrant the conclusion that it is infinitely superior to all other remedies that have been recommended against epilepsy. It is certainly far superior to belladonna in its power of diminishing the frequency and severity of epileptic attacks and epileptiform seizures in general. Nay, more, of warding off the attacks, lengthening the intervals between them, and, in some cases, of bringing on a cure" (vol. i, p. 99). The most recent, and a very high, authority, Dr. J. Russell Reynolds, in his excellent article on Epilepsy (*System of Medicine*, vol. ii, 1868), says: "Bromide of potassium is the one medicine which has, so far as I know, proved of real service in the treatment of epilepsy. . . . In large doses it has scarcely ever failed to give much relief. . . . Given in doses, ranging from ten to thirty grains, three times daily, it has had these effects: In some cases it has completely cured the patient, and the cure has been permanent for years, and is so now. In others it has arrested the attacks, so that none have occurred for periods varying from a few months to two or three years; but on the omission of the medicine the seizures have returned. In such cases the attacks have again ceased on the readministration of the medicine. In a third series of cases it has diminished the frequency and severity of the seizures, but has not removed them altogether; the patients while taking the bromide have had one-half or one-third the number to which they were habituated. Such patients have gone back to the old frequency of recurrence when the drug has been omitted, and have again improved when it has been readministered. In a fourth, but very much smaller number, it has been good for a time, and then has appeared to cease; and in a fifth, and yet smaller proportion, it has been apparently without any appreciable effect. . . . It is to be demonstrated that there is yet something 'specific' in the action of KBr." (p. 280-1).

Dr. Voisin, of the Salpêtrière Hospital, says, that it does not cure absolutely, but diminishes the disorder in a marked degree, lessening and even suppressing the nervous erethism (*Bul. de Thérapeutique*, 1867).

Dr. Edward Fox reports fifty-two cases in which bromide of potassium in not less than twenty-grain doses was found "satisfactory," and ten cases in which it wholly failed, in one of which belladonna was more successful (*St. George's Hospital Reports*, vol. ii, 1867).

Dr. Brown-Sequard, than whom few have had as large experience in this disease, began to use the bromide of potassium in large doses towards the end of 1860. He writes: "I was soon led to associate the iodide of potassium with the bromide: and it became almost at once evident, that, in most cases of epilepsy (whether idiopathic, symptomatic, or sympathetic, but especially in that form, which is much more common than it is admitted to be, in which that convulsive affection is allied with, or due to, a congestion of the base of the brain or its meninges), these two remedies did more good than either of them alone. By the end of 1861, after I had ascertained that the bromide of ammonium has a special therapeutic influence in cases of congestion of the medulla oblongata and of the upper parts of the spinal cord, I began to associate that salt with the bromide and iodide of potassium in the treatment of epilepsy. . . . That mode of treatment has been submitted to an extensive trial, which leaves no doubt as regards its superiority. Although certainly it does not often cure permanently, it usually diminishes considerably the violence and the frequency of the attacks, and is much more successful than the various modes of treatment by the best remedies against epilepsy, such as atropine or belladonna, the sulphate of copper, the nitrate of silver, strychnine, valerian, zinc, digitalis, or the means of counter-irri-

tation by applications of ice, moxas, croton oil, &c., on the spine or the head" (*Lectures on the Diagnosis and Treatment of Functional Nervous Diseases*, 1868, p. 81-2). His usual prescription is:

*R.* Potassii Iodidi, ʒj; Potassii Bromidi, ʒj; Ammonii Bromidi, ʒijss.; Potassæ Bicarbonatis, ʒij; Infus. Calumbæ, fʒvj. *M.*

A teaspoonful before each of the three meals, and three teaspoonfuls at bedtime, in a little water.

In syphilitic epilepsy, the iodide of potassium is increased to five or six drachms. Where the attacks begin with violent laryngismus, the bromide of ammonium is raised to three or four drachms, and the bromide of potassium diminished by two drachms. The following rules are laid down:

The quantity of these medicines to be taken daily should be large enough to produce an evident though not complete anæsthesia of the fauces and upper parts of the pharynx and larynx, this quantity varying, with the patient's idiosyncrasy, from forty-five to eighty grains of the bromide of potassium, and from twenty-eight to forty-five grains of the bromide of ammonium, when only one of these salts is given, and a lesser quantity of each, but especially of the ammonium, when given together.

These remedies very rarely produce a good effect in epilepsy without causing an acne-like eruption in the face, arms, neck, shoulders, and there seems to be a positive relation between the intensity of the eruption and their efficacy; it is important, therefore, when there is no eruption, and also when it begins to disappear, to increase the dose, unless the dose given in the twenty-four hours is already so large that any increase brings on great sleepiness in the daytime, a decided lack of will and of mental activity, dulness of the senses, drooping of the head, considerable weakness of body, and a somewhat tottering gait.

It is never safe for a patient taking these drugs to be even one day without them, so long as he has not been quite free from the attacks for at least fifteen months.

The debilitating effect of the bromides ought to be lessened by the use of strychnine, arsenic, the oxide of silver, ammonia, cod-liver oil, cold douches or shower-baths, and wine and a nourishing diet. There is an antagonism between strychnine and the bromides, and, when prescribed together, the dose of the bromides must be increased.

Iron and quinine should not be given in epilepsy, unless complicated with anæmia or malarial poisoning, except, perhaps, the double salt of the citrate of iron and strychnine.

A gentle purge every five or six weeks maintains the power of the bromides (BROWN-SEQUARD, *l. c.*, p. 84-6).

In night-seizures the bromide of potassium would seem to have less influence than in day-fits (DUCKWORTH WILLIAMS).

From the writer's personal experience with the bromide of potassium in epilepsy, from all the published evidence of its effects in the disorder which is definite, and not vague and general, and from the inquiries he has made, and what he has seen in the practice of physicians who have long and properly employed it, he must repeat what he wrote in the first edition of this work 1866: he has never seen, nor heard of, nor found recorded, one single well-authenticated case of the permanent cure of epilepsy, under its use alone, or when combined with either of the other salts of potassium or ammonium, however long or judiciously administered. As the evidence now stands with respect to the value of this medicine in

the treatment of epilepsy, it may be said, that it most often exercises at once a marked influence over the severity of the paroxysms, and lengthens the intervals between them; that in cases of the disorder apparently pathogenetically similar to those that have been benefited by its employment, it may have no good effect; that in cases of long standing it frequently fails to give any relief; that its control over the disorder seems to be in inverse proportion to its duration; that when its administration is continued for a long time, the remedial power at first shown is apt to diminish and finally to cease altogether; and, lastly, that there is, as yet, no proof of its curative property.

Clinical familiarity with epilepsy, and a history of its therapeutics, go to show that there are no grounds for belief in any specific against this affection, and the physician who promises a cure, or even relief, by means of any one remedy in this disorder, runs the risk of damaging not only himself, but his art. It cannot, however, be doubted, that, in a certain number of cases of epilepsy not apparently caused by any coarse cerebral lesion, the severity and frequency of the seizures may be greatly abridged, so that the sufferers may be brought to enjoy often a great degree of immunity and comfort; the proportion of such cases to the whole number afflicted, is, perhaps, few, but is still sufficiently numerous to give hope and encourage to further trial. Such results, however, can only be surely gained by means which will increase and develop vital power generally. But it should be borne in mind that a tonic treatment does not alone consist in the administration of a tonic drug. Whilst many cases of epilepsy demand the use of tonic medicines to fulfil certain present indications, they should in no sense be looked on as curative, or even remedial, but only as adjuvants, and when they have done their work should be laid aside. Of this class of remedies, none perhaps is more valuable than arsenic, given in minute doses. Where there is anæmia, iron or manganese is required. But a general restorative system must be adopted and persevered in. The food should contain, as soon as the state of the digestive organs will permit, a certain proportion of fatty and oily constituents, and in most cases, cod-liver oil may be given with advantage. The hypophosphites, as a vehicle for the introduction of phosphorus into the system, would seem to be of service in improving the general nutrition. The maintenance of the activity of the cutaneous function is of the first importance. In the beginning an occasional vapor or hot-air bath may be taken; afterwards tepid salt or fresh-water baths, followed by a general grooming of the skin. Exercise in the open air, and gymnastics, both measured by the strength of the patient, are to be insisted upon, as well as such means as will promote the expansion of the lung-tissue. With physical training, mental and moral training should be combined.

Such are the general principles of the rational or restorative treatment of epilepsy, which, it is believed, will give a larger measure of success than a reliance upon any one of the innumerable specifics that have had questionable and temporary repute, and which have been happily styled by Dr. Trousseau, "therapeutic rubbish" (*fatras de moyens thérapeutiques*). But if a successful issue is to be had, perseverance and confidence, both on the part of the physician and the patient, are chief conditions of success, and this should be fairly stated at the outset to the sufferer and his friends.]

Although the medical treatment of the adult epileptic is so unsatisfactory, yet the treatment of epilepsy occurring in children

during teething is almost always successful.\* The practice, on the child falling into a fit, is immediately to place it into a warm bath, and to pour cold water on its head, to lance its gums, and to administer an enema. These means generally restore the child; and the after-treatment consists in the application of a few leeches to the head, to purge the bowels with calomel, either alone or combined with some other cathartic, and to diminish the quantity of the diet. These means are all that the case will admit of, and they are very generally successful. Bleeding should be used with great moderation; for these fits in children seldom affect the Intellect, and have a tendency to subside spontaneously in a very few months. When depletion, however, is carried to excess, the child's health is greatly broken, and the probability is that the brain is rendered more irritable and the fits more frequent. Slight opiates, by soothing the irritation of the mouth, are useful in every stage of the complaint; and when greatly debilitated, some mild tonic treatment may be necessary to restore the little sufferer.

**Dietetic Treatment.**—In the adult the diet should be light, and the patient should live temperately. He should live by rule, for the tendency of epileptics is to gorge themselves with food or drink. He should rise early, and take regular exercise in the open air, keeping his head cool and his feet warm. The diet of an infant so affected should be, if possible, its mother's milk, with or without arrowroot. If above three or four years of age, its diet should consist entirely of farinaceous or of other light vegetable food.

#### ACUTE AND CHRONIC HYDROCEPHALUS.

**Definition.**—*An effusion of serous fluid between the membranes of the brain, or into its ventricles. The affection may be acute, congenital, or caused by disease or defective development of the brain during foetal life, or it may occur at some period in after-life as an original disease.*

**Pathology.**—Hydrocephalus was very little known till Dr. Whytt published his *Observations on Dropsy of the Brain* in 1768; but since that period, Dr. Fothergill, Dr. Watson, Dr. Cheyne, and a large number of other writers, have contributed to illustrate its nature.

There are a few cases in which effusion of serum into the ventricles, or into the cavity of the arachnoid, is unaccompanied by any morbid appearance of the brain or of its membranes whatever; and thus there are many instances in which hydrocephalus is not demonstrably inflammatory. More commonly, however, some lesion of the brain or its membrane does exist. Thus the substance of the brain is often marked with more bloody points than usual; the *septum lucidum*, the *fornix*, and other parts forming the walls of the ventricles, are often found in a state of softening—sometimes so soft that Golis gives a case in which water could be expressed from it as from a sponge. The membranes also are sometimes con-

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\* [This form of convulsion has no relation pathogenetically with true epilepsy, which happens as well in children as in adults (see p. 355), and the text is calculated to mislead.—EDITOR.]



gested, or opaque and thickened, with spots of lymph, evidently the effect of a low inflammation.

The quantity of fluid effused varies from a few teaspoonfuls to seven or eight ounces, and of this the greater part is generally contained in the lateral ventricles, which from this cause are often so enlarged and distended that the finger placed on the brain immediately over the ventricle, is sensible of a distinct fluctuation, while the anterior portion of the fornix is often so raised as to cause a free communication with the third ventricle, and perhaps with the fourth—at least, the effused fluid is found likewise distending those cavities. The quantity of fluid effused between the membranes is also often very great, sometimes filling the whole cavity of the arachnoid as well as the ventricles. Dr. Abercrombie has found serum effused even between the cranium and dura mater, and so also have other observers—a circumstance hardly known in any other disease. The choroid plexus or ventricular membrane, although in general pale and healthy, yet sometimes has the intercellular tissue so infiltrated that it appears studded with small cysts.

The more frequent occurrences are tubercles in the brain or membranes, and some congestion, perhaps, of the mucous membrane of the intestinal canal; but whether the latter is a primary affection, or the result of the violent medicines which are sometimes had recourse to in this affection, is not determined. The peculiar green color of the stools, so frequent in this disease, seems to be imparted to them in the lower portion of the intestine, the fecal contents of the upper portion being of a pale drab color, while the bile in the gall-bladder is of a yellow color.

[Pathogenetically there is nothing in common between acute and chronic hydrocephalus. In the latter, which is a dropsy of the cranium, most often congenital, the symptoms during life are due to the mechanical action of a variable amount of fluid, causing enlargement of the head. The former is not a common disorder, what is described under the name being generally tubercular or granular meningitis; but there can be no doubt of the occasional existence of a disorder, due to certain nutritional changes (so-called inflammatory action) of the membranes of the ventricles, followed by more or less effusion into those cavities, and to which the name of acute hydrocephalus may properly be limited. It is rapid in its course, ending within a few days to a week or two. The post-mortem appearances in the brain are thus described by Dr. Beveridge, of Aberdeen (*Medical Times and Gazette*, vol. ii, 1868): Surface and substance of the brain natural. The fluid in the ventricles is clear, and a granular exudation-layer covers the floor of the ventricles. This layer, thin, soft, grayish, and easily overlooked on account of its resemblance to the parts which it rests on, is composed of granules, exudation-corpuscles, and imperfectly formed cells; it is seen covering the optic thalamus and corpus striatum, and is most abundant towards the forepart of the ventricle, where, in the angle between the rounded anterior end of the corpus striatum and the crura of the fornix, it accumulates to the greatest extent, so as sometimes almost to fill it. At or opposite the foramen of Monro it causes a very peculiar appearance: there the exudation, so far from closing the opening, prolongs it on either side into the ventricle, converting it into a sort

of short canal, which opens by oval apertures with sharply defined borders, placed not vertically, but so obliquely as to be nearly horizontal, quite clear of the edge of the fornix and visible at once on opening the ventricle from above; so that instead of its being necessary to look underneath the apex of the fornix to see the foramen, the openings of it appear like two oval eyes between the fornix and the corpus striatum, and are in plain view without disturbing or touching the parts in any way. This appearance gives the impression as if a soft granular layer had settled down on the floor of the ventricle, filling up its angles and interstices, but prevented from covering the foramen of Monro by the steady set of a current through that opening.]

The first thing that strikes us on examining those patients who suffer from the chronic form of the disease is the enormous size of the head. The adult head averages about twenty-two inches in circumference. Dr. Bacon gives the case of a child whose head at three months had attained the enormous size of twenty-nine inches in circumference (*Med.-Chir. Trans.*, vol. iii). The head of Cardinal, a celebrated hydrocephalic man about London, long in St. Thomas's Hospital, and who afterwards died at Guy's, measured thirty-three inches and a half. There are instances, however, in which the cranium has been found unusually small, and of a conical shape, the sutures being closed before birth; and in these cases the children are still-born, or die shortly after delivery. When the disease comes on at later periods of life, and after the sutures are closed, the size of the skull is natural, the cavities within the brain distended, and its substance wasted and anæmic.

The *form* of the hydrocephalic head is also sometimes very irregular, one side being much larger than the other, while the bases of the orbits are for the most part convex instead of concave, thrusting the eye unnaturally forwards. On cutting through the skull the bones are found to be remarkably thin and transparent. The sutures, although generally closed towards the base of the skull, are commonly separated from each other by a wide extent of membrane at their superior portions. If, however, the patient should survive for several years, the membranous portion becomes ossified by a number of points forming "ossa Wormiana," and the sutures are thus partially closed. In some very few instances the sutures not only close, but the bones of the skull have a morbid thickness.

The membranes of the brain are generally thickened, and the fluid found effused either into the cavity of the arachnoid, into a cyst, or into the ventricles of the brain. When the fluid is contained within the cavity of the arachnoid, the brain is sometimes so compressed that there are instances in which hardly a vestige of that organ remains. A singular and rare variety of this affection occurs when the arachnoid sometimes protrudes through the *fontanelle* or open suture, and the *dura mater* and integuments yielding, a pyramidal bag, with its apex downwards, forms externally, which hangs low down the back like a jelly-bag.

When the effused fluid is contained in the ventricles, those cavities are found exceedingly dilated. The convolutions have no depressions, but appear unfolded. The *corpus callosum* is much

raised, the *septum lucidum* is torn and destroyed, or the gray commissure destroyed, and the white commissure elongated to the extent of an inch, so that the ventricles communicate. The parts at the base of the brain also, as the *corpora striata* and *thalami optici*, have scarcely any existence. In fact, the brain seems expanded into a large sac, in which the medullary and cortical substances are so confounded as to be undistinguishable. In Dr. Bacon's case the brain and membranes, even the dura mater, had ruptured, and a probe passed easily through the ethmoid bone into the nose, by whose orifices a considerable dribbling of the fluid took place during life. Golis met with a case in which the water was contained in a cyst the size of a goose's egg, situated between the hemispheres of the brain of a child aged six years, and who died, the cyst being entire.

The quantity of fluid contained in the cranium in cases of chronic hydrocephalus varies from a few ounces to a few pounds. In the case of Cardinal it was found to exceed ten pints (nine pints in the cavity of the arachnoid, and one pint in the ventricles). Other cases have been, however, recorded in which the quantity has amounted to twenty pints.

Authors have greatly differed as to the nature of this disease. Some, considering it a mere increase of fluid from functional activity, have named it *dropsy of the brain*, while others have as constantly referred it to an inflammatory origin; but they have generally concurred in describing an acute and chronic form of the disease.

[Mr. Prescott Hewett has described the deviations of the vault and base of the skull found in chronic hydrocephalus. The former are quite obvious; those of the latter less so. He calls attention to the orbital plates, which are either driven downwards, presenting a plane surface oblique from before backwards, or they may be perpendicular, or even convex, bulging into the orbit, so as to reduce it to a mere chink. This deviation gives proof that the dropsy is in the ventricles, and not merely in the sac of the arachnoid, although ventricular dropsy may happen without this sign. In arachnoideal dropsy the fluid is limited to the upper and lateral parts of the surface of the brain, and therefore cannot press upon the bones at the base. The orbital deviation may be recognized during life: the eyes, more or less driven out of their sockets, have a marked downward direction; a great part of the pupil is hidden beneath the lower lid; and the white of the eye is much more uncovered than usual. The next important deviations are those of the middle fossæ, most marked in the lateral regions, where, by the bulging out of the bones, the base in some cases undergoes extension and strange alterations. Sometimes a large pouch bulges outwards, and projects into the cheek, giving it the look of being blown outwards. The origin of such pouches is in the descending horn of the lateral ventricle. A case of Vrolik's is cited, in which this portion was cut off from the common cavity and dilated, forming an expansion in the corresponding part of the skull (*St. George's Hospital Reports*, vol. i, 1866).]

**Symptoms.**—Acute hydrocephalus is divided into three stages; the first stage, according to Dr. Cheyne, being that of increased

irritability; the second, that of diminished sensibility; and the third, that of convulsions or palsy.

The first stage may be either sudden in its attack, or be preceded for several days by giddiness, so that the child stumbles or falls at play; by a furred tongue, constipated bowels, and perhaps offensive breath. At length the senses of sight and of hearing become morbidly acute; he starts at slight noises—complains of intermitting headache—rests his head on his nurse's lap—occasionally complains momentarily of his head—and then, after a time, rises up and plays again. As this stage advances, the pulse rises, the skin is hot and dry, the urine scanty, the stomach irritable, the bowels constipated, perhaps painful, the stools black and offensive, while the brow is knit, and the pupil of the eye contracted or expanded. The most remarkable feature, however, is a great fretfulness of temper, so that the child is not merely pettish but quarrelsome. If he sleeps, his sleep is short, uneasy, moaning; he also grinds his teeth, rolls his head, and when he wakes up it is with a scream. "We are led to suspect some deeply-seated evil from the frantic screams and complaints of the head and belly, alternating with stupor, or rather lowness, and unwillingness to be roused" (CHEYNE).

In much of this description some of the phenomena of *meningitis* may be recognized, and, doubtless, a great number of the acute cases are associated with this morbid state already described (page 283, *ante*).

The second stage commences when effusion has taken place. The pulse, instead of being rapid, is then as slow, perhaps slower, than natural; but this is chiefly when the patient is in a horizontal position, for if he attempts to sit up, it again becomes rapid. The sickness is also abated; nevertheless, the child lies in a state of stupor and of great unwillingness to be moved, with his eyes half-closed, dull, and heavy, or perhaps staring or squinting, the pupil being still contracted or expanded,—and he often suffers from double vision. The stupor, however, is still interrupted by exclamations or shrill piercing screams, while the tremulous hand of the little sufferer is incessantly engaged in picking his nose or mouth.

In the third stage the patient either sinks or recovers. If the event is unfavorable, the pulse again rises, the eye becomes red and dim, and the child, delirious, is often attacked by partial or general convulsions; or one limb or one side may be palsied. From this point the powers of life gradually sink, till at last death closes the scene. If the patient should fortunately recover, the stupor subsides, the countenance becomes more natural, the bowels more regular, the secretion of urine perfectly restored, and at length his health, though long broken, is gradually re-established. The duration of this acute form of the disease is estimated at about three weeks, each stage averaging about a week.

There are two forms of chronic hydrocephalus, the internal and the external, or hydrocephalus in which the membranes protrude. In either case, when this disease is fully formed, whether it be congenital or subsequent to birth, the child is generally of the most

feeble intellect, irascible, often epileptic, and of extreme muscular debility, so that, if not palsied, he is hardly able to walk. Dr. Baillie met with an instance of chronic hydrocephalus in a man aged fifty-six, and whose ventricles contained six ounces of serous fluid. His chief symptoms were pain in the head, and a loss of Memory so great that he could recollect only five words, which he continually reiterated to express all his wants. Cardinal, whose case has been mentioned, had more Memory, and he prided himself, says Dr. Elliotson, in being able to say, "The Belief," but he usually stumbled when he got to "Pontius Pilate." This man was epileptic, of very feeble intellect, and so irascible as to be always quarrelling with the patients, and would have been extremely difficult to manage except for his muscular debility. Heberden, however, mentions a case in which eight ounces of water were found in the ventricles of the brain, and yet no symptoms of hydrocephalus existed during life.

[In chronic hydrocephalus the blood-circulation and nutrition of the fundus of the eye suffer changes, which are explained by the compression of the cerebral substance from the accumulated fluid. As the fluid collects and the pressure increases, the ophthalmoscope shows: (1.) Greater vascularity of the papilla and choroid, with dilatation of the veins, which preserve their natural color. (2.) An increase in the number of the vessels of the choroid. (3.) Partial or total serous infiltration of the papilla. (4.) Atrophy of the choroid and its vessels. (5.) More or less marked atrophy of the optic nerve, which may be complete (BOUCHUT).]

**Causes.**—The remote causes of hydrocephalus are often extremely obscure; but exposure to cold or heat, errors in diet, falls or blows on the head, the retrocession of a cutaneous eruption, or the extension of an inflammation of the ear, are among the most common. Disordered function of the liver or alimentary canal is also a frequent cause, and so is dentition, or the presence of worms; and the circumstance of a child being seized in consequence of its feet having by accident been put into a bath of boiling water will show that any other extreme irritation will equally produce it. Many morbid poisons also will occasion it, as that of *scarlet fever*, of *pertussis*, or of *measles*; of constitutional diseases, *tuberculosis* is the most common exciting cause.

The ages of infancy and childhood exercise a most remarkable predisposing influence upon this disease. At those periods the rapid growth of the brain, the irritation of dentition, and the great susceptibility of the nervous system generally, are all powerful causes of determination of blood to the head. The greatest number of attacks, according to Percival, occur between the second and the fifth year; or, as a more general law, the disease occurs from infancy to twelve years of age. Children with large heads and precocious intellects, and more especially those of a scrofulous diathesis, are its most frequent victims. One warning may be learned from this disease—namely, that it is said to be most common in the children of parents addicted to drunkenness, and from this cause it often runs in families.



Although it has been stated that hydrocephalus may occur during foetal life, and is more common in the early periods of infancy and childhood, yet adult age is not altogether free from it; and Golis has mentioned three cases of persons attacked in old age, two of whom were above seventy, while the other, perhaps less advanced in life, suffered from this affection for ten years. It seems sometimes to run in families; at least, Frank mentions a family of seven children, all of whom were born with this disease; and Golis another, in which six children were aborted hydrocephalic at six months; while three others, born at the full period, were attacked shortly after birth.

**Diagnosis.**—Acute hydrocephalus is distinguished from typhoid fever by the screaming, rolling of the head, grinding of the teeth, and by the absence of the peculiar state of the tongue which marks the latter disease. The external characters of chronic hydrocephalus are so extremely marked that it is hardly possible to mistake them. The diagnosis generally is established from the general external appearance and phenomena. There is always intense marasmus, although food may be taken.

There is a morbid state resembling the disease which is neither *acute* nor *chronic*, to which Dr. Watson gives the name of *spurious hydrocephalus*. To three great men of the past we owe our knowledge of this condition—namely, Gooch, Abercrombie, and Marshall Hall. In children, from a few months to two or three years of age, of small make and of delicate health, from exposure to debilitating causes, this morbid state not uncommonly supervenes. It is indicated by heaviness of the head and drowsiness. The child lies on its nurse's lap unable or unwilling to raise the head. It seems half asleep, one moment opening its eyes and the next closing them again, with a remarkable expression of languor: the eyes are unattracted by any object put before them, and the pupils remain unmoved on the approach of light. The breathing is irregular, sighs are occasionally expressed, and the voice is husky. The tongue is slightly white, the skin is not hot, but sometimes colder than natural. In some cases there is now and then a slight and transient flush. Diarrhoea has often existed for some time, or the child has been severely purged by medicines, or having been weaned, has ceased to thrive since its change of food. When this condition of things occurs in a more elderly child, it has been generally brought about by depletion, by loss of blood, or by medicines. As the more marked symptoms are sometimes ushered in by extreme irritability and a feeble attempt at reaction, cases of this kind (which Dr. Marshall Hall named “the hydrocephalaoid disease”) have not unfrequently been mistaken for acute hydrocephalus, and treated accordingly, the patient being generally leeched out of its life. In very young children the diagnosis is sometimes very difficult between congestion and exhaustion, between fulness and emptiness.

Dr. Watson, from whom these characteristic notes are taken, suggests the following test: “As a guide to diagnosis, take notice of the state of the unclosed fontanelle. If the symptoms proceed from plethora, or inflammation, or an approach to inflammation, you will

find the surface of the fontanelle convex and prominent, and you may safely employ and expect benefit from depletion. If, on the other hand, the symptoms originate in emptiness and want of support, the surface of the fontanelle will be concave and depressed; and in that case leeches or other evacnants will do harm, and you must prescribe better diet,—ammonia,” brandy in arrowroot, milk from the mother’s breast, if possible, and all such means as will tend to foster and nourish an infant.

**Prognosis.**—The chances of recovery in the first stage are many, if the patient be properly treated. At any subsequent period the prognosis is most unfavorable, and Dr. Cheyne estimates the loss from confirmed hydrocephalus at six to one, and perhaps this is near the truth. If acute hydrocephalus shall unequivocally declare itself, it will be rapid, and almost inevitably fatal (ALISON). The immediate danger in the cases of chronic hydrocephalus is not great, but few patients survive the age of puberty; Cardinal, however, lived to the age of thirty-two. Aurival speaks of another instance which reached forty-five; and Gall of a third who survived till fifty-four.

**Treatment.**—The acute form of hydrocephalus is only successfully combated in the first or earliest stage. As the first symptoms are those characteristic of inflammation of the brain, and especially of its meninges, there is perhaps no class of cases in which the sanative powers of judicious bloodletting become so apparent as in children in whom the disease has been observed early and carefully watched. Dr. Alison relates the following highly illustrative case:

“A boy aged eight years, of rather delicate habit, and who had complained occasionally for some weeks of headache and disordered bowels, temporarily relieved by laxatives, calomel, antacids, and a careful regimen, but recurring, and attended with gradually increasing febrile symptoms, and *shooting* pain of head; impatience both of light and sound; pulse gradually rising to 108, distinctly sharp, and beginning to intermit, and then nausea and vomiting, not referable to any medicine taken, but gradually increasing, until it recurred every time that he sat up in bed, for nearly twenty-four hours; and a slight but quite perceptible squint showing itself. The full action of laxatives and one application of leeches, as well as cold to the head, having failed to make any impression on this course of things, he was bled at the arm (which in such circumstances and at that age has often appeared to me distinctly preferable) to *twelve ounces*, and the blood was *sizy*. I do not know what further evidence we could have had of the existence of such inflammation within the cranium as would infallibly, if let alone, have gone on within a few days to delirium, stupor, dilated pupil, slow pulse, succeeded by very frequent pulse, convulsions, and death, and have left after it the usual appearances of the *acute hydrocephalus* (of Whytt, Cullen, and Abercrombie, now described), dryness of the membranes on the surface of the brain, distension of the ventricles of the brain with serum, and some of the marks of inflammatory action, either on the membranes or surface of the brain. Instead of this, however, I am quite certain that from the time of the bleeding at the arm this boy *never once vomited*, that the intermission of the pulse was *never again observed*, nor did he again complain of lights or sounds. The pain of the head, although less violent, continued in the evening of the same day, and *twelve leeches were applied* within eight

hours after the bleeding, and from that time he never once complained of this symptom, nor admitted that he felt it; and *from this moment he recovered perfectly*, and much more rapidly than he had done from much slighter febrile attacks previously; neither has he suffered from that time to this (now thirty years) any return of serious disease" (*Edin. Med. Journal*, p. 777, March, 1856).

In short, all the symptoms vanished in twenty-four hours, most of them during the flow of blood, and never recurred,—a change, under the circumstances, so sudden and sanative as is rarely if ever seen after the use of any other remedy for the same combination of symptoms. With reference to the sanative influence of general bloodletting in children for inflammatory diseases, if performed at the outset, Dr. West observes that such depletion is as important a remedy as in the adult; nor will the most energetic employment of any other antiphlogistic measure enable us to dispense with bloodletting. In a healthy child two years old, a vein (if easily found) may be opened in the arm, and *four ounces* of blood allowed to flow, if faintness be not earlier produced, without our having any reason to apprehend that the plan adopted is too energetic. The immediate effect produced is greater than that which follows local depletion, and the quantity of blood abstracted is less (West, *Diseases of Children*).

The first thing to be done by way of medicine is to purge the patient. The purgative is not of great moment, provided it acts freely. Some prefer two to five grains of gamboge, others five grains of calomel with thirty grains of compound jalap powder. Such doses are to be followed up by a black draught, or the sulphate of magnesia. So difficult is it to obtain the action of purgatives in this disease, that doses of three times the strength for adults have been in some instances required; but such large doses are never to be given until the inefficiency of smaller ones has been ascertained (Dr. Watson's *Lectures*, No. xxvi).

The stools are generally black, or extremely offensive; and this state of the bowels corrected, the disease, if sympathetic, often ceases. If, however, the head be not relieved, some leeches should be applied to the temples, and the head should be shaved and surrounded with some cold evaporating lotion, as with a towel dipped in cold spring water, or in vinegar and water, or in solution of the muriate of ammonia.

If the disease be advanced, no efficient treatment has as yet been determined. The symptoms do not yield to the lancet like those of simple inflammation. *Mercury* has also been used to a great extent, but with little success. In urgent cases, for instance, mercurial ointment has been rubbed on the back and thighs, even in very young children, to the extent of half a drachm to a drachm three or four times in the twenty-four hours. *Calomel* also has been rubbed on the gums to the extent of three or four grains every four or five hours, and it has likewise been given by the mouth in doses of two grains every third or fourth hour. *Mercury* given in these large doses, it must be remarked, seldom produces salivation; for Dr. Clark says he never saw that effect in children

under three years of age, except in three cases. But the remedy is not successful, and more generally produces *spinage-like* stools, and irritates the alimentary canal. In France the mercurial treatment has been so unsuccessful that some practitioners have even tried a most opposite remedy, namely, *quinine*, but the result has been equally fatal. *Blisters*, *moxas*, and other modes of cauterization have been used as auxiliary treatment, but without apparent benefit.

During the course of the disease the diet should be slops and light puddings.

In congenital hydrocephalus the unassisted efforts of nature seem incapable of effecting a cure, and it is extremely problematical if medicine is of any use. When, however, the case is deemed hopeless, the propriety of evacuating the water by means of an operation may be entertained. Golis has given the names of twenty-seven writers who have expressed themselves in favor of it, especially if the fluid be slowly evacuated, and at several repetitions of the operation; yet he himself, along with seven or eight others, proscribed it altogether as cruel and useless. But it has been successful; and when the operation is performed, it seems an axiom that the fluid should be allowed to escape gradually, for otherwise extreme faintness and collapse may be expected. In such cases small doses of ammonia, or a few teaspoonfuls of brandy and water, may revive the little patient. Should reaction take place, however, at a subsequent period, a few leeches and a cold lotion ought to be applied to the head. It seems also determined that the younger the child the greater are the chances of success; for if it lives a few years the sutures of the cranium, though open at the top, are united by bone towards the base of the skull, and thus present a mechanical obstacle to their closure; consequently, the operation is more apt to fail. If this disease should occur in after-life, blisters and mercury to salivation are the remedies mostly relied on.

SUNSTROKE. [SYN., HEAT FEVER; HEATSTROKE; INSOLATION.]

LATIN, *Solis ictus*, [*Insolatio*]; FRENCH, *Coup de soleil*; GERMAN, *Sonnenstich*, [*Hitzschlag*]; ITALIAN, —.

**Definition.**—An affection of the nervous system, associated with vertigo, and sometimes with headache, or the gradual accession of listlessness and torpidity, with a desire to lie down (LONGMORE, BARCLAY). These febrile phenomena may culminate in more or less sudden and complete insensibility, without the power of sense or motion, the breathing rapid, and getting more and more noisy as death approaches. Convulsions of the extremities usher in a complete state of coma, in which the patient gradually dies. The approach of death is indicated by the failure of the heart's action, the fluttering of the pulse, the irregularity of the respiration; and the fatal event may supervene within five minutes to a few hours after the disease has become fully expressed. Death is either by

syncope, apnoea, or by a combination of the two. In cases which recover, various sequelæ are apt to supervene, such as forms of paralysis, more or less complete, choreic movements, melancholia, and other forms of insanity.

**Pathology and Symptoms.**—This singular and fatal affection of the nervous system has been described under a great variety of names—*e. g.*, *Heat apoplexy*, *Heat asphyxia*, *Coup de soleil*, *Insolatio*, *Ictus solis*, and lastly, *Erythismus tropicus*. Nevertheless the full expression of the disease not unfrequently occurs at midnight. The name implies a common, and certainly a most powerfully exciting cause of a disease which has been variously and erroneously described as of the nature of apoplexy, or of some form of continued fever.

A very great variety of views have been put forward regarding the pathology of this remarkable disease; and the following account of its nature is based upon the very interesting accounts which have been given by many Indian medical officers, and especially by Deputy Inspector-General Mr. Longmore, when on duty with the 19th Regiment at Barrackpore; by Surgeon Butler at Mean Meer; and by Dr. Barclay, of the 43d Light Infantry, while on the march from Jubbulpore to Calpee.

Instances of the occurrence of *sunstroke*, and the circumstances under which it has been observed to occur, will best convey any idea of the nature of this disease.

One of the earliest accounts of *sunstroke*, in which its nature was distinctly recognized, is that given by Mr. Russell, of the 73d Regiment, while in medical charge of the 68th, in May, 1834. The regiment had then recently arrived in Madras—a fine corps of men in robust health. The funeral of a general officer being about to take place, the men were marched out at an early hour in the afternoon, buttoned up in red coats and military stocks, at a season, too, when the hot land winds had just set in, rendering the atmosphere dry and suffocating even under the shelter of a roof, and when the sun's rays were excessively powerful. The funeral procession forthwith advanced, and after having proceeded two or three miles, several men fell down senseless. As many as eight or nine were brought into hospital that evening, and many more on the following day. Three men died—one on the spot and two within a few hours. The symptoms observed (and they were alike in the three cases) were, first, excessive thirst and a sense of faintness; then difficulty of breathing, stertor, coma, lividity of the face; and in one whom Dr. Russell examined, contraction of the pupil. The remainder of the cases (in which the attack was slighter, and the powers of reaction perhaps greater, or at all events sufficiently great) rallied; and the attack in them ran on into either an ephemeral or more continued form of fever. The symptoms of these cases did not more nearly resemble each other than did the post-mortem appearances. The brain was healthy in all; no congestion or accumulation of blood was observable. A very small quantity of serum was effused under the base of one; but in all three the lungs were congested, even to blackness, through their entire extent; and so densely loaded were they, that complete obstruction



must have taken place. There was also an accumulation of blood in the right side of the heart, and in the great vessels (*Medical Gazette*, "Graves's Clinical Lectures," vol. i, p. 181).

A nearly parallel example is related by Sir Ranald Martin, as having occurred in the experience of Dr. Miligan, of the 63d Foot, from the exposure of his corps to the sun during a military funeral at Madras. The greater number of the men were in the prime of life; but there were some old soldiers who had served twenty years and upwards in the West Indies, and who were much broken down by service and intemperate habits. The entire corps had just arrived from the Australian colonies, where spirituous liquors can be had on easy terms. The regiment landed at Madras in the month of May; and from the date of the "untoward circumstance" of the funeral, the hospital became filled with cases of fever. Two men dropped down and died on the very day of the funeral, and for several days afterwards the fever cases augmented considerably (*The Influence of Tropical Climates*, p. 205).

The dreadful march of the 48d Light Infantry from Jubbulpore to Calpee furnishes the most instructive and melancholy examples of *sunstroke*. It must be remembered that the regiment had previously marched throughout the greater part of the length of the Madras Presidency—a march extending from the 24th December, 1857, to the 17th of April, 1858, when the regiment arrived at Jubbulpore. During this first march the regiment enjoyed great immunity from sickness, owing to the sanitary precautions which had been taken. The march, however, told seriously upon the men in general. They lost condition, and in a great measure their robust appearance, and were in urgent need of rest. But after a rest of five days only, they had to commence the march from Jubbulpore to Nagode, a distance of 163 miles, arriving there on the 8th of May. The heat on the march was excessive, and it told very much on the health of the men, already exhausted as they were by a previous march of almost unexampled length. After having been *four months and thirteen days in the field, and after they had marched 969 miles*, a fatal case occurred; and from that date cases of *sunstroke* gradually increased in frequency. At Nagode the regiment remained eight days; and although the indications of exhaustion in the altered looks of the men, their loss of flesh, and their evidently failing strength, were so obvious that they forced themselves on the observation of every one, *yet the men were ordered to continue their march to Banda*. The periodical hot winds were blowing at the time, day and night, with scarcely any intermission, and the heat of the weather was almost unbearable. After leaving Nagode they were obliged to encamp for four days in the bottom of a deep and narrow ravine, with nearly precipitous sides upwards of a mile in height. The heat in this valley was insufferable, and exceeded anything the regiment had ever been exposed to before, or that they were exposed to afterwards. The thermometer varied during the day from 115° Fahr. to 118° Fahr. in the largest tents, and in the smaller ones it reached 127° Fahr. Night brought but little relief. On one occasion Dr. Barclay ob-

served the thermometer standing at 105° Fahr. at midnight. Such was the overpowering effect of the heat in this "valley of the shadow of death," that even some of the natives were struck down, and died with all the symptoms of *sunstroke*, in less than an hour. The number of cases among the men of the regiment, especially during the first day, was very great. They were carried into the hospital tents at every hour of the day and night; and although a large proportion of them recovered, two officers and eleven men were buried under one tree in the neighborhood of the camp. Marching was resumed on the 24th of May. On the 27th the regiment arrived at Banda, a distance of about 100 miles from Nagode, having lost during the march two officers and nineteen men.

The health of the regiment improved daily during a short stay at Banda; but the men were still in a miserable state of exhaustion when the regiment again began to march for Kirwee, on the 3d of June. At Kirwee five men died from *sunstroke*, and on the return to Banda two more died. The regiment now marched for Humeerpore on the 28th of June, *en route* for Calpee. The weather was again excessively hot, and the men suffered very severely. They were by this time completely worn out and prostrated. There was scarcely a man in the regiment whose strength was not reduced to a level with that of a child; and the officers were not in a very much better plight. Many men broke down altogether, and had to be carried as it could best be managed—in *doolies*, in sick carts, or on baggage and commissariat carts after they were filled. It was painful to see many others who, a few months before, had been in robust health and full of vigor, staggering from weakness as they endeavored to keep up with the column, throwing themselves down completely exhausted at every halt, and scarcely able to rise from the ground when the "*assembly*" sounded. Calpee was reached on the 5th of July, and between the 28th of June and the 7th of July one officer and five men had died.

These details are given for the purpose of showing *the effects of protracted exposure to intense heat in a body of men in the field debilitated by fatigue and want of rest*. It may now be shown how the effects of protracted exposure to intense heat in a body of men may operate upon them when in *quarters*; as described by Deputy Inspector-General Longmore. He records sixteen cases of *sunstroke* as having occurred in the 19th Regiment between the 23d of May and the 14th of June, 1858, when they were quartered at Barrackpore. The period was marked by an unusually elevated degree of temperature, and generally by great dryness of the atmosphere. The quarters were of a temporary and imperfect nature, some of them being merely hired *bungalows*.

Of the sixteen cases, five occurred in non-commissioned officers, and eleven in privates of the regiment, the proportion attacked being greatly more among the former than among the latter: their occupations, especially when on orderly duty, caused them to be more exposed than the men with whom they lived. Simple expos-

ure to the external atmosphere and the solar rays were not of themselves sufficient to induce the disease. Of the five non-commissioned officers attacked, two were on duty at the time as orderly sergeants; one had had fever for several days, but had not been exposed to the sun on the day of attack; one had been slightly exposed; the fifth not at all. Of the privates, eight were attacked by the disease within doors, and three while on sentry. No cases occurred of sudden sunstroke,—i. e., of insensibility instantly induced by the direct rays of the sun in a man previously healthy.

The characteristic feature with regard to atmospheric temperature, when Mr. Longmore's cases were observed, was the little variation of it night or day. There was no rain; and the ground and buildings became so heated that, long after sunset, the radiation of heat maintained a high temperature within doors. Prolonged high atmospheric temperature was recognized as the essential cause of the attack; but nervous depression from solar exposure, fatigue, and previous illness, were associated with that prime or essential cause.

But a most important element of causation is still to be mentioned,—namely, *the influence of vitiated air when men are congregated without sufficient ventilation*. One-third of the cases, and nearly half the deaths, occurred under such circumstances; so that, *in quarters*, predisposing causes of *sunstroke* may be comprised in the following conditions: (1.) Prolonged atmospheric heat, with a dry and rarefied state of the air; (2.) Nervous exhaustion; (3.) A contaminated atmosphere (LONGMORE, TAYLOR); while (4.) An increase of the average prevailing temperature sufficed to act as the more immediate exciting cause for the development of *sunstroke*.

The experience of Surgeon Butler at Mean Meer has led him to write, that “when the thermometer ranges beyond 98° Fahr. in crowded barracks, cases of apoplexy almost invariably occur.” Dr. Crawford also, in writing on *sunstroke*, notices an electric condition of the atmosphere as influencing the respirability of the air, and refers especially to that peculiar state “in which the hairs of a horse's tail repel each other, in which the hairs of the head stand on end, in which a man exposed to its influence becomes irritable, headachy, and restless, without knowing exactly what is the matter with him. Such a state of atmosphere will generally be found to exist in localities where cases of *sunstroke* occur, whether such localities be the crowded barrack, in the still more crowded cantonment, the tented field, or the march in column through the still valley, the deep gorge, or the thick forest.” Dr. Barclay notices that cases of *sunstroke* occur with increased frequency immediately before a thunderstorm, and that they cease as soon as the electrical discharge has taken place.

From the accounts given by these several observers it seems clear, as Dr. Barclay observes, that the symptoms of the disease are liable to be greatly modified by accidental causes, and that those phenomena which are most prominent under one set of circumstances are either absent altogether under another, or so very much less urgent as scarcely to attract observation—that the disease, in fact,

varies in several important points according to the nature of the circumstances in which it occurs.

The phenomena which at any time have presented themselves under the various circumstances detailed by Mr. Longmore, Dr. Barclay, and others, may be summed up as follows: When careful observations are made, the affection seems gradually induced by protracted exposure to extreme heat in a dry and rarefied air, combined with a vitiated atmosphere from defective ventilation, or with physical exertions of an arduous character, implying excessive fatigue of extreme duration, so as to bring about great debility and weariness of the body. Sleep at last cannot be obtained, or it is greatly interrupted, and of short duration. Deterioration of the general health is thus progressive, while altered looks and loss of flesh indicate extreme exhaustion. The skin becomes rough and scaly, and perspiration ceases. The heat of the surface increases to an intense degree; but accurate records of the temperature of the body in cases of *sunstroke* are not yet in existence. The bowels tend to become obstinately constipated. The urine becomes copious, and the calls to pass it are frequent; or even incontinence may prevail (LONGMORE, BARCLAY). Under such circumstances those morbid phenomena intervene which are noticed in the definition.

Most observers are agreed that intemperance cannot be charged with being the immediate exciting cause of the disease, although it is a universally acknowledged predisposing cause. The men of Dr. Barclay's regiment were extremely temperate, robust, and well-formed; and Mr. Longmore gives a no less favorable account of his men as to temperance. The drunkard, indeed, is rarely to be found in the ranks under circumstances favorable to the development of *sunstroke*. He is either in prison, or skulking at some dépôt, or in some hospital, where he may be left for want of transport; or, if with a regiment, "the pains" come on opportunely, to save him from fatigue and danger. The vascular system of an habitual drinker soon shows unmistakable indications of over-stimulation in the suffused eye, the bloated countenance, the profound sleep which follows the slightest indulgence, and the subacute meningitis which sooner or later supervenes in India (CRAWFORD). Such are not the kind of cases which furnish the deaths from *sunstroke*. When exposed to the stimulus of a tropical sun, such cases may sink under it, as they would under any other great excitement; and a debauch or an ephemeral fever will alike predispose a man to an attack of *sunstroke*, inasmuch as both may bring about that state of nervous depression which seems essential to the occurrence of this disease.

In Dr. Barclay's experience the deterioration of the health of his men in the field was progressive. For a long time before the occurrence of the first case of *sunstroke* every one had suffered more or less from "prickly heat," the severity of the affection being, as a rule, in proportion to the amount of perspiration from each individual; and when the heat of the weather became still more intense, one of the first symptoms of its producing an injurious effect was the gradual disappearance of this cutaneous eruption, the skin be-

coming rough and scaly, and the perspiration ceasing. In many cases the interruption of perspiration appeared to be complete—not the slightest feeling of dampness being perceptible in any part of the dress at any period of the day. On this point Dr. Simpson observed to Dr. Morehead,—“Every man seized with sunstroke, and who could answer questions, informed me that he had not perspired for a greater or less extent of time,—sometimes not for days,—previous to being attacked, and that he had enjoyed good health as long as he had perspired, but that on the perspiration being checked, he felt dull and listless, and unable to take much exertion without making a great effort” (*Researches on Disease in India*, p. 617). The heat of the surface became at the same time much increased. The bowels became obstinately constipated. The appetite gradually failed; and a feeling of nausea was generally complained of, the sight of food often exciting loathing. In other instances there was nearly complete anorexia.

The urine became copious and limpid, and the calls to pass it frequent; so much so that Mr. Longmore especially notices a frequent desire to micturate as constant in all the cases in which there was an opportunity of ascertaining the point; and he justly remarks, “If this symptom should prove to be a general precursor of the attack, it might be rendered valuable as an indication of the approaching danger, which, by early and proper care, might then probably be averted; and its presence at a time when heat-apoplexy was prevalent would make the surgeon alert to obviate the more serious symptoms which might be expected to follow” (*Indian Annals*, vol. vi, 1860, p. 399). Sleep from the first was much interrupted, and the periods during which it could be obtained became gradually shorter, until at length no sleep could be got during the night. The pulse was frequent, sharp, and small. The tongue white posteriorly, but seldom foul or dry. Thirst was seldom very urgent. Vertigo was frequently complained of, but headache rarely; and, as a general rule, there was no complaint of pain. The general complaint was extreme debility, weariness, and prostration on any exertion; vertigo, nausea, and in many cases incontinence of urine, more particularly after exposure to the sun, “I cannot hold my water,” being a very general answer to the first inquiries of the medical officer. These premonitory symptoms were attended with rapid and progressive emaciation. No one during the march (in the ravines of Northern Bundelcund, so well described by Dr. Barclay) became the subject of *sunstroke* without having previously suffered from some or all of these premonitory phenomena. Such phenomena, however, prevailed in a much larger proportion of cases in which symptoms of *sunstroke* did not supervene at all; and they seem to stand somewhat in the same relation to *sunstroke* that the premonitory diarrhoea does to *cholera*.

The attacks of *sunstroke* came on generally when the men were in their tents, during the day sometimes, but in several instances during the night, and only in one instance on the line of march. The patient had generally been lying down, often seemingly asleep, or trying to induce sleep, when the attention of his comrades would



be directed to him by his hurried and heavy breathing, and on attempting to rouse him he was found to be insensible. In other instances he started up suddenly, and attempted to escape from the tent, staggering about, and struggling violently when laid hold of, evidently much alarmed, and anxious to escape from some imaginary object of terror; but in a very few minutes he became insensible.

In one or two instances the first symptoms of the disease was reputed to have been an uncontrollable burst of laughter, without any apparent cause, and in sad enough circumstances,—insensibility and death speedily following. In a few instances the patient would come to hospital himself, or with the assistance of his comrades, exhibiting some of the symptoms (premonitory) already detailed, when insensibility or a tendency to sleep would gradually supervene.

[In 60 cases observed at the New York Hospital in 1854 by Dr. H. S. Swift (*New York Jour. of Med.*, 1854), the seizure was sudden in the midst of work, “with pain in the head, a sense of fulness and oppression in the epigastrium, occasionally nausea and vomiting, general feeling of weakness, especially of the lower extremities, vertigo, dimness of vision, and insensibility. Surrounding objects appeared of uniform color; in a great majority of cases this was blue or purple. In one instance everything was red, in another green, and in another white.”]

The time of the day at which most of the cases occurred is important in connection with the elevation of temperature. Ten out of sixteen cases occurred between the hours of 2 and 5 o'clock P.M., and five cases between 5 and 9.30 P.M. (LONGMORE). In Mr. Cotton's experience at Meerut the seizure usually happened towards evening. The thermometer generally indicated the maximum temperature of the twenty-four hours to be about 4 P.M.; and the variation in the thermometer-range was very slight from 2 P.M. to sunset; and even for some hours after sunset the temperature scarcely at all declined (LONGMORE).

[In Swift's 60 cases the attack happened in 3 between 8 and 11 A.M.; in 40, between 11 A.M. and 4 P.M.; and in 17, between 4 and 9 P.M.; the large majority of cases occurring during the maximum of the day temperature.]

When the disease was fully expressed, the symptoms were constant and regular. The patient lay on his back, without sense or motion, breathing rapidly, and as death approached, more and more noisily, from the vibration of the *uvula* and the *velum pendulum palati*; and although such *stertor* was present in most of the fatal cases, yet it never approached in degree to that which is common in true apoplexy. The eyes were *fixed, and turned slightly upwards*, becoming gradually more and more glassy, as if from the formation of a film over the cornea; the *pupils greatly contracted* (generally to the size of the head of a pin); the *conjunctivæ pinky, the color gradually becoming deeper*; the congestion at first deep-

seated, and the first symptom of it a pinky zone around the cornea, the superficial vessels afterwards becoming affected. *The face was invariably pale*; the surface dry, harsh, and burning to the touch, far beyond what Dr. Barclay ever felt in any other disease. The heat of skin greatly exceeded that occurring in pneumonia, and was without parallel in the experience of Mr. Longmore.

[The range of body-temperature is shown in the following table :

Cases and Years.	Temperature (Fahr.)	Observer.
1844, . . . . .	99°; 104° after death, . . .	Dowler.
1845, . . . . .	111°, 112°, 113°, . . . . .	"
1847, . . . . .	109°, . . . . .	"
1847, . . . . .	106°, . . . . .	"
1848, . . . . .	105°; 110° after death, . . .	"
1863, . . . . .	108°, . . . . .	H. C. Wood,
1863, . . . . .	108°; after death, . . . . .	"
1863, . . . . .	109°, . . . . .	"
1863, . . . . .	109°, . . . . .	"
1863, . . . . .	104°, . . . . .	"
1863, . . . . .	106°, . . . . .	"
1863, . . . . .	109°; after death, . . . . .	"
1866, . . . . .	109.5°, . . . . .	Levick.
1866, . . . . .	109°, . . . . .	"
1866, . . . . .	106°, . . . . .	"
1866, . . . . .	112°; after death, . . . . .	"
1867, . . . . .	105.5°, . . . . .	"
1866, . . . . .	109°, 108.15°, 108.8°, . . .	Bäumler.]

The *heart's* action was very rapid and sharp (BARCLAY), excited and irregular (LONGMORE), the impulse and pulsation in the carotids being perceptible to the eye from a considerable distance. The pulse was frequent and sharp, and at first moderately full, giving the idea of a thinner fluid than blood circulating beneath the finger. Frothy mucus, sometimes clear, at other times of a brown color, was in most instances ejected from the mouth and nose for some time before death, and often in large quantity.

When the disease was about to terminate fatally, the heart's action soon began to fail, the pulse to flutter, and the breathing to be irregular; and in a period varying from a few minutes to a few hours, death closed the scene. In nearly all the fatal cases there was occasional convulsive muscular movements of the extremities up to the time of death. These generally ushered in a state of complete coma, in which the patient gradually sank (LONGMORE). [In 60 cases observed by Swift there were convulsions in 24.]

In a large proportion of the cases, however, from the commencement of the attack to its termination in death, the patient never moved a limb, or even an eyelid; and a comparatively small number of the cases on the march from Jubbulpore to Calpee were from the first attended with convulsions. These generally began in the upper extremities, or in the muscles of the face, and in some cases they did not extend farther, the patient either becoming rapidly insensible or recovering. In other instances they extended to the whole of the voluntary muscles, and were of the most violent description, ceasing frequently for from two or three to fifteen or twenty minutes, and recurring again with increased severity. In a few in-

stances the nervous irritability seemed as much increased as in hydrophobia; and some patients appeared to be in a state analogous to *somnambulism*. Although unconscious, and incapable of understanding or of answering questions, yet the countenance indicated the greatest terror,—the eyes rolled wildly about; and a few drops of water poured on the ground near him were sufficient to throw him into the most violent convulsions, and to elicit from him screams of agony. In most of these cases the convulsions ceased some time before death. The symptoms then became identical with those which characterize the ordinary course of the disease. In a few, however, the convulsions continued to the last; and in one or two death took place when the body was still contorted by them (BARCLAY).

The mortality from *insolatio* is equal to 42.734 per cent. (BARCLAY); or 43.3 per cent. (BUTLER); one-half the cases (SWIFT).

[Sir Charles Napier states that out of 44 cases of sunstroke which happened at Nassurpoor on June 15, 1843, 43 were fatal. Of 53 cases reported amongst the prisoners at Andersonville in June, July, and August, 1864, all were fatal (Dr. JOSEPH JONES, *United States Sanitary Commission Medical Memoirs*, p. 581).

Dr. Morehead gives the following table:

Observer.	Cases Treated.	Deaths.
Mr. Hill's (collected cases), . . . . .	504	259
Dr. Taylor, Gazeepore, . . . . .	115	16
Mr. Longmore, Barrackpore, . . . . .	16	7
Mr. Lofthouse, 14th Light Dragoons, . . . . .	80	10
Dr. Simpson, 71st Regiment, . . . . .	25	6
Mr. Ewing, 95th Regiment, . . . . .	60	17
Field Hospital, Hansi, . . . . .	29	10
	<hr/> 929	<hr/> 825]

In the cases which terminate favorably a gradual remission of the urgent symptoms takes place; but the irregularity of the heart's action and oppressed breathing may persist during the next day; and if the patient has been exposed to the influence of malaria, paroxysmal febrile phenomena may supervene.

The patient cannot be considered free from danger till the skin becomes cool and moist (SIMPSON, BARCLAY); indeed, a relapse of all the worst phenomena may occur even after free perspiration and sleep have been procured. Dr. Crawford relates such a case: An orderly being left in charge of the patient during the night, with instructions to keep a cold lotion to his head, and to call the surgeon in the event of any change occurring, no matter how slight; the surgeon, visiting the hospital at one in the morning, when summoned to see another patient then taken ill, found the orderly asleep, and, to his horror, his patient moribund,—the face swollen, of a dark livid color, the eyes protruding from their sockets, with stertorous breathing and spasmodic twitchings of the muscles of the chest and arms. He died shortly afterwards.

**Morbid Anatomy.**—In Mr. Longmore's cases, in which a vitiated atmosphere, from the want of ventilation, was associated with the

extreme heat, the appearances after death were those usually found in death by asphyxia—namely, excessive engorgement of the lungs, amounting to complete obstruction of the pulmonary circulation. Some parts of the lungs had all the appearance of true interstitial apoplexy. [In six autopsies made by Dr. H. C. Wood the heart was rigidly contracted.] Cerebral congestion was less marked in character and less constant in amount, and it seemed to be secondary to the failure of the due performance of pulmonary functions, resulting, perhaps (as Mr. Longmore suggests), from loss of tone in the vessels, and from the enfeebled action of the heart, consequent upon the imperfectly oxygenated blood it was receiving. The congestion of the head was generally expressed by engorgement of the vessels of the *pia mater*, and *choroid plexus*, and by numerous *blood puncta* in the substance of the cerebrum, as shown on section. The appearance of the brain indicated generally sanguineous determination without serous effusion; and when serous effusion had taken place, it was generally into the cavities of the lateral ventricles, and sometimes into the subarachnoid space.

[In six fatal cases examined by Dr. H. C. Wood, the venous trunks of the cerebral meninges were engorged, but there was no congestion of the brain substance. Dr. Flint says there was marked cerebral congestion in all the autopsies at the Bellevue Hospital which he saw. In Dr. Levick's case, reported in the *Pennsylvania Hospital Reports*, vol. i, the vessels of the dura mater were found full of dark liquid blood, which was easily pushed along by the handle of the scalpel. The brain was softened, breaking down with its own weight, or by the slightest pressure, and there were myriads of minute dull-red points on the broken surfaces. The plexus choroides was of a dark purple hue; there was no effusion into the ventricles, and on the under surface of the brain a little uncoagulated blood. The liver in the latter case was full of liquid blood, which poured from it, when it was pressed, like water from a saturated sponge. The spleen was of the usual size, and of very nearly natural consistence.

In Dr. Christian Bäumler's case (one of heatstroke in a sugar refinery), the veins of the membranes of the brain were greatly congested; the brain rather moist, with large blood-spots, but otherwise normal (*Medical Gazette and Times*, August 1, 1868).]

The blood is always fluid (MOREHEAD).

[In Dr. Levick's case the blood, under the microscope, exhibited one or two rouleaux, with patches of irregularly-arranged, shrivelled, crenated corpuscles; its reaction was faintly alkaline. Dr. H. C. Wood found the blood to have an acid reaction.]

Various opinions are entertained with regard to the mode of action of the various agents which combine to produce this singular affection of the nervous system. The phenomena during life and the post-mortem appearances are in accordance with death from coma, slowly induced, or from syncope. The manner in which heat acts in the production of such asphyxia as is seen in cases of *sunstroke* has been variously interpreted by writers on the subject; and observations are very much to be desired as to the

exact range of temperature of the body-heat in such cases. It is known, however, to be excessive; and the increased temperature of the blood, which results from prolonged exposure to great heat, must have a deleterious influence upon the constitution during the metamorphosis of tissue going on under such circumstances. Intense heat applied to the whole body tends to produce death by *syncope*, as in concussion of the brain (ALISON). Heat acting on the peripheral distribution of the nerves, and accumulating in the system, as it seems to do in *sunstroke*, produces such an effect on the heart, the lungs, and the brain, as to produce the phenomena of *syncope* and *coma*.

The pre-existing cutaneous derangement in all the cases, the total inaction of the skin, its dryness and intense heat, betoken an accumulation of heat in the blood which cannot fail to influence the delicate textures of the brain and lungs. The "embarrassed and heavy breathing;" the "sense of weight over the sternum" (LONGMORE); the "hurried and heavy breathing" (BARCLAY); the "catching at the chest;" the "constricted feeling, as if of approaching suffocation, caused by wind at a temperature of 112° Fahr." (McGRIGOR), indicate that physiological state of "*anxietas*" which prompts to such acts of inspiration as are seen on the approach of *syncope*, or of *apnœa*, from depression of the nervous influence of the *medulla oblongata*.

Pollution of the blood, from the prolonged continuance of function under such circumstances as impair the normal action of the skin, the lungs, and the kidney, produced by the atmospheric conditions already described, and the phenomena of the disease in its severe form, denote the culmination of functional efforts to get rid of the rapidly accumulating elements of disintegration which must have resulted,—as indicated by progressive emaciation, augmentation of animal heat, and total suppression of the cutaneous function. Cases of sunstroke occurring "in quarters" seem under such circumstances to die from *coma*, inducing *apnœa*; and the most common complication is undoubtedly pulmonary congestion from oppression of the *medulla oblongata*, evidences of which are found on post-mortem examination in the majority of fatal cases, as originally pointed out by Dr. Marcus Hill (*Indian Annals*, vol. iii, October, 1855); and afterwards by Mr. Longmore (*l. c.*, vol. vi, July, 1859, p. 396). In those cases of death by *coma*, the most striking point in the post-mortem appearances is the enormous congestion of the lungs; in which Dr. Parkes remarks, that although he has dissected men in a very large number of diseases, both in India and in England, he has never seen anything like the enormous congestion observed in two or three cases of this kind (*Practical Hygiene*, p. 345).

Pulmonary engorgement, however, is not always present. On the march and in the field the functional phenomena are chiefly of the cerebral, spinal, and sympathetic systems, as indicated by a painful state of nervous irritability from over-stimulation of intense heat. The long exposure of the eyes to the glare of the sun in camp may account for the more constant occurrence of their congested state



in cases of *sunstroke* in the field, compared with such cases occurring in quarters.

The pallor of the face and of the surface generally seems also to be more an attribute of the disease in the field than in quarters, the cases being probably more anæmic in the field and more plethoric in quarters (BARCLAY). Practically, it has been observed, as Dr. Barclay points out, that there are at least four different ways in which death may occur in cases of *sunstroke*; which are necessary to be borne in mind with regard to the line of treatment necessary to follow.

(1.) The affections of the nervous system alone, more particularly those which occur during active exertion "in the sun," when the intense heat acts on the surface with the greatest power, producing at last a condition similar to severe concussion, and more or less instantaneous death by *syncope* (ALISON, CRAWFORD, BARCLAY, MOREHEAD). (2.) Death may be prolonged, when pulmonary complication may occur from destruction, more or less complete, of the pulmonary circulation, and death by *asphyxia* ensues. Or, (3.) There may occur cerebral congestion, and death by *coma*. These states may, and generally do, coexist together; and symptoms of either may predominate. (4.) Recovering from the immediate effects of these conditions, the patient may die two or three days afterwards, a febrile attack succeeding, with serous effusion within the cranium.

[Dr. James J. Levick, of Philadelphia, in the first volume of the *Pennsylvania Hospital Reports*, has discriminated between two pathological conditions, which are usually spoken of, and described, as *sunstroke*. In one, there is simple loss of nerve-force caused by over-exertion during exposure to a high heat. It happens alike in the open air, under the almost vertical rays of the summer's sun, and in the close and heated atmosphere of the sugar refinery, furnace-room of steamers, laundry, and crowded barracks. Its symptoms are a feeble and moderately frequent pulse, moist skin, head generally hotter than the trunk, little or no change in the pupil, and a tendency to *syncope* on the slightest exertion. In such cases there is no apparent lesion either of the solids or of the blood, and they generally recover under proper treatment.

But another and severer form of disorder is a common result from exposure to the sun's rays, or to a high temperature of the atmosphere either within or without doors. It comes on suddenly, or with slight premonition, as giddiness, confused blending of colors, and sharp pain in the head; the subject falls unconscious, with stertorous breathing, restlessness, and convulsions. The skin is pungently hot, the body-temperature ranges from 104° Fahr. to 110° Fahr., and the pulse is so quick that it often cannot be counted. When fatal it is generally within six hours from the seizure, though death may be almost immediate. Its victims are the young, the robust, and unacclimated. Drs. Levick, Gerhard, and H. C. Wood hold that the most obvious, and, as they believe, constant and essential pathological condition is an *altered* state of the blood; a loss of its life, seen not only in the change of its physical properties, in the non-coagulating fibrine, in the shrunken corpuscles, in their escape from the vessels, forming myriads of petechiæ, but also in its rapid putrefaction, beginning even before general death had occurred. To this form of *sunstroke* Dr. George B. Wood has given the name of *Heat Fever*, and Dr.

Levick believes that, "whether determined by the resemblance of its symptoms during life or of its phenomena in death, it finds its proper nosological classification in close association with typhus fever, spotted or petechial fever, and the plague" (*l. c.*, p. 383.)]

**Causes.**—Heat, and atmospheric conditions of the nature already indicated, seem to concur with the following predisposing circumstances to induce cases of *sunstroke*: (1.) Plethora and unacclimation; (2.) Debilitating causes of every kind, particularly such as lower the tone of the nervous system or increase its irritability, *e. g.*, excessive fatigue and prolonged exposure in extreme temperature, prolonged marches, bad ventilation in tropical temperatures; (3.) A febrile state, from whatever cause. As a general rule, Dr. Barclay found that plethoric men incur greater danger from exposure than others; (4.) Intemperate habits; (5.) Exposure to an atmosphere highly charged with electricity (CRAWFORD, BARCLAY); (6.) During the season of prevalence of sunstroke the temperature would seem to have ranged from 96° to 120° Fahr. in the shade—extremes of atmospheric heat chiefly observed on the Coromandel Coast, Central India, the Northwest Provinces, Scinde, and the Punjaub (MOREHEAD, *l. c.*, p. 615).

The effects of the constant stimulation of excessive heat in producing nervous irritability and pervigilium cannot be doubted (MARCH, BARCLAY), and Indian medical officers can bear witness, from personal experience, to the extreme misery resulting from such excessive stimulation, in combination with the conditions already mentioned.

[The official statistics of sunstroke in the United States Armies during the first two years of the civil war were: For the year ending June, 1862, 420 cases and 17 deaths, of which there were in the Atlantic Region 253 cases and 7 deaths, in the Central Region 163 cases and 9 deaths, and in the Pacific Region 4 cases and 1 death. For the year ending June, 1863, there were 1199 cases and 57 deaths, of which 651 cases and 15 deaths were in the Atlantic Region, 547 cases and 42 deaths in the Central Region, and one case and no death in the Pacific Region (*Circular No. 6, Surgeon-General's Office, November, 1865.*)

During nineteen months, January, 1862, to July, 1863, 64 cases of sunstroke were recorded in the field reports of the Confederate forces serving in Virginia; 16 in North Carolina; 33 in the department of South Carolina, Georgia, and Florida; 13 amongst the forces serving along the Gulf of Mexico, and 58 amongst the large armies, in and about Vicksburg, and in Mississippi, Alabama, Kentucky, and Tennessee; or 184 cases in an average monthly mean strength of 160,231 officers and men—eleven-hundredths of 1 per cent.

For the following tables the writer is indebted to the kindness of Dr. ELISHA HARRIS, Registrar of Vital Statistics, Metropolitan Board of Health.

TABLE SHOWING THE NUMBER OF DEATHS FROM SUNSTROKE IN THE CITY OF NEW YORK, FROM 1855 TO 1867, INCLUSIVE, WITH MEAN TEMPERATURE OF THE MONTHS OF JULY AND AUGUST.

Years.	Mean Tem- perature of July.	Mean Tem- perature of August.	Deaths from Sunstroke.	Years.	Mean Tem- perature of July.	Mean Tem- perature of August.	Deaths from Sunstroke.
1855, . .	78	74	14	1863, . .	74	75.5	133
1856, . .	77	71	17	1864, . .	73	76	87
1857, . .	75	72	14	1865, . .	78	71.5	11
1858, . .	74	72	14	1866, . .	80	71	314
1859, . .	74	72	5	1867, . .	74	73.5	7
1860, . .	75	72.5	12	Total, . .			1471
1861, . .	74	73	15				
1862, . .	71	72	44				

TABLE SHOWING DEATHS BY SUNSTROKE AND THE EFFECTS OF HEAT IN THE METROPOLITAN HEALTH DISTRICT OF NEW YORK, FROM JUNE 20TH TO AUGUST 8TH, 1868, AND THE RANGE OF THERMOMETER AND BAROMETER FOR EACH DAY.

DATE.	Number of Cases.	BAROMETER.		THERMOMETER.	
		Minimum.	Maximum.	Minimum.	Maximum.
June 20,	3	29.70	29.71	82	92
" 21,	—	29.75	29.85	70	78
" 22,	—	29.90	29.91	60	66
" 23,	—	29.95	30.01	61	80
" 24,	—	30.05	30.15	60	77
" 25,	—	30.15	30.21	60	74
" 26,	—	30.01	30.19	64	80
" 27,	—	29.85	29.95	70	86
" 28,	2	29.90	29.95	74	86
" 29,	—	30.07	30.14	70	84
" 30,	—	30.20	30.21	70	83
July 1,	—	30.20	30.21	71	84
" 2,	1	30.20	30.21	71	88
" 3,	2	30.14	30.20	72	94
" 4,	3	30.04	30.15	78	96
" 5,	7	30.01	30.07	80	101
" 6,	1	30.10	30.11	70	80
" 7,	1	29.91	30.11	67	80
" 8,	1	29.93	29.95	73	87
" 9,	—	29.95	30.01	70	86
" 10,	—	30.03	30.07	70	84
" 11,	2	30.01	30.11	78	95
" 12,	—	30.00	30.01	80	102
" 13,	32	30.04	30.08	86	103
" 14,	37	30.01	30.06	80	96
" 15,	41	29.85	30.01	80	100
" 16,	41	29.85	29.96	79	95
" 17,	8	29.98	30.00	73	87
" 18,	5	29.98	30.01	75	90
" 19,	1	29.91	30.01	76	93
" 20,	4	29.90	30.00	66	80
" 21,	1	29.95	30.03	65	80
" 22,	1	29.85	29.91	76	90

DATE.	Number of Cases.	BAROMETER.		THERMOMETER.	
		Minimum.	Maximum.	Minimum.	Maximum.
July 23,	—	29.90	29.90	70	82
" 24,	—	29.75	29.86	72	84
" 25,	1	29.65	29.81	72	88
" 26,	1	30.01	30.05	70	86
" 27,	1	30.05	30.06	73	84
" 28,	1	30.05	30.10	68	81
" 29,	—	30.10	30.11	70	84
" 30,	2	30.05	30.11	74	90
" 31,	—	29.91	30.03	75	93
Aug. 1,	4	29.70	29.85	80	94
" 2,	—	29.65	29.73	80	92
" 3,	—	29.80	29.91	79	94
" 4,	3	29.95	30.05	70	85
" 5,	—	30.13	30.24	70	82
" 6,	1	30.20	30.21	65	80
" 7,	—	30.01	30.10	65	82
" 8,	—	29.75	29.91	74	80

Months.	Mean Pressure of Atmosphere.	Mean Temperature.	Average Humidity.
June, .	30.02	68.00	69.80
July, .	30.00	81.02	68.00

Total number of deaths by sunstroke and the effects of heat, . . . . . 206]

Total number of deaths by sunstroke and the effects of heat, . . . . . 208]

**Treatment.**—Keeping in view the nature of this disease, and the various modes in which death may approach, the line of treatment may be indicated as follows, on the authority of Dr. Barclay:

With regard to the class of cases in which death tends to occur suddenly from *syncope*, there is little opportunity afforded for treatment; but the measures indicated are—the *cold douche*, keeping the surface wet and exposed to a current of air, or assiduously fanned, exclusion of light as far as possible, the immediate employment of stimulants, external and internal, by the rectum as well as by the mouth. *Depletory measures of any kind are not to be thought of.* In the less rapidly decisive cases prompt treatment is of the greatest use; while delay is fraught with the greatest danger. The patient must be immediately stripped of his outer clothing; and, being placed in a semi-recumbent position, the cold douche is to be applied, from a height of three or four feet, over his head and along his spine and chest, his extremities being at the same time sponged over with cold water. Relaxation of the pupil is the first symptom that shows the good effect of the treatment, which may require to be repeated several times, on account of returning insensibility; but if there is any evidence of failure of the pulse, this treatment must be discontinued, and the application of cold to the head is then all that can be borne. The hair is to be cut short as soon as possible, and a blister applied to the nape of the neck, the surface having previously been well sponged over with the *acetum lyttæ*. When the first violence of the attack is subdued, increasing confidence in the ultimate result may be indulged in so soon as vesication takes place; and in cases where insensibility recurs after an interval of ten or twelve hours, it may be removed by the application of a second blister to the vertex; and which may be again repeated, there being no doubt as to the good effect it produces. A blister may also sometimes be applied along the spine in the worst cases. Stimulation by the use of the electro-galvanic current, with the moist sponges applied along the sides of the neck, chest, and epigastrium, ought also to be employed. *Sinapisms* ought generally to be applied to the extremities, and to the chest or sides.

As soon as possible after the employment of the douche, a strong purgative enema ought to be given, those of a stimulant nature being preferred (such as those indicated at page 327). But as the enema may have to be repeated several times before any effect is produced on the bowels, it may be advisable to let the first enema be of a simple purgative character; and afterwards let it be followed up by turpentine enemata.

If cerebral congestion is indicated by the state of the eyes already described, a few leeches to the temples may relieve the congestion (LONGMORE, BARCLAY); but the prevailing opinion among officers of experience in the treatment of this disease is against the employment of bloodletting by venesection, even in severe cases. In all the cases which have been recorded in which it has been employed, it seems to have been generally hurtful, and to have hastened the fatal termination.

In cases where the breathing is much oppressed, and the bronchial

tubes loaded with mucus, the patient should be turned occasionally over on his face and side.

In the convulsive form of the disease, where the greatest irritability of the nervous system prevails, the douche is found to be inadmissible, from the agony which it occasions; and in such cases Dr. Barclay has found great benefit from the inhalation of chloroform. After a few inspirations the convulsions for the most part ceased, and sleep was very easily induced; but in one or two instances, after a considerable interval of consciousness, febrile symptoms increased in severity, coma supervened (probably effusion having occurred within the cranium), and was followed by death. But the cases in which chloroform can be used are comparatively few; and very great care is necessary in its employment, so that the inhalation may be suspended at once, as soon as any effect is produced upon the pulse. [The internal administration of chloroform has been recommended, and used with alleged success, by Dr. A. P. Merrill, of New York, and others.]

[In the first form of sunstroke, that in which the affected person is "only overcome by the heat," from its exhausting action upon the nervous system, rest in the horizontal position, and the free use of stimulants, best administered by the rectum, and moderate cold affusion to the head, carefully watching that it does not increase the symptoms of prostration, are all that is necessary. The tendency to death, in such cases, is by cardiac syncope, and when not immediately fatal, they will generally recover under this treatment.

In the second form, where, it would seem, the blood is, from the outset, seriously compromised, with an increased body-temperature as a constant symptom, the proper principle of management is to obtain a rapid and considerable reduction of the blood-heat. The best manner of doing this must depend upon the general condition of the subject, the state of the circulation, and the thermometric indications. Either tepid body-baths with cold affusion to the head and back of neck, or general cold affusions, or cold whole-baths, may be used, to reduce excessive temperature. Dr. Levick (*Penn. Hosp. Rep.*, 1868, *Am. Jour. Med. Sciences*, October, 1866), recommends rubbing the whole body over with pieces of ice, as large as can be conveniently handled, and keeping pieces in the axilla, until there is returning consciousness, which may not be for several hours; after which iced wine and water may be given. Of 7 cases of heatstroke treated in this way, in the Pennsylvania Hospital, during the summer of 1866, 6 got well, while of 12 cases treated with stimulants by the mouth and rectum, cold affusions and full bath, 7 died. The ice treatment was suggested and first carried out with happy results by Dr. B. Darrach (*Am. Jour. Med. Sciences*, January, 1859).

The sequelæ of sunstroke—persisting headache, fixed or shifting, pain in the back, choreic trouble, especially of the forearm and hand, convulsive disorder, mental weakness—require rest, attention to the condition of the skin, and change of climate. Dr. McLean recommends, when the pain is fixed and severe, long-continued counter-irritation of the nape, and a course of iodide of potassium. He says: At Netley during the invaliding season we are never without such cases, and very obstinate and intractable some of them are. I have seen some men discharged quite unrelieved by treatment (*l. c.*, p. 165).]



**Measures for the Prevention of Sunstroke.**—(1.) All weak and sickly men should be weeded out and left behind, if a march is to be undertaken during hot weather in India; (2) The costume should be suitable for the early morning hours before sunrise, as well as for the scorching period which follows, when the men are for the most part in their tents. It should consist of materials of slow conducting power, of a color by which heat is not readily absorbed, and should be as loose and light as possible. A flannel shirt should be worn, to prevent the men being exposed to sudden chills, and a flannel belt round the loins may be worn with advantage, except in the hottest weather. The shirt-collar should either be open, or made so wide as to prevent all risk of its pressing injuriously on the veins of the neck. Above all, some other form of knapsack should be devised than that at present in use. Competent authorities, strongly urged by Dr. Parkes, have devised a pack which is a great improvement on that now in use, and which does away with the use of cross belts over the chest, so injurious to the functions of the organs within the chest. The march ought not to be commenced too early in the morning. The troops should be on the *new* ground about an hour after sunrise, and the pace should never exceed three and a half miles an hour. There should be a halt for seven or eight minutes every hour, or oftener if the men are exhausted, and a longer halt half way, when each man should have a cup of coffee and a biscuit. They ought also to have their ration before starting in the morning. An ample supply of water should be provided for the men by "*bheesties*" being attached to each company, and always compelled to march with it. No man should be allowed to fall out without being accompanied by a non-commissioned officer, whose duty it should be to bring him to a medical officer at once, if sick, and if not, to bring him up to the column at the halt. All men so falling out should be brought up to hospital tents for examination immediately on the arrival of the regiment in camp. The men should carry nothing on the march but their rifles and ammunition,—the quantity of the latter being kept as small as is consistent with safety. They should be allowed to march "easy" and loosely clad, more particularly in passing through jungles or ravines. No halt should ever take place on such ground when better may be had within a moderate distance. When the sun is up, halts should be so timed that shelter may be obtained by open *topes* of trees. Camps should be formed on as high and open ground as possible. The sites for camps in India, marked out by the "official" pillars, are generally the most objectionable that could have been selected (BARCLAY, INDIAN SANITARY COMMISSIONERS, and others). As much space should be allowed between the tents as the ground will admit of. Tents should be pitched as speedily as possible, camels and elephants being provided for their transport,—carts never. "*Kus-kus*" mats should be kept constantly wet.

Troops in the field during the continuance of the hot winds in India should have the best description of tents that can be got for hospital purposes, and be provided with the best known means for keeping them cool. An abundant supply of water in camp is of the

utmost importance, care being taken that the bags containing it are in good repair, hung up within easy reach of the men, and kept always filled with water. Sentries should be under cover during the heat of the day; and the men should be instructed, whenever they go out in the extreme heat of the sun, to put a wetted towel or thick handkerchief over the head, under the cap, and around the back of the neck and face. The men should be encouraged to take exercise during the cool of the evening; or, at all events, to leave their tents, so as to permit of their thorough ventilation; and wherever it is practicable, bathing should not be omitted.

Rations of spirits ought certainly to be discontinued in India. It may be a question as to what should be substituted instead; but all authorities are agreed that the system of "spirit rations" tends to convert young soldiers into drunkards.

The sleeplessness which sometimes is a premonitory symptom of the disease has been relieved by opium, *provided there were no evidence of commencing insensibility*, and followed by a purge, if necessary (M. WRENCH, late 12th R. Lancers).

## INSANITY.

**Definition.**—*A mental state in which acts of Conception, Judgment, or Reasoning persistently express themselves as different from the states of Feeling and modes of Thought usual to the individual in health (COMBE). There is then a deficient, impaired, or perverted power of the Will, or an uncontrolled violence of the Emotions, with perverted Sensations and Instincts. Such complex mental states are separately or conjointly produced by disease of the brain, ordinarily of insidious approach, generally without fever, and resulting from specific morbid action of the hemispherical ganglia, and tending to become chronic or persistent. Such conditions render the patient legally an irresponsible being, and unfit him eventually for the performance of the social and political duties of life (ESQUIROL, WINSLOW, BUCKNILL, TUKE).*

**Pathology and Morbid Anatomy.**—Many theories have been propounded to explain the nature of Insanity. They may be resolved into two, as at present entertained, namely,—(1.) The *metaphysical, functional, or spiritual* theory; and (2.) The *cerebral* theory.

The "functional" or "spiritual" theory, which inculcates the belief that Insanity is an affection of the immaterial principle, is at variance with all reasoning. Such a belief is in direct opposition to positive, well-recognized, undeniable data. It is an almost universal belief that the brain is the material instrument by which that thinking principle, the Mind, manifests itself, whether it be by the unseen phenomena of *Conception, Judgment, Reasoning, and Instinct*, or by the more obviously expressed phenomena of *Volition, Emotion, and Sensation*. To consider, then, those *subjective* phenomena which collectively, in their various manifestations, constitute "Mind"—an immaterial essence—as liable to disease apart from all derangement of the material organ, the instrument with which it is so closely and indissolubly united—is to believe in a most incongruous,

unphilosophical, unphysiological doctrine. The more consistent theory is that which is known as the *cerebral theory*; and which is now entertained by most of those eminent physicians who have made Insanity a special study and subject of treatment. Among the most able exponents of this theory in our country may be mentioned Drs. Forbes Winslow, Bushnan, Conolly, John Charles Bucknill, Daniel H. Tuke, Laycock, and Lauder Lindsay; and from their lucid descriptions of the insane I have drawn up the following account of Insanity, sufficient for the scope of this text-book.

The belief which this so-called *cerebral* theory inculcates is, that the instrument through which the phenomena of Mind are expressed is the part diseased;—that the encephalic nervous textures are primarily implicated. And as it is consistent with the pathology of disease in every other organ and texture of the body, that the part may be diseased without our means and instruments of research being able as yet to demonstrate such morbid state to the senses, the diseased state being expressed through one or more disordered functions of the frame; so is it with the brain. It is an organ of such exquisite delicacy, both of structure and of function, that important and extensive structural changes may, and often do exist, which neither our naked eyesight nor our touch can appreciate, and which can only sometimes be demonstrated, if at all, by various complex methods of research.

In those cases, also, of Insanity, where the manifestations of the mental phenomena are simply disordered and perverted, but not abolished, it is consistent with the known pathology of disease in other parts to expect *the very slightest* structural change, such as may rather be expressed as a *tendency* to those morphological lesions which occur between the blood and the elements of texture, and which are only manifested through vital phenomena—insidious, because unseen in their approach—and often inappreciable after death to the most experienced observers. Analogous to this morbid state in the nerve-tissue of the brain is the local morbid state which attends that complex morbid process known as *inflammation*, and to that essential part of it to which Virchow gives the name of *parenchymatous*. It is expressed in the altered vital morphological phenomena between the blood and the minute elements of tissue, and which, in some textures, has been appreciated by the microscope as a mere “cloudy swelling” of the minute elements of tissue, such as may be seen in cartilage and in some of the so-called non-vascular tissues.

The phenomena of Insanity also offer the most conclusive data showing that the brain-tissue is impaired, especially in those cases where the manifestations of Insanity obviously yield to remedies.

Those pathological doctrines are, moreover, always to be regarded with distrust whose tendency is to hold out no hope of cure. Such is the tendency of the *metaphysical* theory of insanity.

Believing in the *cerebral doctrine*, and acting upon it, the prospects of cure are hopeful, *if the case is diagnosed early, and remedies are judiciously applied.*

"If cases of Insanity are brought within the sphere of medical treatment in the earlier stages, or even within a few months of the attack, Insanity, unless the result of severe injury to the head, or connected with a peculiar conformation of chest and cranium, and an hereditary diathesis, is *as easily curable as any other form of bodily disease for the treatment of which we apply the resources of our art*" (WINSLOW).

It is a serious error, on the one hand, to act upon the belief that *physic cannot make a man think otherwise*, when "one man thinks himself a king, another a cobbler, and another that he can govern the world with his little finger." On the other hand, it is equally erroneous to act upon the belief, "*that no man was ever reasoned into Insanity or reasoned out of it.*" It is only by a proper combination of *medical and moral treatment* that the first manifestations of Insanity are to be controlled and ultimately effaced.

"The existence of so vast an amount of incurable Insanity within the wards of our national and private asylums is a fact pregnant with important truths. In the history of these unhappy persons—these lost and ruined minds—we read recorded the sad, melancholy, and lamentable results of either a total neglect of all efficient curative treatment *at a period when it might have arrested the onward advance of the cerebral mischief*, and maintained Reason upon her seat, or of the use of injudicious and unjustifiable measures of treatment under mistaken notions of the nature and pathology of the disease. . . . Experience irresistibly leads to the conclusion that we have often in our power the means of curing Insanity, even after it has been of some years' duration, *if we obtain a thorough appreciation of the physical and mental aspects of the case, and perseveringly and continuously apply remedial measures for its removal*" (Winslow in *Lettsomian Lecture*, pp. 59, 61).

The testimony from morbid anatomy which illustrates the pathology of Insanity, and which more directly supports the cerebral theory, is that which has resulted from observations made upon the *bulk* of the brain, upon its *absolute* and *specific* weight, and upon the relative bulk and weight of the gray and white substance of which it is composed. The original observations of Dr. Bucknill with regard to the *insane*, and of Dr. Sankey with regard to the *sane*, furnishing especially valuable data for comparison, are those which must be regarded as the initiative of observations from which we hope yet to learn much. Dr. David Skae, of the Morningside Asylum, near Edinburgh, and Dr. Boyd, of the Somerset County Lunatic Asylum, have more recently confirmed some of these observations, already noticed at page 270, *et seq.*, and have in some measure extended them. Such observations show generally,—(1.) That the absolute weight of the brain is slightly increased in the *insane*—a conclusion which is also consistent with the fact, established especially by Bucknill, that in many cases of Insanity the absolute size of the brain is materially diminished relatively to the capacity of the cranium. (2.) This increase in absolute weight appears to depend chiefly on an increase in the weight of the *cerebellum* compared with the *pons Varolii*, the *medulla oblongata*, and the

*cerebrum*. The general result is, that the *cerebellum* in the insane is heavier in relation to the *cerebrum* than it is in the sane. (3.) Dr. Boyd records the singular fact that almost invariably the weight of the left cerebral hemisphere exceeds that of the right by at least the eighth of an ounce (Royal Society, Feb. 28, 1861; also, *Med.-Chir. Trans.*, vol. xxxix). (4.) On arranging the weight of the brain according to the form of Insanity under which the patients labored, the following average results were obtained:

AVERAGE WEIGHT OF THE ENCEPHALON.

In <i>Mania</i> , . . . . .	54 ounces	11½ drachms.
In <i>Monomania</i> , . . . . .	51 "	11¼ "
In <i>Dementia</i> , . . . . .	50 "	5¼ "
In <i>General Paralysis</i> , . . . . .	49 "	12¼ "

The absolute weight, therefore, is greatest in *mania*, and least in the *general paralysis of the insane*; while also the *cerebellum* decreases similarly in weight through the same series, with the exception, that in the *general paralysis* of the insane it presents the highest average. Generally it appears that in cases of *acute mania* (which is a form of Insanity generally of comparatively short duration) there is the smallest amount of increase in the relative weight of the *cerebellum*; while in *general paralysis*—a disease of more prolonged duration—the greatest increase takes place. (5.) The results of observations in the specific gravity both of the gray and white matters of the brain show an increase in the *insane* compared with the same textures in the *sane* (SANKEY, SKAE). (6.) It is consistent also with the records of these observers that the *mode of death* has an influence upon the specific gravity; and, generally, it may be stated that when cerebral symptoms are well marked, such as by convulsions, strabismus, and the like, and when the case terminates by *coma* or by *apnœa*, the specific gravity is higher than when the symptoms are associated with exhausting disease, such as phthisis, and when the case terminates fatally by *anæmia* or *asthenia*. The high specific gravity of the gray and white matter in the former class of cases averaged 1.041, while the average specific gravity of the whole brain in similar cases, as observed by Dr. Bucknill, varied from 1.040 to 1.052; the average specific gravity of the whole healthy brain being only 1.036. (7.) Dr. Bucknill's more recent observations show the most essential change to consist in shrinking of the substance of the brain, with degeneration of the nerve-cells, or a relative atrophy of its substance by a deposit of inert matter (*Med.-Chir. Review*, Jan., 1855). (8.) On comparing the specific gravity of the gray substance in the different forms of mental disease, the lowest appears to occur in cases of *dementia*, where, however, it is 0.003 above the average in the *sane*. The next highest specific gravity occurs in cases of *melancholia*, the next in *general paralysis*, the next in *mania*, and the highest in *epilepsy*. In some of these, however, and probably in all, the *mode of death* appears to influence the specific gravity. For instance, in the *paralysis of the insane* terminating by *coma* or *apnœa*, the specific gravity of the whole brain



has been 1.040; while in similar cases terminating by *syncope* or *asthenia*, the specific gravity of the whole brain has not exceeded the average, nor gone beyond 1.039. The average specific gravity of the white substance is lowest in cases of *mania*, next in *dementia*, higher in *general paralysis*, higher still in *monomania*, and highest in *epilepsy* (*Edin. Monthly Jour. of Med. Science*, October, 1854).

The general results of the more crude examinations of the cranium and its contents in cases of Insanity, in this and other countries, show that, in a very large proportion of cases, there are found some degree of thickening and opacity of the arachnoid, serous effusions into the subarachnoid tissue, into the arachnoid sac and ventricles of the brain, or of serum with lymph more or less gelatiniform, accompanied sometimes with increased, sometimes with diminished vascularity of the brain and its membranes, thickening and adhesion of *dura mater*, gritty state of the *pia mater*, turgescence of cerebral vessels, or of *puncta vasculosa*. Lesions are also more frequently found at the anterior and superior portions than at the base of the brain. Some cases prove fatal by a series of apoplectic seizures when the bloodvessels are extremely atheromatous. In that specific form of paralysis supposed to be peculiar to the insane there also appears to exist a peculiar softening of the gray matter, not indicated by any change appreciable to the eye, but by layers of the gray matter stripping off easily with the membranes, to which it often adheres; by the readiness with which the gray matter is broken up by a stream of water; and by changes in the contents of the brain-cells, as observed microscopically. The morbid state of the brain in the insane may extend to a considerable depth into the gray matter composing the hemispherical ganglia; but the whole of these ganglia are generally more or less implicated, in conjunction with the tubular fibres passing from the hemispheres through the vesicular neurine. Dr. Lindsay has noticed œdematous softening of the central parts of the brain in certain of the cases he has examined. Such changes are generally associated with great vital and nervous depression. In a valuable paper "On the Blood in the Insane," Dr. Lindsay has shown that although the blood is in some cases characterized by particular morbid states, such states are nevertheless not peculiar to insanity. He is of opinion that, save in extreme and exceptional cases, there are no specific abnormalities of the *urine*, *blood*, *post-mortem appearances*, *craniological measurements*, or of *facial characteristics* in the *insane* as compared with the *sane*. With regard to the urine, the same industrious observer remarks, that his results differ widely from those published by Dr. Sutherland and other eminent authorities, especially as regards the view concerning the elimination of *phosphates*, and the connection of such elimination with Insanity (*Journal of Psychological Medicine*, July, 1856, p. 488). The general result of his experience is that there is virtually no special or distinctive Pathology of Insanity.

It is necessary to bear in view that crude morbid appearances occurring in the Insane are also found more or less frequently in the brains of persons who have died of other diseases, and without any manifestations of mental impairment of the nature of insanity.

**Causes.**—In modern times Insanity is a disease unhappily of frequent occurrence, and it has been supposed to be extending in proportion to the degree of civilization. It does certainly appear, from an interesting paper by Dr. Winslow, in *The Journal of Psychological Medicine* for July, 1857, that diseases of the brain and nervous system are not only of more frequent occurrence, but that a certain unfavorable type of cerebral disorganization tends to develop itself in the present day. This type of cerebral disorder may be characterized as being—(1.) Insidious in its approach; (2.) Tending to that form of Insanity which frequently terminates by suicide; or (3.) Leads at an early age to softening of the brain, at an age—the prime of life—when the Intellect ought to be in an active and vigorous condition of exercise and of health.

The remote causes of Insanity are of a Moral or Physical nature. Of the patients principally admitted into the different hospitals of France, Italy, and Belgium, about *one-tenth* have their insane state attributed to falls, blows on the head, the abuse of mercury, of other Physical causes not determined. The remainder of the cases have their insane condition ascribed to Moral influences—as religion, having been crossed in love, jealousy, family disputes and family cares, reverses of fortune, wounded pride, disappointed ambition, anger, fright, excess of study, libertinage, and drunkenness. It is sometimes difficult, if it is always desirable, to separate Physical from Moral causes in our speculations on the Etiology of Insanity. The action of Moral influences in producing insanity is so striking that the passing events of the day often impress upon the disease its peculiar characteristic. When magic and witchcraft were believed in, Europe was overrun with persons who supposed themselves possessed by the devil. When the Pope was at Paris, that singular event caused many religious monomaniacs,—a form of Insanity, says Esquirol, which shortly after disappeared.

The causes of Insanity are of a nature producing in the patient, in the first instance, *emotional* changes only, either by the sudden and violent agitation of the Passions, or by the long-continued influence of circumstances operating more insidiously upon the Mind, and producing an habitual state of abnormal feeling. There is no description of Insanity which, if traced to its source, may not be found either to consist in perverted Emotion, or to emanate from that origin (Bucknill in *Med.-Chir. Review* for January, 1854). Dr. Hood's statistics give the proportions as follows: *Moral causes*, such as anxiety, grief, uncontrolled Emotions, in 40.8 per cent. of the males, and 32.7 per cent. among females. *Physical causes*, in 19.8 per cent. of males, and 21 per cent. of females. The chief of these Physical causes are *old age*, abuse of alcoholic liquors, bodily illness, and the critical period in females.

The principal predisposing causes are *age*, *sex*, *hereditary descent*, and *disease*.

**Age.**—Infancy is nearly exempted from madness, and so also is childhood, except in congenital cases. Esquirol, however, gives the case of a child between five and ten years old, whose *mono-*

*mania* lay in attempting to destroy both her father and mother. Insanity, however, as a general principle, seldom breaks out till after puberty, when the Passions are fully developed. Dr. Thurnam's table shows that the greatest liability to the disease exists at the ages between twenty and thirty; while the statistics of Hanwell make the period from thirty to forty to be the most liable to Insanity.

*Sex.*—It has been much disputed which sex is the more liable to insanity. Esquirol, from returns obtained from the different insane establishments of London and Paris, considered the numbers to be nearly equal; but Dr. Thurnam's tables show that males are more liable than females to attacks of Insanity, in the proportion of 53 per cent. of males to 46 per cent. of females. An approximation as to the influence of social position on the patients shows a larger proportion of Insanity among the unmarried than among the married population, in proportion to their respective numbers.

*Hereditary Origin.*—The testimony of almost universal experience establishes the fact of a very general hereditary transmission of Insanity, varying from 26 to 69 per cent. of the cases. This is remarkably instanced among the high nobility of France and other countries, who almost in every instance intermarry, and are allied by blood to each other, inculcating a sad lesson to those parents who consult, in the marriage of their children, their present interest rather than the health of their descendants. This hereditary tendency to Insanity in the aristocracy is greatly insisted on by Esquirol, who states that only one-third of pauper female lunatics were ascertained to belong to families in which Insanity had previously existed; while more than one-half of the female lunatics of the higher classes were thus connected. In general, children born before the Insanity of their parents, are less liable to this disease than those born after the attack; also, children born of parents diseased in one line are less liable to it than parents diseased in both lines of descent. The condition of the mother during gestation has often a striking effect on the mental health of her future offspring. Esquirol observed that during the French Revolution many pregnant ladies, whose minds were kept constantly in a state of alarm and anxiety during that period, brought forth children who, in their infancy, were subject to convulsive or other nervous diseases; and in their youth either to *mania* or *dementia*. The form of Insanity chiefly transmitted appears to be *dementia*; the mother's Insanity being chiefly transmitted to daughters, and that of the fathers to sons. Legislative enactments regarding the intermarriage of persons tainted by Insanity are greatly to be desired; and certainly the concealment of Insanity, with a view to marriage, ought to render marriages null and void which are concluded under such circumstances.

*Disease.*—Certain diseases, also, are powerful predisposing, or even exciting causes of Insanity, such as *epilepsy*, which gives rise to a large number of the most incurable cases. Derangement of

function or structure of the uterus is another powerful predisposing cause; and many persons become deranged after severe fevers or attacks of "*sunstroke*," in climates like that of India. Dyspepsia is also a frequent forerunner of Insanity.

**Symptoms and Forms of Insanity.**—No unobjectionable classification of the forms of Insanity can be propounded; and, as Dr. Daniel H. Tuke justly states, any classification must be regarded merely as a chart by which we may shape our course, having only the prominent points marked or partially delineated upon it. So far, also, as cause can be ascertained, the same cause does not always produce the same result; for in different individuals it will occasionally produce very opposite forms of Insanity. Dr. Lindsay justly observes, as a result of his prolonged experience, that "a very large proportion—perhaps a majority—of hospital cases of Insanity cannot be referred *simpliciter* to the heading of any *nosology*." Changes of type in the disease are also not uncommon; such that a woman, for example, previously suffering from *melancholia* may become *erotomaniac* or *maniacal*. *Dementia* is a frequent sequel of all the other forms of Insanity. Such changes of type are of constant occurrence; and it is by no means unusual to find the same case of Insanity, at different stages of its progress, presenting the characters successively of *mania*, *monomania*, *melancholia*, and *dementia*. The differences, therefore, between *forms* of Insanity cannot be regarded as *essentially* very great.

The following is the classification (a modification of Heinroth's) suggested by Dr. D. H. Tuke in his and Dr. Bucknill's *Manual of Psychological Medicine*, first edition, p. 89:\*

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\* I am indebted to Dr. Lindsay for the following synopsis:

"The following synopsis of the dominant symptoms, principal phases, or general conditions of mental aberration, may be considered sufficient for the purposes of the student, as being at once comprehensive and general, accurate (so far as it goes) and practical. It makes, however, no pretence to rigid philosophical or scientific exactitude, or to exhaustive completeness—an exactitude and completeness which its author believes to be unattainable. But it may claim to be more simple and less technical, and hence more *likely to be easily remembered*, than the majority of the many and varied classifications or Nosologies of Insanity that have been offered during the last half century:

I. Insanity may be—

- a. *General* [Examples, Mania, Dementia]; or
- b. *Partial*. [Example, Monomania].

It may—apparently, though perhaps not *really*—affect

- a. All the faculties or powers of the mind [Examples, Mania, Dementia];
- b. Or any *one* or *two* of their groups, viz.:
  - 1. Intellect proper [Example, Delusional Insanity];
  - 2. Moral sentiments [Example, Moral Insanity];
  - 3. Propensities or passion [Examples, Dipsomania, Kleptomania, Erotomania].

II. It may be—

- a. *Congenital* [Example, Idiocy]; or
- b. *Acquired* [subsequent to birth and generally to puberty].

## CLASSIFICATION OF DISORDERS OF THE MIND, INVOLVING—

CLASS I.—*The Intellect.*

	FORMS OR PHASES OF INSANITY.
<i>Order 1.</i> Development incomplete.	{ IDIOCY. IMBECILITY. DEMENTIA. MONOMANIA (Intellectual). INCLUDING Delusions. Illusions. Hallucinations.
<i>Order 2.</i> Invasion of Disease after development.	

## III. Its characteristic may be—

- a. Excitement* [Example, Acute Mania];
- b. Depression* [Example, Melancholia];
- c. Neither*—No departure from normal character or conduct [Examples, Some cases of Delusional Insanity and Moral].

IV. It may or may not be accompanied by *Delusions*, which may be either—

- a. Partial and uniform* comparatively [Example, Monomania]; or
- b. Numerous and variable* [Examples, Some cases of Delusional Insanity].

V. The following *Propensities* or tendencies may be exhibited—any one or combination of them :

- a. Destructiveness*—
  - 1. *To life*—Suicide, homicide;
  - 2. *To property*—Clothing, furniture, glass, &c. [Example, Pyromania];
- b. Alcoholic intoxication* [Example, Dipsomania];
- c. Theft* [Example, Kleptomania];
- d. Sexual aberration* [Example, Erotomania];
- e. Abstinence from food.*

## VI. It may be—

- a. Simple* ; or
- b. Complicated* [with other diseases].

## The chief complications are—

- 1. *Epilepsy* ;
- 2. *Paralysis*, which may be—
  - a. Partial* ; or
  - b. General* [Example, General Paralysis or Paresis] ;
- 3. *Various*—
  - a. Functional* ; or
  - b. Organic* affections of the—
    - 1. *Lungs* [Example, Phthisis] ;
    - 2. *Stomach* and intestines [Example, Dyspepsia] ;
    - 3. *Uterus*, &c.

## VII. Each of the main forms of Insanity—

- { *a. Mania,*
  - { *b. Melancholia,*
  - { *c. Dementia,*
  - { *d. Amentia*—
- may be developed in *different degrees of intensity* [Example, Dementia is represented by mere facility of character—by silliness, imbecility, or weak-mindedness of every grade, onwards to utter fatuity].



CLASSIFICATION OF DISORDERS OF THE MIND—*continued.*CLASS II.—*The Moral Sentiments.*

FORMS OF INSANITY.	
Order 1. Development incomplete.	MORAL IDIOCY (?).
	MORAL IMBECILITY.
Order 2. Invasion of Disease after development.	MORAL INSANITY.
	MELANCHOLIA—
	1. Religious.
	2. Hypochondriacal.
	3. Nostalgic.
	EXALTATION, regarding
	1. Religion.
	2. Pride,
	3. Vanity.
	4. Ambition.

CLASS III.—*The Propensities.*

Order 1. General.	MANIA (usually a disorder of all the faculties).
	HOMICIDAL MANIA.
Order 2. Partial.	SUICIDAL MANIA.
	KLEPTOMANIA.
	EROTOMANIA.
	PYROMANIA.
	DIPSOMANIA.

**1. Idiocy.**—*An abortive condition of the intellect from defective development of the brain. Subjective phenomena are not capable of being exhibited, and therefore cannot be inquired into. The condition is congenital* (ESQUIROL, COKE, PRICHARD, BUCKNILL, TUKE).

The characters of the idiot, as portrayed by Dr. Tuke, vary according to the degree in which the cerebro-spinal system is involved. In the lower or more abject forms of idiocy the functions of *organic* or *vegetative* life are ill performed; nutrition is most imperfect; the power of reproduction is small; and the patient would perish were it not for the assistance of others. The functions of animal life are likewise impaired, and he may be scarcely alive to external impressions, or possess the power of executing spontaneous acts. In the most abject state of idiocy he is blind, deaf, and dumb, while the excretions are passed involuntarily (*op. cit.*, p. 95). From this abject state there are grades of Intellectual and Moral capacity among idiots; and generally it may be stated that the greater the organic deformities the more marked will be the imperfections of Sensibility and Intelligence, and the smallest heads appertain to the most degraded class of idiots; but a small head is not a necessary accompaniment of the condition. On the contrary, many idiots have large heads. The abject condition of the idiot is obvious in his vacant stare, in the thick everted lips, the slavering mouth, the irregular teeth, the gums often swollen, the frequent strabismus, the general want of symmetry, the absence or defect of the senses of sight, hearing, and speech, taste, and smell. Notwithstanding the

staggering gait, he is constantly moving, if on his feet; and, if seated, he has a difficulty in balancing himself (Tuke, *loc. cit.*).

2. **Cretinism.**—This condition, which is endemic in many countries, and is occasionally sporadic, has been already noticed under the subject of "Goitre" (vol. i, p. 791). Although it seems that some cases are congenital, yet Dr. Tuke, from personal observation, and from the Report of the "Sardinian Commission," believes (*op. cit.*, p. 104) that there is no pathognomonic sign by which cretinism can be recognized at birth; but that a certain combination of symptoms permits the prognosis in childhood of the future development of cretinism. After the fifth or sixth month he describes the infant cretin as presenting the following symptoms: "The development of the body proceeds very slowly; the child, though weak, is remarkably stout, and appears swollen; the color of the skin is somewhat dusky, sometimes yellow, sometimes natural; the head is large, the *fontanelles* widely separated, and sometimes all the sutures disjointed. The expression is stupid; the appetite is voracious; and much time is passed in sleep. The belly is swollen; the extremities are generally attenuated; the neck is thick, but not always goitrous; teething is not completed for many years, and is generally accompanied by an offensive salivation, and frequently by convulsions. Usually the child cannot stand before its sixth or seventh year; and it is then that it begins to articulate certain sounds, supposing it has not been deaf from birth. The voice is hoarse and shrill, and words are spoken with difficulty." His stature is diminutive. His head is of great size, but flattened at the top, and spread out laterally; and the dissections of Virchow show that the parts at the base of the cranium are early ossified in the form of a rectangle. Early union of the bones takes place, and with such early union arrest of growth occurs at that part of the skull, while various compensatory developments continue in other parts. Hence the *prognathous* face, and the sinking of the root of the nasal bone. Irregular and partial union of the sutures at an early age is a frequent morbid condition of the Insane, associated with an atrophic condition of the *gyri* below the site of union. The oblique downward direction of the orbit in *cretins* is brought about by the compensating growth of the skull generally, and more especially of the *malar*, the *frontal*, the *temporal* bones, and *zygomatic* arches, in consequence of the deficient development of the *sphenoid* bone. The stunted development of the bones at the base of the skull gives a very short distance between the front and middle part of the cranium; while the diminished growth of the nasal septum and of the jawbones gives a *prognathous* form of face alike to the cretin, the negro, and the monkey. Thus the character of the face in the cretin, as Dr. Tuke observes (*op. cit.*, pp. 105–6), remains unchanged from puberty to old age. The eyes are generally affected with strabismus; the zygomatic arch is large, and the mouth of large size; the lips thick, and the lower one hanging down. The lower jaw is small, retreating, and its angle very obtuse. The predisposition to cretinism appears to be hereditary; and, as noticed by Dr. Watson, it has a close but ill-understood connection with goitre, so that, with

few exceptions, cretins are goitrous; and he also observes, that “when both parents are goitrous for two generations in succession, the offspring being in the third generation, are sure to be cretins” (Lect. xliii, vol. i, p. 788). Fodère makes an observation to the same effect.

3. **Imbecility** is synonymous with—

4. **Dementia** is an impaired mental condition, “*a disorder of the intellect characterized by loss or feebleness of the mental faculties.*” The condition is arrived at in several ways, capable of being traced in the mental history of the patient. Some have previously afforded examples of *melancholia*, when they were perfectly conscious of all that passed around them. Some have been *maniacal*, others have suffered from the severe delirium of fever or sunstroke; but after a partial recovery from these immediate affections, by slow gradations the mental faculties become dulled, confused, and finally obliterated. Others, again, lose their faculties by reason of extreme old age,—*senile dementia*,—“the last infirmity of noble minds” (Tuke, *op. cit.*, p. 113). All such cases are remarkable by their conversation and acts, which greatly resemble infancy; and the mental alienation is indicated by the disorder of ideas, affections, and determinations. Feebleness is the essential characteristic of this form of mental disorder; and there is abolition, more or less marked, of all the sensitive, intellectual, and voluntary faculties. On the slightest excitement, some Dements are liable to maniacal outbursts. “If dementia is long continued, its outward signs become well pronounced in the face of the patient. The vacant and puzzled look, the lack-lustre eye, the weak smile, the meaningless laugh, betray the vacuity of Mind” (Tuke, *op. cit.*, p. 115).

Its varieties are *acute and chronic dementia*. Dements may become paralytic, and a thickness of speech may be the first symptom of its approach. After a time the speech is more manifestly affected, followed by a loss of power in the limbs of one side, more marked in the lower extremity, so that the step is feeble and straggling. In the last stage they are completely paralytic, and only able to utter a few unintelligible sounds,—*Paralysis of the Insane*—syn., *General Paralysis*.

5. **Monomania** is a term which comprehends various forms or phases of *delusional* Insanity. There is generally an undue intensity and exaltation of the Conceptive and Perceptive Faculties. The Insanity is more or less partial, in such a way that some one Passion or idea so entirely possesses the patient as to lead often to dangerous conduct. The modes by which the monomaniac gives expression to his particular *delusion* are endless; and the mental affliction is especially indicated by *delusions*. A fixed idea overcomes the Will, and the brain, from its diseased condition, fails to correct the delusions under which the patient labors, by the normal exercise of the Faculties. Reason is unable to dissipate them; the exercise of observation fails to discredit them; and *delusions*, more or less persistent, are the result.

It is hardly possible to understand the nature of this phase of Insanity without being well aware that every sense is liable to ex-

press, by the mode in which it performs its function, the existence of cerebral disease; as when light things feel heavy, small things seem large, hot things feel cold; or when the senses are liable, from the irritation of the brain or other cause, to become morbidly active, the patient seeing persons or hearing discourses when no such person is present, and no such discourse is related.

Much objection has been taken to the term "*delusion*;" yet the "symptoms of delusion are still accepted in our courts of justice as the most authentic mark of insanity, and as the essence of cerebro-mental disease." The term may be thus defined, from the gist of the writings of Drs. Bucknill and Tuke: *A delusion is a belief in the existence of things which have no existence in reality, or an erroneous perception of the nature of things, or of their relation to each other, occasioned by cerebro-mental disease*; or again (as Dr. Bucknill defines it), *An intellectual error caused by the pathological condition of the mind, and displaying itself in false Sensation, Perception, or Conception* (*op. cit.*, 317). A few instances of such morbid sensations, perceptions, or conceptions, are the following:

Dr. Falconer mentions a case in which cold bodies felt intensely hot to the patient, who could not move without believing he was burnt. Esquirol mentions a lady who, being recommended a lavement, was desirous of administering it herself. No sooner, however, was the syringe put in her hands than she threw it away with an expression of horror, stating that it felt so heavy that she believed it to be filled with mercury, and that they wanted to make a barometer of her body. A gentleman, whose Mind was in every other respect perfect, had constantly the sensation of his mouth being full of pieces of broken glass: while another, curious in his table and choice in his wines, believed everything tasted of oatmeal porridge. The sight is often the medium of morbid perception. Dr. O'Connor met with a patient recovering from measles, to whom every object appeared diminished to the smallest possible size. Baron Larrey mentions a person who saw men as big as giants; and another patient, on recovering from typhus fever, who felt himself to be ten feet high, his bed eight feet from the floor, and the opening of the chimney as large as the arch of a bridge. The celebrated Pascal always believed he saw a precipice on his left hand, and had a chair placed on that side to prevent his falling into it. The ear, also, is likewise often affected. It hears "the airy tongues that syllable men's names." A gentleman riding by a barracks at evening-call never got the sound of the bugle out of his ears for nine months; and everybody knows that Dr. Johnson always entertained a deep impression that, while opening the door of his college chambers, he heard the voice of his mother, then many miles distant, calling him by his name, "Sam! Sam!"

It is remarkable, also, that *hallucinations* sometimes occur when the organ is itself destroyed through which they would be *objectively* expressed, thus showing their *subjective* nature. Esquirol, for example, attended an insane merchant, who, though laboring under *gutta serena*, not only heard persons talking to him, but saw visions that perfectly enchanted him. He had also under his care a Jewess

who was blind, and yet saw things the most strange. She died, and the optic nerve, from its commissure to its entrance into the sclerotic of the eyeball, was found atrophied, so that the transmission of any objective impression was impossible. He mentions, also, two other women absolutely deaf, who had no other delusion than that of hearing every night invisible persons addressing them. Such are instances of *hallucinations*, and the images thus excited are described to be as vivid as those produced by objective causes; so that the patient, when insane, entirely believes the empty and false forms he sees, the ideal sounds he hears, to be real and substantial. Nothing can persuade him of the non-reality of any one of them. Like Macbeth, he insists, "If I stand here, I saw him." It is only by the occurrence of a temporary *hallucination* that we can explain the apparition of the ghost of Cæsar to Brutus, promising to meet him at Philippi; or of the familiar spirit which conversed with Tasso; or of the demon of Socrates, and such like.

In Insanity, if a part of the body be diseased, the Imagination often personifies the disease into some strange reality. There are constantly patients in hospitals for the insane, who, suffering pains in the crown of the head, believe they are caused by worms gnawing the brain; or, suffering pains in the stomach, believe that organ to be filled with serpents, frogs, or mice. A woman, for many years a patient at Salpêtrière, who suffered severely from abdominal pains, believed she had a whole regiment of soldiers in her abdomen, and when the pains were severe, that they fought with each other. Another woman, called by the patients "Mère de l'Eglise," believed she had in her entrails all the personages of the New Testament, and occasionally those of the whole Bible. At other times she believed the Popes held their councils in her abdomen. She died, and the abdominal viscera were found adherent to each other and to the peritoneum.

Individual instances of monomania might be classified under *chronic mania* or *melancholia*, according to their character.

6. **Moral Idiocy**, or congenital defect of the Moral sense, while the Intellectual Powers are not deficient, but sometimes superior, is a morbid condition not easily established. There are cases, however, which now and then, throughout the periods of childhood, boyhood, and youth, exhibit all the evidence one can have of an inert Moral nature, associated with an activity of the animal propensities. Over such a mental condition, threats, rewards, or punishments exercise little or no control. There is said to have been at one time in the Richmond Lunatic Asylum, in Dublin, a man who exhibited a total want of Moral feeling and principle, but who possessed considerable intelligence, ingenuity, and plausibility. Dr. Crawford wrote of him that he had never been different; that he had never evinced the slightest mental incoherence on any one point, nor any kind of delusion. He appeared, however, so totally callous with regard to every Moral principle and feeling; so thoroughly unconscious of ever having done anything wrong; so completely destitute of all sense of shame or remorse, when reproved for vices or crimes; and he proved himself so utterly incorrigible throughout life, that



Dr. Crawford expresses himself as almost certain, that any jury before whom he might be brought would satisfy their doubts by returning him insane. The "exact counterpart" of this case was admitted into the New York State Asylum (Benedict's *Annual Report*, 1850). Dr. Tuke gives many other instances besides these (*op. cit.*, p. 181); but it is still very doubtful if *Moral Idiocy* can be recognized as apart from impaired Intellect; although feeble Moral Powers and Volition, or dangerous peculiarities of temper, may be occasionally associated with good intellectual abilities.

7. **Moral Insanity as Developed in Adult Life.**—A standard of *mental health* is to be sought for in the natural and habitual character of the patient; who being thus compared with himself, it will be seen that a change in his feelings and conduct sets in gradually. Moral Insanity has been more or less clearly defined by Drs. Prichard, Tuke, and Bucknill. Dr. Tuke observes that the adult about to become insane in this respect is "more absorbed or reserved, and on any provocation, however slight, is unreasonably irritated. He becomes suspicious, liable to attribute false motives to his friends or to others, and to cast ungenerous reflections upon his nearest relatives." He is observed to be morose. The alteration of the man from his former nature is complete; and some act of an outrageous character is at last committed. In other cases, an individual has been subjected to over-exertion of mind, his powers overtaken, or his feelings put upon the stretch in consequence of anxiety or unaccustomed responsibility. He then finds himself susceptible to the slightest Emotion. Sleep and rest are lost; he is conscious of more or less uneasiness about the head,—a sense of tension, and dull aching pain; and at last he is unequal to the discharge of his usual duties. Certain impulses and tendencies begin to distress his Mind, because he knows they are alike repugnant to his Reason and to the dictates of his Moral nature. Often the impulse is to do violence to himself or others; or it is simply to break glass or articles of furniture (*op. cit.*, pp. 185, 186). The *Moral Insanity* thus expresses itself simply as a tendency to disordered emotional excitement, which affects the course of Thought and action, without destroying the reasoning process in any other way than by supplying wrong materials to it. There may be no disorder of the Intellectual Powers, or any delusion whatever (CARPENTER). *Reason* does not reign supreme; it is simply retained possession of while the *Feelings and Emotions* are beyond control. Circumstances in life create feelings and prejudices which prevent the morally insane patient from passing through life smoothly. As regards the *Intellect*, the patient is not insane; but he cannot control his Feelings or Emotions.

Among patients morally insane, physical health, as Dr. Tuke observes, is almost as frequently deranged as it is among those whose Intellect is manifestly disordered; and the cases not unfrequently terminate in some unmistakable physical disease, such as general paralysis. In a large number of cases the patient has been, in childhood, or is, subject to epilepsy; and there can be no doubt that convulsions in infancy are, in relation to their ultimate effects on the Mind, not sufficiently recognized. They often pass away

and are forgotten, but some portion of the cerebral tissue has doubtless received an injury which, in any other tissue of the body, would be easily recovered from, but which permanently injures the delicate tissue of the brain of the child, so that his Moral or Intellectual Powers are impaired; and the result is painfully perceptible as the child grows up to manhood (*op. cit.*, p. 187).

The diagnosis is to be made between this form of Insanity and mere vicious propensities; and the only ground of diagnosis, as pointed out by Dr. Bucknill, lies in the mode of causation. "Moral insanity is always preceded by an efficient cause of mental disease, and there has always been a notable change in the emotions and the propensities following" (*l. c.*, pp. 328, 329).

8. **Melancholia.**—It is a "*disorder of the intellect with depression, often with suicidal tendency.*" A state of melancholy is often the first indication of Mental disease. It may precede *mania*, and it is associated with or supervenes upon other forms of Insanity. It may supervene suddenly, as when it is the immediate consequence of grief; or it may come on gradually, as the mere exaggeration of a naturally melancholy frame of mind. Sudden melancholia is rare. Premonitory symptoms generally indicate a period of incubation more or less prolonged and sufficiently obvious. A state of depression often follows upon a state of *mental elation*, or on prolonged mental exertion and occupation, which suddenly ceases. It also succeeds the mental exaltation produced by inebriating drinks. These effects, however, are generally slight and transient, and the cases of this kind ought to be separated from those cases in which the depression becomes persistent. In *Persistent melancholia* the "relish for existence" becomes less and less, the spirits become depressed, and the man feels unequal to the ordinary duties which call him into public life. In the domestic circle he becomes silent, and seeks entire solitude (*TUKE, op. cit.*, p. 148).

"He makes his heart a prey to black despair;  
He eats not, drinks not, sleeps not, has no use  
Of anything but thought; or, if he talks,  
'Tis to himself." (*DRYDEN*).

And even although he is "cheerless, moody, and taciturn," as Dr. Tuke observes, "he appears to be unceasingly revolving in his mind his unfortunate condition, which, while he regards it as worse than that of any other person, he stills believes to be good enough for a miserable wretch like himself." His Propensities are to indolence and general indifference. He reads nothing, writes to nobody, shuns all exertion. One dominant Propensity alone is too often active, namely, self-destruction (*CONOLLY*); while obstinate abstinence from food and drink is a common feature. There is greatly increased susceptibility of the Emotions; and the subjects of *melancholia*, in its early stage, are easily moved to tears by trivial circumstances. All consolation is disregarded by such patients. They are beyond persuasion. Nothing can subdue their Will, convince them of their error, quiet their alarms, or allay their fears. Nothing can remove their prejudices, overcome their repugnances,

or conciliate their aversions. Nothing can divert them from the engrossing thoughts that occupy their mental energies, and take possession of their Emotions. Occasional remissions of the affection may deceive the patient's friends for a time; but the disease progresses till the patient is either placed under the care of competent guardians, or he voluntarily seeks the tranquillity of an asylum. But, fortunately for the chances of cure, a love for and appreciation of the ridiculous is often associated with the tendency to *melancholia*. Cowper, who penned "John Gilpin," is a familiar example. Carlini, a French comic actor of reputation, consulted a physician to whom he was unknown, on account of attacks of profound *melancholia* to which he was subject. Amongst other remedies, the doctor recommended the diversion of the Italian Comedy. "Your distemper," said he, "must be rooted indeed if the acting of the lively Carlini does not remove it." "Alas!" said the miserable patient, "I am the very Carlini whom you recommend me to see; and while I am capable of filling Paris with mirth and laughter, I am myself the dejected victim of melancholy and chagrin" (Prefect's *Annals of Insanity*, p. 404, quoted by Dr. Tuke, *op. cit.*, p. 150).

The ultimate course of cases of *melancholia* varies with the constitution of the patient, his age, the degree in which the disorder is complicated, and the existence of hereditary predisposition to mental disease. It tends to pass into *dementia*; but the tendency thereto is much less marked than in *mania*. The prognosis is more favorable in simple *melancholia* than when complicated with disorder of the Intellect.

*Among the earliest mental phenomena are forgetfulness, abstraction, simple depression of spirits, alterations of the Affections towards children or other near and dear relations, restlessness, religious dreads, delusions, alterations of the Instincts, such as hunger.*

*"Among the earliest physical symptoms of melancholia are loss of sleep and disturbed dreams. The digestive organs are frequently deranged, the tongue is unnaturally red or loaded, there is fulness at the epigastrium, and the alvine evacuations are deficient in bile. The tongue is sometimes flabby, pale, indented at the edges; a fixed dull pain, or an ill-defined sense of oppression, is often experienced in the head. The pulse is generally slow and compressible. The urine is often pale, sometimes high-colored, depositing lithates. The skin varies; it is usually harsh, but not unfrequently it is moist and clammy. The uterine functions are more or less disordered, and in a large majority of cases are suspended. In men the reproductive instinct is in abeyance"* (Tuke, *op. cit.*, pp. 152, 153).

Constipation, long-continued and habitual, associated with solitude and family cares in persons formerly healthy, and of excitable and lively dispositions, appear in some cases to have acted as an exciting cause of *melancholia* (Dr. W. H. O. Sankey, *Med. Times and Gazette*, September 19, 1863).

*Melancholia* comes next in frequency of occurrence to *mania*. It is frequently hereditary; and all its varieties are disposed to be remittent. "It is generally observed that the remission takes place in the latter part of the day, the patient suffering most acutely in

the early morning, and for some hours subsequently." In those cases which have a suicidal tendency this remittent character should be borne in mind (Tuke, *op. cit.*, p. 171).

The principal forms which *melancholia* assumes are—(1.) *Religious*; (2.) *Hypochondriacal*; (3.) *Nostalgic*.

In the *first* form of melancholy the impress is given to its character by the religious tendencies of the patient, the exciting cause being not rarely traceable to the fiery denunciations of well-meaning but injudicious preachers. There are some preachers whose great power in the pulpit is to be attributed to the excitement and alarm they are able to produce upon susceptible listeners. They delight to suspend such persons over the bottomless pit, in order to drag them up again when they fancy they have sufficiently impressed them with most unchristian terror. Such preachers were wont to be banished in Pagan times by a law of Marcus Aurelius.

In the *second* form, that of *hypochondriasis*, the morbid mental state is expressed by the *exaggeration or increase, to a morbid degree of intensity*, of that property which every one possesses more or less, by virtue of his physiological and psychological endowments of creating around him, or within himself, sensations which are not the result of external impressions or corporeal condition; but which, having their origin in the Mind (subjective), are represented and appreciated by the material organs of the body. It consists essentially in the transference of a phenomenon (subjective or mental in its origin and essence) into what appears to be a real material change, appreciable sometimes by others (REYNOLDS). It is often expressed by the sense of touch, combined with a morbid imagining, so that the patient believes himself to be strongly metamorphosed, changed into some inanimate thing, or he loses all knowledge of his personal identity; and this form of disease is sometimes combined with other *delusions*. The odd conceptions of the patients under these circumstances are singular enough. Men have imagined themselves to be so much butter or putty, and in the one case to be unable to bear heat for fear of melting, and in the other have forbore to walk, lest their legs should be crushed by the weight of their body. One man keeps the house imagining he is too large to pass through a given doorway; and when he is pushed through he screams, and will affirm that his flesh is being torn from his bones. Another imagines he is a pump; that his arm, which is in perpetual motion, is the handle; and bitterly complains that the inhabitants will let him have no rest, morning, noon, or night. A third goes round to his neighbors, believing that he is a seven-shilling piece; and hopes, if his wife should bring him to their shops, they will neither take him in payment nor give change for him. A fourth supposes himself transformed into a beer-barrel rolled along the streets. A fifth, that he is a mutton-chop, and insists that he shall be taken daily to the butcher to be trimmed. Bishop Warburton speaks of a person who thought himself a goose-pie, a circumstance referred to by Pope in his sketch of hypochondriasis,—

“A pipkin there, like Homer’s tripod, walks;  
Here sighs a jar, and there a goose-pie talks.”



Among other singular forms of *hypochondriacal Insanity* is a belief in an absolute change of sex. Dr. Arnold saw a man who fancied himself in the "family way;" and Esquirol speaks of a male patient who fancied himself a woman, and felt insulted if the slightest liberty was taken with his dress. Some have thought themselves converted, like Nebuchadnezzar, into wild beasts. Some patients imagine they have no soul, others no body, others that they are absolutely dead. One gentleman, approaching his ninetieth year, so far lost his mind that he assembled his family around him, and announced to them that he was dead; begged, in communicating the sad intelligence to his absent friends, they would say he went off easily, and expressed himself a little scandalized that the windows were not closed on the occasion, and entreated, as a last favor, for one pinch more out of his favorite snuff-box before he was finally screwed down. A soldier, who received a severe wound at the battle of Austerlitz, believed he had died, and that the body he had now got was not his own. Another, that he was guillotined during the French Revolution, and had not only lost his own head, but, somehow or other, had got a new one. A third, that his head had been put on his shoulders with the face towards his back; and, lastly, some believe they have not only lost their heads, but can see them rolling on the ground.

It is seldom, however, that *hypochondriasis* is of so simple and harmless a nature. More commonly the Affections are subverted, and those who ought to be most dear to the patient by the ties of relationship become most hateful. The Mind, too, is more commonly swayed by some destructive Passion to effect some object criminal in itself. *Delusions* are neither necessary nor essential symptoms. *Hypochondriasis* is often one of the worst concomitants or sequelæ of dyspepsia (WATSON).

*Third*, The *nostalgic* form of *melancholia* (sometimes expressed by the term *nostalgia*) makes itself obvious by an inordinate desire to return to one's native country when far away from home, and to which is added the apprehension, on the part of the patient, that he may never be able to return. The prophecy of the inspired writer seems ever ringing in his ears, when he wrote—"Weep ye not for the dead, neither bemoan him; but weep sore for him who goeth away: for he shall return no more, nor see his native country" (Jeremiah, chap. xxii, ver. 10). Army surgeons often witness such cases of home-sickness. Ninety-seven soldiers in the French army fell a sacrifice to this disease between 1820-26; and the celebrated Baron Larrey came to the conclusion that the mental faculties in *nostalgic* patients were the first to undergo a change. Decided aberration of Mind was present in all the cases which he has recorded, expressed by exaltation of Imagination, especially in extravagant delusions respecting their homes. This mental excitement was accompanied by increased heat of the head and acceleration of the pulse. There was redness of the conjunctivæ, and unusual movements of the patient were frequently observed. Uncertain pains occurred in various parts of the body. The bowels were constipated. There was a general feeling of oppression and



weariness; an inability to fix the attention; and conversation was apt to be unconnected. A sense of weight and pain pervaded the viscera. There was also sometimes partial paralysis of the stomach and diaphragm, and symptoms of *gastritis* or *gastro-enteritis* would supervene. Under these circumstances prostration of strength ultimately becomes extreme, mental depression keeps pace with the decline of the body, the patient lies weeping, sighing, or groaning, and a propensity to suicide is not unfrequent when the debility becomes extreme. General paralysis is common; but death is the result of a gradual exhaustion of the vital powers (Tuke, *op. cit.*, p. 156).

The Dutch, the Swiss troops, the Highlanders, and the Irish, are those soldiers amongst whom this form of insanity has been mostly noticed, and the disorder is apt to be prevalent during extreme height of the barometer.

9. **Mania**, having its origin in disordered Emotions, is essentially a disorder of the Impulses or Propensities in the first instance, tending to more or less "*disorder of the Intellect with excitement.*" One or more of the passions is almost always exalted in *mania*, of which there are two forms—namely, *acute mania* and *chronic mania*; and furious expressions of passion, of prolonged duration, are very generally present in the acute form of this disease. It has in almost all instances its stages of incubation. At first there may be only apparently trifling irregularities in the Affections. The maniac may be at the outset of his disease either sad or gay, active or indolent, indifferent or eager, but he soon becomes impatient and irritable (ESQUIROL). He neglects his family, forsakes his business and household affairs, deserts his home, and yields himself to acts which strikingly contrast with his ordinary mode of life. Delirium and Reason begin to alternate with each other. Periods of composure and agitation succeed each other, and so do acts the most strange and extravagant. The kindest love and tenderness of domestic life serve but to irritate and provoke, so that to remain in the bosom of his family excites the patient by slow degrees to the highest pitch of fury.

It is seldom in *mania* that the patient, as in *monomania*, is only insane on one subject. His mind, says Esquirol, is a perfect chaos; all is violence, effort, perturbation, and disorder. He confounds time and space, associates persons and things the most unnatural, creates images the most unreal, and lives isolated in feelings and reasoning from all the rest of the world. He hates all whom he was wont to love, and wishes to overthrow and to destroy everything. The female maniac, perhaps in health the model of candor and virtue, gentle and modest, an affectionate daughter, a devoted wife, and a good mother, becomes in this disease bold and furious, exposes her person unmoved to the gaze of every eye, is blasphemous and obscene, respects no law either of decency or humanity, and threatens her father, strikes her husband, or perhaps murders her children.

In another class of cases the premonitory symptoms are characterized by gloom and despondency, upon which the maniacal ex-

citement supervenes. There is generally a marked departure from the patient's former state of health. *Insomnia* is one of the most important and earliest symptoms. The functions of the body are more or less deranged, and fever may prevail, sometimes severe.

Special forms of mania may be noticed under the following heads:

(a.) **Homicidal Mania** in some cases is the result apparently of delusions, of suspicion, or of implacable enmities against supposed foes. A plausible reason is generally assigned for the attempt in such cases; the victim is represented as having systematically annoyed, or irritated, or conspired against the lunatic. In other cases the attacks are the offspring of momentary, uncontrollable impulse, without cause *quoad* the persons assaulted. The pretext for assault is then frivolous in the extreme—*e. g.*, "he could not help it," or he did it "for fun." Such homicidal impulse and attempts of the most persistent and dangerous kind may coexist with a perfect knowledge of right and wrong, and their bearings on human actions—with perfect ability also to manage business affairs, though of a complex pecuniary character—with perfect propriety in maintaining most of the relationships, or of discharging most of the social or public duties of life—with deportment often the most polished and gentlemanly, the most considerate and kind (Dr. Lindsay, *Rep.* xxxvii, p. 42). Gall gives the case of a man at Vienna who, after witnessing a public execution, was seized with an uncontrollable propensity to kill, although he had a clear consciousness of his situation, expressed the greatest aversion to commit such a crime, shook his head, wrung his hands, and cried out to his friends to keep away. Pinel mentions the case of a person who exhibited no other unsoundness of mind than this propensity to murder; so that his wife, notwithstanding his tenderness for her, was nearly being destroyed, he having only time to warn her to flee. In the intervals of the paroxysm he expressed the greatest remorse, was disgusted with life, and attempted several times to put an end to his own existence. Esquirol mentions a woman seized with sudden paroxysmal impulses to destroy her children, and only saved them by locking the bed-room door and throwing the key away. Metzger relates a similar case of a nurse who requested to be discharged, giving as a reason that every time she undressed the child, struck by the whiteness of its skin, she had an irresistible desire to rip open its belly. The deadly purpose is accomplished in many different manners and times. Sometimes the murder is long premeditated and the victim marked out, the patient concealing a knife about his person till an opportunity for effecting his object presents itself, though that period be remote. In other cases the destructive propensity seems the result of a sudden impulse. Esquirol mentions the case of a patient who was sitting by the fire with other patients, when he suddenly seized a chamber-pot and broke it over his neighbor's head: fortunately, he was immediately secured. In a lucid interval he stated that he had made this homicidal attempt in consequence of his brother having appeared to him at that moment, crying out, "Kill him! kill him!" Others, again, are so aware of the approach of the attack that they entreat to be con-

fined, in order that they may not commit the mischief to which they seem irresistibly impelled.

(b.) In **Suicidal Mania** there is an irresistible propensity on the part of the patients to destroy themselves. Some of these unfortunate persons, not having resolution to put themselves to death, have killed others, in order to suffer a judicial death. One woman reasoned, "In order that I may die I must kill some one," and accordingly she attempted to kill both her mother and her children. Some of these tragedies are most terrific. A man in a paroxysm of Insanity is related to have killed his wife and three children, and would have killed the fourth had it not escaped. After these horrible sacrifices he ripped open his own belly; but the wound not being mortal, he again drew out the instrument, and pierced himself through and through. This man had enjoyed a good education, and was of a mild disposition. In a large number of the cases the suicidal propensity is developed in connection with *religious melancholia*—a form most difficult to eradicate or conquer; and from its inveteracy, the forerunner often of incurable Insanity.

The propensity to commit suicide is in some persons so great that many destroy themselves although in possession of fortune, of station, of objects of affection, and apparently in every other respect in the fullest enjoyment of happiness. The ingenuity of the patient in providing means for his own destruction is often singular. Some have thrown themselves under the wheels of a wagon; and recently it has not been uncommon for them to cast themselves before the locomotive of a railway train in full speed; others have drowned themselves in an incredibly small quantity of water; others have most ingeniously strangled themselves; and others, more closely watched, have swallowed all sorts of heterogeneous articles—pins, needles, bits of broken glass, nails, buckles—in short, any and every hard substance they could force down their throats. Pinel gives the case of a man who had cut off one of his hands with a hatchet before his arrival at Bicêtre, and afterwards, in spite of his bonds, attempted to tear the flesh off his thigh with his teeth.

(c.) **Pyromania**.—The derangements of the Emotions and of Reason may take other forms than murder; and arson is one of the more common. Some seem impelled to this criminal act by the mere sensual gratification of the excitement, confusion, noise, and bustle consequent on the conflagration; delight in the blaze, the ringing of the bells, and in the thronging of the people. It may also result from a process of reasoning, or from acting upon some delusion, as in the destruction of York Cathedral by Martin, effected under a feeling of Divine impulsion, and of his being commissioned thus to purify the house of the Lord.

(d.) **Kleptomania** is an irresistible desire to steal. Gall mentions that the first King of Sweden was always stealing trifles; and a countess at Frankfort had the same propensity. Esquirol gives the case of a lady of an exactly opposite character. Her Insanity consisted in a ceaseless dread of appropriating what did not belong to her; she therefore combed her hair an endless number of times in the day, examined her dress minutely every time she put it on or

took it off, felt in her shoes, turned up the chairs, looked under her plate, and thus consumed many hours in the day in endless cares lest something of value might have adhered to her dress.

Such are some of the forms or phases of a malady whose varieties are endless. It may be objected by some to the account here given of Insanity, that whereas book descriptions of the disease generally refer to what are regarded as typical cases, which are selected, exceptional, and extreme, yet in the great bulk of the insane the phenomena or symptoms of the malady may be either so complicated and intermixed, or so obscure and ill-marked, that it is not only impossible but futile to attempt a scientific enumeration or classification of all.

**Diagnosis.**—There are two especially weighty reasons why it is important to obtain a correct diagnosis—namely, *first*, with reference to the necessity of an early application of remedies; and *second*, with reference to the question as to whether or not the person presumed to be insane is or is not legally responsible for his acts.

Dr. Winslow has shown with what inexcusable neglect affections of the brain are generally treated by the public, and the lamentable amount of ignorance that unhappily exists in the non-professional mind respecting these disorders,—a neglect and ignorance which, by sins of omission, often suffer the sacrifice of valuable human life to occur. The overwrought brain meets with but little attention and consideration when in a state of incipient disorder. While medical advice and remedies are eagerly sought for trivial organic or functional disorder in other parts of the body, serious well-marked symptoms of brain disorder are often entirely overlooked and neglected. Such symptoms are not unfrequently permitted to exist for months without causing the faintest shadow of uneasiness or apprehension in the mind of the patient or his friends. These premonitory indications of cerebral mischief, or *prodromata*, as they are technically called, consist of *morbid alterations of temper, depression of spirits, amounting sometimes to melancholia; headache, severe giddiness, inaptitude for business, loss of Memory, confusion of Mind, defective power of mental concentration, the feeling of brain lassitude and fatigue, excessive ennui, a longing for death, a want of interest in pursuits that formerly were a source of gratification and pleasure, restlessness by day and sleeplessness by night.* Any one or more of these symptoms obviously indicate an unhealthy state of the functions of the brain and nervous system; but their insidious mode of approach, and the unwillingness of friends to believe that anything is wrong with their relative, rarely if ever permit the symptoms to attract attention till some phase of insanity becomes unmistakably developed. If a person previously in a state of bodily and mental health is conscious that abnormal changes are taking place in his Mind—that trifles worry and irritate him, that he feels his brain unfit for work, that his spirits flag, that he tends to magnify all the evils of life; if, moreover, he is observed to be fanciful, if he imagines things to exist which have no existence apart from himself, if he believes that kind friends ill-use and slight him; if, besides, symptoms like these, or analogous to these, are associated with headache, derange-



ment of the digestive organs, want of continuous sleep, the friends of such a sufferer may rest assured, and the patient may perhaps be convinced, that the state of his brain is abnormal, and he may be induced to commit his case to the careful consideration of a Physician. Symptoms of severe bodily fatigue, associated with extreme depression of spirits, mental exhaustion, reverie, paroxysms of melancholy, partial somnambulism, or hallucinations manifesting themselves at an early period of life, must be regarded as important psychological phenomena—deviations from the state of health, requiring the most careful and cautious Moral and Intellectual training, combined with medical and hygienic treatment; more especially to be persevered in if despondency become more marked, or if gloomy thoughts and apprehensions of an early death lay hold of the Mind. Of the numerous and seemingly increasing cases of suicide which occupy a place in our daily newspapers, in most of them well-marked symptoms of physical ill-health, disorder of the brain and nervous system, may be traced to exist before the act of self-destruction. In upwards of a hundred recent cases Dr. Winslow has shown this connection to have existed. These cases are full of interest, and demand the most attentive study by the student who would make himself acquainted with the earliest symptoms and most distressing results of Insanity. They are recorded in *The Psychological Journal* for July, 1857, already referred to.

To obtain a correct and early diagnosis, with a view to attain either or both of these objects, one "*only safe rule*" is to be observed; for in the existing state of legal and medical science there is no uniform test of Insanity, either of a legal or of a medical kind, which can be safely or certainly applied. This rule consists in a close and thorough appreciation of the physical and mental aspects of the existing condition of the presumed lunatic, at the period of his supposed Insanity, compared with his prior physical and mental manifestations, which were regarded as his natural and healthy state, and which had not been observed to be different from those of other men—"a comparison of the individual with his former self."

This point was originally insisted on by Dr. Combe; and the necessity of making the Mind of the individual patient, and not that of the Physician, the standard of comparison by which to determine the sanity or insanity of the patient, cannot be too strongly urged. The man must be the measure of himself; and this principle is found to be of universal application in all physiological and pathological investigations. For example, before the Physician can judge of the condition of the urine passed by a man in disease, he must know the conditions of that man's urinary excretions when they are in a normal state. Scarcely two men are alike in this or in any other respect regarding their excreta,—age, weight, height, and many other circumstances, materially modifying the result.

In judging, therefore, of all cases of presumed Insanity, the Intellect must be considered in relation to itself,—the manifestations of Mind *now* must be compared with those which have been *hereto-*



fore expressed by the same individual; and if mental phenomena are ascertained to exist of a morbid kind, compared with those which have been expressed before, and especially if there be any symptom of cerebral disease, the individual may be fairly deemed insane, and, if so, legally irresponsible for his acts. Between the criminal and the insane mind there are important relations, and it can be shown—as there is reason to believe valuable evidence and strong testimony exist to prove—that a large amount of crime is connected by hereditary predisposition and descent with minds diseased. A large field of usefulness is here opened to the Politician, the Lawgiver, and the Physician, from which future generations can alone hope to reap the benefits—when crime and lunacy may, perhaps, diminish together.

The following rules, compiled from Drs. Bucknill and Tuke's admirable work on *Psychological Medicine*, ought to be adhered to in diagnosis: (1.) *Learn as thoroughly as possible the antecedents and history of the patient.* One of the great difficulties to overcome in the diagnosis of Insanity, especially in the endeavor to ascertain the antecedents of an attack, is the great risk the Physician runs in being misled by the interested statements of friends and relatives. They too frequently act on the principle that "what they wish to be, that they believe." They may wish their relative to be considered sane, or the contrary; and they may believe him to be so when he is not, or the reverse; and they will, at first, invariably disguise or deny circumstances which might be thought discreditable to the presumed patient or themselves. The Physician will, therefore, often find himself surrounded by relatives of the patient from whom he can derive little information which is unbiassed and trustworthy. He will find the household divided against itself; and seeing that such is the case, his best policy is to become a good and patient listener; and if he has a good memory, and is quick in perception, he will arrive at conclusions the more readily that he avoids all cross-examinations. With prudence and caution he ought then to seek out people who have known the patient, but who are neither friends nor neighbors, whose evidence will often be more truthful and useful to him. One great aim of such inquiry ought to be to ascertain the existence or not of hereditary predisposition, and of previous attacks of Insanity. (2.) *Estimate the value of the hereditary tendency, upon the following principles:* (a.) The Insanity of one parent indicates a less degree of predisposition than that of a parent and an uncle; and still less than that of a parent and a grandparent, or of two parents; (b.) The Insanity of a parent and a grandparent, with an uncle or aunt in the same line, may be held to indicate even stronger predisposition than the Insanity of both parents; (c.) The Insanity of a parent occurring after the birth of a child, without predisposition, is of no value in the formation of an hereditary tendency; (d.) If several brothers or sisters, older and younger than the patient, have become insane, the fact tells strongly in favor of predisposition, although neither parent nor grandparent may have been so. (e.) The insanity of cousins cannot yet be determined as

worth anything in favor of predisposition, except in corroboration of other and weightier facts (BUCKNILL, *op. cit.*, p. 272). (3.) *Ascertain if there has been any change of habits or disposition.* The Physician will thus learn what kind of a man the patient has been when in health. The over-susceptible rather than the eccentric man is the more likely to become insane. (4.) *Exercise the greatest tact and discretion in the personal examination of probably insane patients.* Obtain an introduction to the patient in as natural a way as possible; and above all, avoid commencing any conversation which will tend to divulge the object of the visit. In the incipient stage of disease the patient is generally suspicious and hostile, contrivance and great tact being required to open up a conversation. It may even be necessary for the Physician to be a party to some deception; but he must bear in mind that the discovery of even the slightest deception by the patient may have a most prejudicial influence on future treatment. As a general rule, it is best to engage the attention, and obtain at least the good-will of the patient by sympathizing inquiries respecting bodily ailments, or concerning such things as he knows the patient takes a lively interest in; and the first aim of the Physician must be directed to placing himself on good terms with his patient. "For this no general directions can be given. He must employ that tact, derived from good sense and knowledge of mankind, without which he will find himself lame and impotent in the field of medical practice amongst the insane. The most difficult cases are those in which differences of opinion and of interest exist among the members of the patient's family; and when the patient has quietly been told by some one of the family that it is wished to prove him insane, and to place him under confinement, and that a doctor is coming to examine him for that purpose. The Physician, under such circumstances, must then do the best he can; and if this is but indifferently well done, he may content himself with the reflection that the fault is not his" (BUCKNILL, *op. cit.*, p. 279). (5.) *Observe any peculiarities of residence or dress.* Many circumstances testify to a want of order and direction in household affairs where the head of the family is insane; in the room occupied by the patient things are liable to be out of place, especially as regards the decoration of the walls and the arrangement of the furniture. A love of order is rarely seen among the insane. (6.) *Study the appearance, demeanor, and general conduct of the patient.* (7.) *Notice any peculiarities of bodily condition.* This is necessary, especially as regards plumpness or emaciation, the state of the skin, the pulse, the tongue, and the temperature, as indicating impaired bodily health.

[1. The body-temperature in the insane is higher than natural. 2. The temperature is highest in phthisical mania, gradually falling in the following order: General paralysis, acute mania, melancholia, mania, mild dementia, and complete dementia. 3. Dementia is the only form of insanity where the average temperature is below that of health. 4. The difference between the morning and evening temperature is much less than in health, and this is owing to the rise in the evening temperature, and not to the lowering of the morning temperature as compared with

the healthy standard. 5. This rise in the evening temperature as compared with that of the morning is in the exact ratio of the death-rate among the various forms of insanity, finding its acme in general paralysis. 6. In general paralysis the average evening temperature is higher in every case than in the morning temperature. 7. The evening temperature of every form of insanity is higher than the evening temperature of health. 8. Excitement in a patient is almost always attended with a rise, as compared with a state of depression, or of repose. The difference averages  $2.2^{\circ}$  in periodic mania with long intervals, and  $1.1^{\circ}$  with short intervals. In general paralysis there may be a difference of  $5.8^{\circ}$  in the same person at various stages of the disorder. 9. The average temperature falls as the patients get older, but the fall is chiefly in the morning temperature. 10. The average frequency of the pulse in the various forms of insanity corresponds with the mean temperature; but the rise in the evening temperature has no corresponding increase in the rate of the evening pulse (CLOUSTON, *Jour. of Mental Sciences*, April, 1868.)

Dr. T. Clifford Allbutt has examined the state of the optic nerve and retina in the insane as seen by the ophthalmoscope.

Of 51 cases of *Mania* symptomatic changes were found in 25; 13 cases were noted as doubtful; and 13 were either healthy, or presented non-symptomatic lesions, as glaucoma, &c. In his paper we find the following propositions: 1. The ophthalmoscope shows symptomatic lesions in many cases of mania. 2. These are most common where other symptoms of organic disease exist, and seem not unfrequently to depend upon meningitis. 3. After a paroxysm of mania there remains a paralysis of the bloodvessels in and about the discs, causing obvious hyperæmia. 4. During the paroxysm there is, perhaps, a spasm of these vessels, as suggested by one case. 5. The permanent changes are those of stasis, of consecutive atrophy, of simple atrophy, or of a mixed character.

Of 38 cases of *Dementia* there was marked disease of the optic nerve or retina in 23; 6 were doubtful; and 9 were healthy. In simple acute dementia, however severe, if independent of organic disease, no optic changes probably take place.

Of 17 cases of *Melancholia* and of *Monomania*, tabled together for convenience, in 3 disease of the eye existed. Anæmia of the retina was, however, commonly found in melancholia.

In 43 cases of insanity dependent upon, or associated with, epilepsy, there was disease of the optic nerve or retina in 15; 9 were doubtful; and 19 showed no change. In most of the cases presenting optic changes, organic disease was known to exist from other symptoms.

In 12 cases of idiocy, there was marked atrophy of the discs in 5; 1 was changing; and 2 were noted as doubtful (*Brit. Med. Jour.*, vol. i, 1868).]

(8.) *Observe any peculiarities of gesture, and the expression of the countenance of the patient.* One of the great difficulties of diagnosis, also, is to distinguish cases of *monomania* from *sanity*. With the exception of some given *delusions*, turning on a small number of fixed ideas, the patient may be rational on all other subjects; and in some instances even the powers of his mind may be superior, and they often are so. One celebrated instance of this kind occurred to the late Lord Erskine. The patient had indicted a most affectionate brother, together with the superintendent of the asylum, for false imprisonment. He was placed in the witness-box,

and the learned Lord was occupied in the defence of the unfortunate concerned the whole day in fruitless attempts to expose it. At length Dr. Kane came into court, and suggested to the learned counsel that the patient believed himself to be the Lord and Saviour of mankind. Lord Erskine then adroitly addressed him in that character, lamenting the indecency of his ignorant examination. The patient at once expressed his forgiveness, and with the utmost gravity and emphasis, in the face of the whole court, said—“I am the Christ.” In a similar case, tried before Lord Mansfield, the patient evaded the questions of the court the whole day, till his Physician arriving, asked him what had become of the princess with whom he corresponded in cherry-juice. Instantly the man forgot himself, and said it was true he had been confined in a castle, where, for want of pen and ink, he had written his letters in cherry-juice, and thrown them into the stream below, and that the princess had received them in a boat. Such answers, of course, immediately terminated the case. (9.) *In medico-legal cases let the Physician avoid becoming a partisan.* He ought never to permit his evidence to be led by counsel on either side on matters of opinion, either directly or indirectly. “Facts observed by himself” are the elements on which alone his reputation can be safe. On all other points let him steadily refuse to give forth the expression of a mere opinion in the witness box.

The student will also find some valuable “*hints for certifying in cases of Insanity*,” by Dr. J. S. Bushman, in the *Medical Times and Gazette*, for August, 1862.

**Prognosis.** As a general rule, the younger the patient the greater are the chances of recovery; but above the age of fifty few are cured. The comparative curability of Insanity in its earlier phases is clearly proven by the records of asylums for the insane, while the advantages of early treatment, and the superiority of hospitals for the management and cure of the insane are well shown by the increasing annual admissions to these institutions. Of those that recover, the exciting cause often greatly influences the cure.

Many cases recover when the Insanity is produced by a temporary excess, provided the patient can be restored to the normal condition of mind. When the Insanity is produced by a permanent excess, recovery is rare. When the Insanity is produced by a permanent excess, recovery is rare. When the Insanity is produced by a permanent excess, recovery is rare.

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it is rare that he survives beyond a twelvemonth after the first symptom—the affection of the speech. The patient may even appear comparatively strong, but great excitement may be followed suddenly by collapse, and a fatal issue supervene sooner than inexperienced persons would anticipate.

In both sexes the recoveries average 51.5 per cent. when the cause of the Insanity is of a moral character, and 33.8 per cent. when the cause of the disease is of physical origin; and the recoveries are only at the rate of 14.6 per cent. when the only cause that can be assigned is hereditary predisposition. The influence of sex on recovery is peculiar. In males the disease terminates at a much earlier period than in females, and its *early* termination in the male is more frequently caused by death. The cases of Insanity in the male sex are not therefore to be considered as more curable, but rather as more fatal; and in the female, also, the cases cannot be said comparatively to be more curable, for although they may not die so readily as the males, the disease may continue in them, passing into a chronic and more permanent state (Hood's *Statistics of Insanity*). On the whole, however, the probability of recovery is greater among women than in men.

From the statistics of Hanwell, by Dr. Thurnam, from 1831 to 1841, the recoveries *per annum of the cases under treatment* have been  $5\frac{1}{2}$  per cent., and the average time required to effect the cure has been between four and five years. The following general result is stated by Dr. Thurnam, and is most important to be held in remembrance regarding the prognosis: namely, That in round numbers, of ten persons attacked by Insanity, five recover, and five die sooner or later during the attack. Of the five who recover, not more than two remain well during the rest of their lives; the other three sustain subsequent attacks, during which at least two of them die. All recoveries cannot, therefore, be regarded as permanent or stable; and the broad rule may be laid down, that when Insanity has once exhibited itself, there is ever afterwards a tendency to relapse; and of the insane in public asylums for the reception of all classes, about 70 per cent. may be reckoned as incurable. The intervals of recurrence or relapse are very uncertain, varying from a month to thirty or forty years.

*Intercurrent bodily diseases* in the insane are of great importance in forming a prognosis, and demand the careful study of the Physician; inasmuch as they are exceedingly insidious and exceptional in the insane as compared with the sane. In the insane the characteristics of bodily disease are masked or obscured by an inertia or torpor of the nervous system. Their febrile type is generally typhoid or asthenic. There is an absence frequently of symptomatic fever. No complaint may be made, and no external evidence of pain or suffering may be given in cases even of *acute phthisis*, where subsequent *necropsy* shows the lungs riddled with vomicæ and full of pus (vol. i, p. 64); or in cases of *phlegmonous erysipelas* going on to the formation of pus in the limbs; or in *pneumonia*, where the lungs are solidified, and normal respiration impossible; or in *organic diseases of the heart*, in *gastritis*, and in other painful diseases, such



as *cancer*, *enteritis*, or *peritonitis*. The possibility of Bright's disease should not be forgotten. Surgical operations may be submitted to without a murmur, as if feeling were completely obsolete. The most serious chest diseases may run their course without cough or expectoration; the excito-motor nervous system would seem to be nearly inert or torpid, and concentration of thought or attention in the insane is in a great measure or quite lost. Sooner or later progressive emaciation and debility, languor, lassitude, and indolence, perhaps anorexia, or sleeplessness, direct attention to the state of the patient, in whom the physical signs then show the extent of the apparently latent disease, perhaps rapidly advancing for some time before towards a fatal termination (Dr. LINDSAY). The usefulness of the thermometer in detecting such latent disease ought not to be forgotten (see vol. i, pp. 64-71). Dr. Lindsay further notices how the type or aspect of Insanity may be quite changed by fatal bodily disease. A patient, from having been passionate, fretful, and abusive, may become affable, mild, and docile. The dying patient sometimes becomes sane towards his last moments, death being preceded by a bright though transient flicker of the light of reason; and the sufferer has even expressed himself serenely, contentedly, happily, as to his latter end, and his transition from life, with all its troubles and diseases, to eternity, with all its joys and comforts (*Thirty-first Report of the Murray Royal Institution, near Perth, p. 14*). The mind may even then be soothed by the hopes and consolations of religion. "The cloud, though not wholly removed, may yet be tinged with a silver lining, and the music of the Gospel truth may help, like the harp of David, to charm the troubled spirit to repose" (Rev. W. D. Knowles, *Thirty-seventh Report, l. c., p. 297*). Such *euthanasia* for the insane is surely to be desired. That Insanity also is sometimes vicarious, or alternates with other diseases, must be remembered in prognosis. It is so sometimes with phthisis, one of the most common complications of Insanity. When the phthisis becomes acutely developed, the patient may become temporarily sane, and *vice versa*.

In the period of convalescence the return of the correct exercise of Judgment is an uncertain and fallacious indication of cure, so long as the Emotions are perverted even in a slight degree from their normal conditions; but immediately the Emotions are controllable and proper, the cure may be considered complete (BUCKNILL, *op. cit.*).

The mortality among the insane (based on the average population of a mixed county asylum, according to Dr. Thurnam) is considered decidedly unfavorable if it exceeds 9 or 10 per cent.; a mortality less than 7 per cent. is highly favorable. The largest mortality is from *dementia*, the least from *monomania*; in the latter, indeed, where there is no tendency to suicide, the duration of life is little abridged; so that premature death is almost in all cases owing to accidental and often preventable causes. Expectancy of life in the insane, in relation to Life Insurance, thus involves questions of great pecuniary importance. Popular as well as professional errors exist on the subject. The insane are separable into

classes, each of which within certain limits is characterized by a different chance or expectancy of life; but statistics on the subject are greatly required (see Dr. Lindsay's *Thirty-second Report*, p. 17).

**Treatment.**—The treatment of Insanity resolves itself into the *medical* and the *moral* treatment. Medicine indirectly acts upon the brain, as it does upon other organs, so as favorably to influence the course of the disease. It regulates the different actions and secretions of the viscera of the body, and thus improves the general health, so that the happiest results are often obtained by the early and judicious use of medicinal agents.

“Cases of severe mental despondency and distress—instances of alienation of mind associated with hallucinations, and with apparently chronic and fixed delusions, accompanied by strong suicidal and homicidal feelings—have all yielded to medical treatment; and thus persons in all grades of life, who, if those conditions had not been fully appreciated, would have fallen victims to their own insane impulses, have been restored to society in a state of mental health. The symptoms which so generally precede the act of suicide—such as depressed spirits, distress of mind, needless alarms and apprehensions as to some foreboding evil, great irritability of temper, and inability to attend to the ordinary occupations of life, excitability, headache, disturbed or sleepless nights, morbidly exaggerated views of the actual ills and circumstances of life—are in many cases certain signs of acute disorder of the brain, requiring medicinal relief, and being manifestly and rapidly benefited by prompt and energetic medical treatment” (WINSLOW).

No uniform method of treatment can be taught. Generally it may be stated that the more the symptoms approach those of *cerebritis*, *encephalitis*, or *meningitis*—in other words, in proportion as they indicate active inflammatory action—general and local blood-letting may be advisable, *but only in cases otherwise suited for such methods of cure*. Local bleeding is not to be confined to the head, for it not unfrequently happens that it may be adopted with reference to a distant viscus. Leeches to the *vulva* and thighs are beneficial in cases of *mania*, *monomania*, or *melancholia*, concurrent with the menstrual period; and to the *sphincter ani* in those cases obviously connected with suppressed hemorrhoids or hepatic congestion. In some instances leeches may be applied with benefit to the Schneiderian membrane, particularly in those cases occurring in early life, and in persons of plethoric constitution and of sanguine temperament. Illusions of hearing or of vision, which had embittered the patient's life, have been removed by leeching behind the ears or over the superciliary ridges. The utility, in *acute mania*, of prolonged hot baths, is much insisted upon by Dr. Winslow. The patients remain from eight to fifteen hours in them, at 82° to 86° Fahr., whilst a current of water at 60° is continually poured over the head. Various details of what is now known as hydro-pathic treatment have recently been introduced into English asylums, such as the Russian or vapor bath, the wet sheet, pack, &c.

*Sedatives*, or agents which modify directly the condition of the

cerebral tissue, constitute very valuable remedies. In recent acute cases they are generally admissible; but it is in the various chronic forms of *melancholia* that they are most useful. Dr. Winslow observes,—

“In suicidal Insanity, when local cerebral congestion is absent, and the general health and secretions are in good condition, the *meconate* and *hydrochlorate of morphia* often acts like a charm, if *uninterruptedly and perseveringly* given until the nervous system is completely under their influence.”

Radical cures have been effected by the occasional local abstraction of blood from the head, the administration of alteratives, the warm bath, and sedatives. Success from the use of sedatives often depends upon a *ready adaptation of the form of sedative to the description of case in which it may be deemed admissible, and a judicious admixture of various kinds of sedatives*. With respect to *opiates*, “that medicine which will allay watchfulness in one will not in another, but, on the contrary, increase it. This is particularly the case with opium, which is rarely found admissible in Insanity in its crude state. It more frequently creates heat and general febrile action than sleep” (SIR WILLIAM ELLIS). In cases, however, of recent excitement, *morphia* in considerable doses has been found most beneficial. So also will *chlorodyne* and *chloro-morphine* be of use in some cases. *Indian hemp* is an extremely useful sedative, not hitherto appreciated sufficiently, for many reasons. (See an excellent paper on its uses by Professor Russell Reynolds, in Beale’s *Archives*, vol. ii. p. 154.) It relieves pain, is *soporific, anodyne, anti-spasmodic*; and while conducing to sleep, promotes at the same time *diaphoresis* and *diuresis*, without producing headache, vertigo, constipation, or impairing the appetite. The dose varies from *one-sixth to one-half grain for a child*, and from *one-third to one grain and a half for an adult*. In cases of mental or emotional disturbance it will be found extremely useful, especially where there is deranged cerebral circulation, with pain and delirium; in cases of incipient Insanity after fever or *sunstroke*; and in cases of *senile ramollissement*. Recently *digitalis, conium, belladonna*, have been extensively employed as calmatives.

*Endermic medication* in Insanity offers numerous advantages, but, in the opinion of Drs. Winslow and Laycock, is too little practised. “In some chronic forms of Insanity—in *dementia* and persistent *monomania*, connected, as was supposed, with morbid thickening of the dura mater, and with interstitial infiltration of the membrane, as well as with exudations upon its surface—the head having been shaved, a strong ointment of the *iodide of potassium* combined with *strychnine* has been perseveringly rubbed over the scalp. In other instances the shaved head has been painted with the mixture of iodine; and both modes of treatment have been attended with benefit. When the mental symptoms are supposed to be associated with effusions of serum, *iodine* applied externally at the same time with minute doses internally of *calomel* or *mercury with*

*chalk* (so as slightly to affect the system) are recommended. This treatment, conjoined with occasional tonics, diuretics, and stimuli, to support the vital powers, is occasionally productive of considerable benefit in cases apparently placed quite beyond the reach of improvement or cure." A solution of *iodide of potassium* constantly applied to the shaven scalp has been followed by improvement in the mental state. Cases of *dementia*, the consequence of *scrofula*, are those in which endermic medication will yield the most satisfactory results (Laycock in *Med.-Chir. Review* for Jan. 1857). *Hypodermic injections*, especially of calmatives and soporifics, have also recently been employed; but their action is so irregular, and there are so many sources of danger, that their use can scarcely be commended.

*Purgatives* may be regularly required. When the bowels are constipated, the form is best determined by the state of the tongue, and sometimes by the idiosyncrasies or proclivities of the patient in regard to medicine-taking. Supposing the tongue to be white and coated, the *sulphate of magnesia*, or other neutral salt, combined with *tincture of hyoscyamus*, in the proportion of ʒj of the former to ℥ xv to ℥ xxx of the latter, in *camphor mixture*, is a formula to be recommended. If, on the contrary, the tongue be clean, the cathartic should be given with some slight bitter, as the *infusion of orange-peel* or of *gentian*. In some cases the bowels are not only exceedingly obstinate, but the patient may be greatly averse to all medicines. In such cases one or two drops of croton oil placed on the tongue or introduced in food produces free evacuations.

Mild purgative treatment formed the basis of a cure in the school of Pinel and of Esquirol; and they usually combined it, in cases of violence, with the application of cold to the head, and of warmth to the lower parts of the body, such as placing the patient in the warm bath and giving him the cold douche—a remedy since more particularly insisted upon by Dr. Brierre de Boismont, of Paris, and Dr. Winslow, of London. The further treatment consists in restoring any other functions that may be in defect or in excess, as the functions of the uterus in the female, and of the liver or heart in both sexes, by the usual remedies applicable for these purposes.

The Moral treatment is by many supposed to constitute the more efficient mode of cure in Insanity; and it must be admitted to be a most important adjunct. The first important rule is to remove the patient at once from his family: in slight cases, in order that he may be induced to exercise such command over himself as he possesses, and to remove him from influences which may have been aggravating his morbid state; and in severe cases, in order to prevent his doing mischief either to himself or others,—are the reasons for this practice. The main feature in the moral treatment of the insane in this country is the abolition and absence of mechanical coercion or restraint. The beneficial action of this system, generally known as "*the non-restraint system*," is now thoroughly recognized in England and Scotland, where it has been gradually established in every asylum since 1847–48. But there are certain exceptional phases of Insanity in which some mechanical restraint is the most

humane mode of treatment—the only mode, indeed, of avoiding certain catastrophes of too common occurrence. In certain conditions of excitement, however, it is proper to place the patient at once in a darkened room, remote from noise and the means of injury to himself or others, so that as few objects as possible may irritate him, just as a patient with his eyes affected is kept in a darkened room. The effect of such seclusion is generally of a soothing character; and in not a few cases of periodic mania it is eagerly sought by the patients themselves.

As convalescence advances the patient should be induced to undertake some manual labor, or some office in the asylum or household, which, by amusing his mind, will invigorate his body, and greatly tend to restore the healthy working of his brain. There are no more powerful medicines than “Occupation,” “Recreation,” and “Education.” Occupation should be such that no time is left for idleness, or for sitting brooding over morbid fancies. The curative results of well-chosen means of Recreation cannot be over-estimated. When the circumstances of the patient admit of it, traveling, which embraces change of air and change of scene as well as exercise, is often highly salutary in incipient cases; and much has of late been done by the judicious introduction of music and other amusements into asylums. Thus, concerts, balls, conversaziones, evening entertainments, pic-nics, excursions, *fêtes champêtres*, athletic games, pedestrian excursions, public amusements in towns, and carriage drives, are all legitimate and well-approved means of maintaining a constant and varied succession of recreation adapted for all classes of the insane. When Reason is restored, and the Affections again fix themselves on their natural objects, *and when the Emotions are under control*, the patient may be allowed to see his friends, and have his attention directed to the affairs and interests of his family; but it should be remembered that the mind remains weak and enfeebled for some time after apparent recovery; and consequently the patient’s restoration to society should be gradual.

With regard to diet, it only requires to be stated that it is often necessary to have recourse to artificial alimentation by the stomach pump in exceptional cases, when food is persistently refused.

An Hospital for the Insane, or Lunatic Asylum, is the most fit and proper place for a “person of unsound mind;” and every asylum ought to be governed by one superintendent, who should be a medical man—an officer of health to the community over which he presides. He ought to have the means of controlling all sanitary arrangements in whole and in detail,—of avoiding overcrowding,—of preventing and destroying effluvia,—and of examining the quality of the food, the water, and the drugs furnished to the establishment. Chemical and pathological appliances ought therefore to be at his disposal for his use. The number of patients who may be thus under the supervision of one medical head in an asylum ought not to exceed 200 (ESQUIROL, AMERICAN COMMISSIONERS IN LUNACY). Premature removal from asylum treatment in opposition to medical advice is greatly to be deprecated; and its baneful results are frequently to be seen, especially in the sad endings of cases of *suicidal melancholia*.



In such cases an acknowledgment should be required of the recipient of the patient, or his friends, that he is removed notwithstanding the assurance given by the medical superintendent that the patient is not recovered, and is unfit for removal (Dr. Lindsay, *Thirty-second Report*, p. 14). Such removals not unfrequently induce a change of type from acute and curable to chronic and incurable mental disease.

The subject of Insanity has been treated of in this text-book because it is a subject which medical men are expected to study, and to be called upon to treat and to deal with in a medical and also in a medico-legal point of view. They have, however, few opportunities at our schools of medicine of studying Insanity as they would any other disease in a general hospital.\* They are, therefore, often called upon to give certificates of Insanity, without perhaps having ever seen or studied a case; and many recent legal decisions have shown that it is not always safe for a medical man to incur the responsibility of signing such certificates, in the present state of the law relating to lunacy. Seeing, also, that the legal and medical views of Insanity are not in unison, nor in accordance of what is known as the correct pathology of the disease, the subject has been treated of as fully as time and space would permit. To learn this important subject in all its bearings, the student is recommended to study the comprehensive work so often quoted and referred to in these pages—namely, the *Manual of Psychological Medicine* of Drs. Bucknill and Tuke, of which a second edition has been recently published; and the valuable reports of the Murray Royal Institution for the Insane, near Perth, prepared by Dr. Lauder Lindsay; [also *Lectures on Mental Diseases*, by Dr. W. H. O. Sankey, Griesenger's *Mental Pathology and Therapeutics*, translated for the New Sydenham Society, and *The Physiology and Pathology of the Mind*, by Dr. H. Maudsley.]

#### [THE DELIRIUM OF INANITION—*Delirium of Collapse*.

(DR. CLYMER.)

**Definition.**—*A special form of delirium, happening in certain acute disorders, notably the exanthemata and continued fevers, towards their decline, or at the beginning of convalescence, and, occasionally, in the course of certain chronic diseases. It is, generally, a symptomatic expression of inanition, either the result of a too rigorous diet, or of the inability of the stomach to retain an amount of food necessary for the proper nutrition of the system.*

**Symptoms.**—This interesting variety of delirium, although not noticed by systematic writers, has been observed by several practical physicians, as Chomel, Louis, Graves, Thore, Sauvet, Griesinger, Trousseau, and Gairdner, and has recently been particularly and well described by

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\* There are exceptions: at Edinburgh, where Professor Laycock and Dr. Skae—in London, where Drs. Sankey and Blandford—and at Aberdeen, where Dr. Jamieson—deliver regular clinical lectures. [At New York, where lectures on Mental Diseases are delivered by Professor W. A. Hammond, at the Bellevue Hospital Medical School, and by Dr. D. Tilden Brown, at the College of Physicians and Surgeons.—EDITOR.]

Weber, of London, and Becquet of Paris.\* The diseases in which its occurrence has been specially noticed are typhoid fever, typhus, scarlatina, measles, erysipelas, variola, diphtheria,† pneumonia, and certain affections of the stomach attended by continuous and uncontrollable vomiting. In acute disorders its outbreak is sudden, at the stage of defervescence, or at the beginning of convalescence, when every symptom is favorable, when there is no fever, and the temperature of the blood, as shown by the thermometer, is natural or nearly so, but the pulse feeble, rather frequent, and, sometimes, irregular. In some instances the surface is bathed in perspiration, the face pale and pinched, the expression anxious, the eyes sunken, the nose, forehead, hands and feet cold, with a look of general collapse. The time of attack is generally in the morning on awakening. The character of the delirium, which may be calm or violent, is peculiar and characteristic, there being nearly always one fixed delusion, the subject being generally what has chiefly occupied the patient's mind just before his illness. In one of Weber's cases, as the patient was entering upon convalescence after measles, and his condition seemed every way favorable, he waked in the morning and began to cry, saying that his employer was about to dismiss him on account of dishonesty, and that he was to be sent to prison: he made several attempts to jump out of the window to escape the policemen, whom he thought were coming to capture him. In another case, under similar conditions, the patient became suddenly excited, declaring that a fire had broken out in the poultry-house, and he must go and put it out. The persistent conviction of the death of a friend or relative is a common delusion. Sometimes hallucinations of the special senses, particularly those of hearing and of sight, are present, friends and attendants being mistaken for persons connected with the delusion, and treated and addressed as such.

An attack may last from twelve to forty-eight hours, the patient then falling asleep, and awakening calm, with the mind clear, and speaking of his delusions as vivid dreams. Sometimes, though very rarely, there is a second attack.

Chomel (*Traité des Dyspepsies*) mentions eighteen cases of an affection of the stomach in which this form of delirium appeared, where, on account of obstinate vomiting, little or no food for a long time could be taken; there were great emaciation, a rapid pulse, but no increase of the body-temperature. Andral (*Clinique Médicale*) relates a case of cancerous ulcer of the stomach, in which the patient starved to death, accompanied by delirium with delusions; after death, no appreciable organic alteration was found either in the brain or meninges. In one of Becquet's cases the patient was suffering from an obscure disease, the nature of which was not made out, but a prominent symptom for some time was obstinate vomiting. There was great prostration, when the delirium, calm and with delusion, suddenly occurred. In another case by the same observer, there

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\* [Leçons de Clinique Médicale, par le Prof. A. F. Chomel, vol. i, Paris, 1834. Recherches sur la Fièvre Typhoïde, par P. C. A. Louis, 2ième ed. Clinical Lectures, by R. J. Graves, Dublin, 1843. Remarques sur la delire consecutive aux Fièvres Typhoïdes, par M. Sauvet. Annales Medico-Psychologiques, Paris, 1845. De la Folie consecutive aux Maladies aiguës, par le Dr. Thore, fils. Ann. Medico-Psyc., Paris, 1850. Griesinger, Traité des Maladies Mentales, &c., traduit, &c., Paris, 1865. Trousseau, Clin. Médicale, t. i, 2ième ed., Paris, 1865. Gairdner, London Lancet, vol. i, 1865. On Delirium or Acute Insanity, especially the Delirium of Collapse. By Herman Weber, M.D., Med.-Chir. Trans., 1865. Du Delire d'Inanition dans les Maladies, par le Dr. Becquet, Archives Generales de Medecine, t. i, 1866.]

† [In one of Weber's cases (No. 8) it occurred after severe simple inflammation of the tonsils and fauces.]

was uterine disorder, with constant and persistent vomiting, so that the patient was unable to take any food for some days before the outbreak of the delirium. In both cases there was a great fall of the body-temperature, with other symptoms of collapse. Both recovered.

**Diagnosis.**—This form of delirium must not be confounded with mental aberrations which occur during the increase and at the height of idiopathic fevers, and other acute disorders. Its nature is different from that of febrile delirium, resembling more acute insanity in the character of the delusions. It often happens when there has been no cerebral excitement in the course of the disorder. "It is," says Dr. Trousseau, "the most frequent of all the nervous phenomena which in the convalescence of typhoid fever require the intervention of the physician, and if we are not aware of its possibility and of its nature, and do not rightly know its course, it is very apt to be mistaken for some serious disorder of the brain."

**Prognosis.**—Though Graves speaks of this form of delirium as "violent and dangerous," yet if early recognized, and properly treated, it readily yields, when there is no organic disease of the stomach, and leaves no traces of disordered intellect.

**Nature.**—The immediate cause of this affection is cerebral atony. It is the expression of an exhausted nervous centre, brought on by insufficient nourishment, and excessive drains upon the system, as hemorrhages, intestinal discharges, &c. The attendant general condition is always one of prostration, and sometimes of collapse. The temperature of the body falls, the pulse is weak and rapid, the impulse of the heart feeble, the extremities cold, and the skin is covered with perspiration. There is probably a sudden and transitory change in the capillary circulation of the brain (WEBER), which is deprived of its natural stimulant, the blood (TROUSSEAU).

**Treatment.**—Large and frequent doses of opium should be given, with the simultaneous employment of stimulants, the temperature of the body being raised and maintained, and food, in such form and quantity as will be borne by the stomach, must be persistently taken.

#### CHRONIC ALCOHOLISM—Syn. CHRONIC ALCOHOLIC INTOXICATION.

LATIN Eq., *Alcoholismus*; FRENCH Eq., *Alcöolisme*; GERMAN Eq., *Trunksucht*.

(DR. CLYMER.)

**Definition.**—A series of morbid phenomena following the prolonged abuse of alcoholic liquors, of which the most characteristic are: digestive troubles, tremor, muscular weakness and restlessness, hallucinations of the intellect and senses, anæsthesia, hyperæsthesia, lessened and perverted brain-functions, and often intercurrent attacks of busy and delusive delirium (*delirium tremens*); later there may be sensory and motor paralysis, and acute mania or dementia. These symptoms are due to the direct irritant effect of the poison upon the stomach, and, by its subsequent absorption into the blood, to the gradual impairment of nutrition, resulting in tissue-changes of which the special one is, probably, granulo-fatty degeneration.

**Symptoms.**—This affection begins very insidiously and after a variable time from acquiring the habit of drinking constantly and largely of spirituous liquors. It is not necessarily preceded by attacks of delirium tremens. Troubles of the digestive organs are first noticed. On rising in the morn-

ing the mouth is parched, the tongue dry and coated, and the breath fetid; with nausea, white stringy phlegm, or greenish or yellowish mucus, is thrown off the stomach either by regurgitation or vomiting (*vomitus matutinus potatorum*, HUFELAND). At the same time, or soon afterwards, a train of nervous symptoms sets in. The patient complains of fidgets, and to keep the limbs still requires the exercise of the will; this is particularly the case at night; wakefulness is annoying, and no matter what the degree of drowsiness may be on going to bed, after a few minutes of dozing, the sufferer wakes, and tosses the rest of the night. As the disorder increases there is persistent muscular tremor of the fingers, hands, feet, legs, and tongue. Huss states that this begins always in the hands, but Dr. Anstie says that in the majority of the cases which came under his observation the lower extremities were first affected; progressive inco-ordination-troubles of the hands and legs are soon added, with weakness in the knees and hips; mental restlessness and infirmity of purpose, which may have been present from the outset, increase, along with the inability to sleep, and when the eyes are closed there are annoying mental delusions, or if the patient dozes he is startled by terrifying dreams.

Cutaneous sensibility varies; in some cases there is decided anæsthesia, limited, however, to certain portions of the surface, or to a limb, and accompanied by flying pricking, or itching, or crawling sensations; in others well-marked hyperæsthesia is prominent and annoying, and Leudet thinks that this is more common than is usually supposed; and believes that it is caused by tissue-change of the spinal cord (*Archives Gén. de Méd.*, t. i, 1867). Besides increased sensibility of the skin, which may be general, or in limited patches, pain—sharp, darting, dull, boring, or fixed—is often complained of; and muscular cramps may be very annoying. A marked exaltation of reflex action, provoked by the slightest excitement, is common.

Dizziness is complained of, amounting in the morning on rising to vertigo; specks of various shapes float constantly before the eyes, and Huss has seen instances where objects appeared peculiarly colored; occasionally there is double vision, or objects looked at begin to move on an elevated plane. Headache, lasting from a few hours to several days, or a feeling of binding around the forehead or occiput, or pressure on the sides, or a weight upon the top, of the head, may each, or in the course of the attack all, be felt. Constriction of the chest-walls, or about the throat, with fits of breathlessness, is very frequent and annoying.

In confirmed cases the tongue becomes glazed and fissured; the breath has a peculiar foul smell; the gastric symptoms worsen, and morning vomiting is constant. The complexion at first of a violet red, becomes of a dull white; the face is bloated; and the nose and cheeks are often covered with a form of acne rosacea. Hemorrhages from the stomach and bowels to large extent may happen (ANSTIE). Visual, aural and mental hallucinations are almost constant and distressing, with obstinate insomnia. There is uncertainty of will, and inability to apply the mind to anything, and an idea of vague and unaccountable dread and suspicion, such as some one lying in wait to do harm. Some patients have a constant impulsion toward self-destruction. A feeling of falling through the ground, or tumbling from a height is sometimes experienced, or a miscalculation of distance in stepping up or down. Emaciation may take place, and the muscles of expression are flabby, the eyes watery and injected, and the conjunctivæ have a yellow chlorotic hue. Hepatic, renal, and pulmonic disease is generally present. Sensory paralysis soon follows, with marked increase of the tremor, often to the extent of paralysis



agitans. The mental powers become seriously compromised—"the most common mental condition being one of general intellectual enfeeblement and moral degradation, marked by cowardice and untruthfulness" (ANSTIE). Muscular incoordination is so great as to hinder walking, and often extends to the arms and hands. Convulsions may happen, or cerebral hemorrhage, and there may be hemiplegic paralysis, or general motor paralysis; or, where there is a taint of insanity, attacks of mania may suddenly break out, or the sufferer may fall into a state of hopeless dementia.

**Patho-Anatomy and Pathogeny.**—The lesions found in chronic alcoholism are of two kinds,—the first interests the connective tissue, and the second is a granulo-fatty degeneration of the proper tissue of the organs.

The changes in the connective tissue are chiefly met with in the liver, brain, kidneys, and serous membranes. Hepatic cirrhosis and fibroid degeneration of the lung are type examples. An analogous alteration is found in the cerebrum; it loses color, gradually lessens in volume, and the convolutions, especially those on the upper surface, become atrophied. The cerebellum and medulla oblongata may be affected in like way. The arachnoid and pia mater are usually at the same time infiltrated with serum, thickened, opaque, often colored with hæmatin, and are scattered over with patches or points. The kidneys are atrophied, very firm and granular. Portions of the lungs, particularly the apices, are in a similar condition (see *Fibroid Degeneration of the Lung*), described by Magnus Huss as chronic pneumonia. The mucous membrane of the stomach has numerous irregular vascular patches, particularly on the smaller curvature and about the cardiac orifice. At the level of these patches, principally at the summit of the folds, there are, sometimes, hemorrhagic clots, or elongated erosions, their bases covered by the coloring matter of the blood. Later, the gastric mucous membrane, dotted with pigmentary spots, becomes hard, as well as the subjacent connective tissue. The mucous membrane of the larynx and bronchia is injected, and studded with blood-points. The portal vein and pulmonary artery undergo changes in their coats. The peritoneum, pleura and dura mater show fibroid degeneration.

Granulo-fatty alteration is seen especially in the liver, which increases in size, and tends to assume a cubic form, which distinguishes it from the fatty liver of phthisis, which always keeps its shape. The kidneys are enlarged and cubical in form; the cells of the tubules are filled with fatty granules, and the cortical surface is smooth and has a uniform yellow tint, with sometimes reddish dottings, from injection of the Malpighian bodies. The cells of the brain and its capillaries, the pancreas, salivary glands, glands of the stomach, epithelium of the bronchial tubes and even of the seminal canals, may all be affected with this special degeneration. The heart is of a bronzed-yellow color, soft, flaccid, and loaded with fat at its base; its muscular tissue loses its striæ and becomes granular; and the organ may be hypertrophied and dilated. The bones and cartilages also undergo fatty change. There is habitual coexistence of deposits of fat in the subcutaneous cellular tissue, mesentery, and epiploon.

The two orders of tissue-changes just sketched do not happen with equal frequency in chronic alcoholism. For example, while fatty change is nearly constant in the liver, Dr. Lancereaux found cirrhosis only 35 times out of 130 cases of chronic alcoholism. The latter writer has directed attention to the likeness between the special tissue-changes produced by alcohol, and those which are constant in old age. In both there are the progressive atrophy of the brain, increase of the cerebro-



spinal fluid, granular and fatty changes in the small vessels, in the muscular tissue of the heart, and in most of the elementary tissues, dilatation of the pulmonary vesicles, fatty change in the bones, &c. Both physiologically and pathologically alcoholism brings on premature old age. Acute pneumonia in the habitual drunkard is very apt to take on the form of the pneumonia of old persons, not only in its anatomical site, the apex of a lung, and the disposition to the formation of abscesses, but also in its adynamic and ataxic symptoms.

The effects of the prolonged and undue use of alcoholic preparations in man are then permanent congestions in the bloodvessels, hyperplasia of the connective tissue of the organs, and granulo-fatty degeneration—evidences of devitalization, arising from disturbed and imperfect interstitial nutrition. "The congestion of the lungs, liver, kidneys," &c., says Dr. Anstie, "seems to be partly due to altered chemical relations between the blood and the tissues of those organs, and partly to a paralytic action of the alcohol upon the vaso-motor nervous system. . . . It is indeed doubtful whether the degenerative changes which result are not in great part due to the direct chemical influence of alcohol upon the nervous tissues. . . . There is much in these changes which reminds us forcibly of the effects on nutrition of tissues produced experimentally by Schiff and Mantegazza, by the section of compound nerves, such as the fifth cranial and the sciatic and crural of the lower limb; and suggests the idea that in alcoholic poisoning the starting-point (or at least one starting-point) of degenerative tissue-changes may consist in paralysis of those nervous branches which preside specially over nutrition. It is highly probable, however, that a considerable portion of the degenerative influence of the continued excessive ingestion of alcohol, is due to a chemical interference with the natural course of the oxidation of the blood and tissues" (Reynolds' *System of Medicine*, vol. ii, p. 85).

**Diagnosis.**—The diagnosis of chronic alcoholism is not usually difficult, provided the history and habits of the patient are known, and the state of his organs, particularly the liver and lungs, be ascertained. Morning vomiting, the characteristic complexion, sleeplessness, tremor, and mental restlessness, often associated with delusions of sight and hearing, together with the evidences of contracted liver or lung, put the case beyond doubt. But all these symptoms do not always exist together; yet there is commonly such a combination that, with a history of intemperance, the disorder is quickly recognized. Beginning general paralysis of the insane has some symptoms in common with chronic alcoholism (see *General Paralysis of the Insane*), and so have lead-poisoning, locomotor ataxy, cerebral and spinal softening, hysteria, but with the history of the case, and a knowledge of the special symptoms of each affection, a correct conclusion should be reached.

Dr. Marcet has met with cases of functional disorder of the nervous system from long-continued and over mental exertion, or from anxiety, or from sudden and violent emotions, showing symptoms identical with chronic alcoholism.

**Prognosis.**—This depends in a degree on the resolution of the sufferer, and the duration of the affection. Total abstinence from all spirituous liquors is necessary for a cure in all cases; and when the disease is not in its advanced stages, if the habit of drinking is given up, recovery from immediate symptoms may be promised. If tissue-changes have happened to any extent, the result is more doubtful. The kidney and liver diseases, consequent upon chronic alcoholism, are incurable, and the same may be said of those of the nervous system. Much relief may

be given, and life may often be made comfortable and be prolonged, but a return to full health can hardly be looked for. The weakness and incoördination troubles in the limbs are often persistent, obstinately remaining, after the other symptoms have been overcome.

**Treatment.**—Total abstinence from all spirituous liquors is the first step, and this may be instantly, completely, and safely carried out, if, as Dr. Anstie remarks, the patient be put on substantially nourishing diet. Concentrated broths, with eggs, milk, cream, tapioca, sago, and the like mixed with them, are the best food at first, and should be given moderately warm, in small quantities, and frequently, beginning before rising in the morning. The principles of treatment of chronic alcoholism are the same as those already laid down by the writer for the treatment of other chronic disorders of the nervous system,—nutrition must be improved, and degrading tissue-changes arrested and repaired. This can best be done by suitable food, scaled to the state of the digestive organs, which latter should be put in order as quickly as possible, recollecting, however, that tone and function will gradually return with improved general health. Chalybeates, tonics, baths, friction of the skin, &c., may all be used to aid reconstruction. “Chronic alcoholism,” writes Dr. Marcet, “is not to be cured in a few days; for although under an appropriate treatment a marked improvement may in most cases occur after a short time, a much longer period will be required to restore the patient to perfect health.”

Dr. Anstie has found quinia the best tonic in this disorder; he gives one-grain doses three times daily, and, where the stomach is irritable, in an effervescing draught with bicarbonate of potash and citric acid. The writer has found a combination of quinia and the alcoholic extract of *nux vomica*, or, what he thinks preferable, the extract of *ignatia*, and sometimes with the double salt of the citrate of iron and strychnia, to answer very well. Dr. Marcet is a strong advocate of the oxide of zinc as an appropriate and effective sedative nervous tonic, and recommends beginning with two grains twice daily, and increasing the quantity until ten, twenty, or even forty grains are taken during twenty-four hours. He has published very satisfactory evidence of the efficacy of this remedy in chronic alcoholic intoxication, and there is no doubt that many of the symptoms are benefited by it. Irritability, restlessness, and insomnia may be controlled by half-drachm doses of sulphuric ether with tincture of *sumbul*, or, when the stomach permits, twenty or thirty-grain doses of the bromide of potassium. The writer has found the tincture of *cimicifuga*, made from the fresh root, meet the indications perhaps better than anything else he has tried. When sleeplessness is persistent, and will not yield to these remedies, the extract of *cannabis indica*, or hypodermic injections of morphia, may be used, but narcotics should not be heedlessly resorted to. A light supper and a bottle of stout will, where there are no contraindicating circumstances, often give a good night's rest.

In advanced cases of the disorder, where the tissue-changes are great, cod-liver oil, the hypophosphates, quinia, manganese, iron, *nux vomica*, and above all arsenic, and either the iodide or bromide of potassium, should be given, along with concentrated food. In such cases the Turkish bath, if well borne, or hydrotherapy, will often be of service.

After trembling, and formication, and hallucinations have ceased, but weakness is complained of, with dulness of the mental faculties, noises in the ears, and *muscæ volitantes*, Magnus Huss recommends an infusion of *arnica* flowers.

See Huss, Magnus: *Chronische Alkohols Krankheit*, 1852. Lancereaux, E.: *Alcoolisme*, *Dictionnaire Encyclopédique*, t. 2ième, 1865. Marcet W.: *On Chronic Al-*

coholic Intoxication, 2d ed., Am. reprint, 1868. Leudet: Archives Générales de Médecine, t. 1er, 1867. Foville fils, A.: du Delirium Tremens, de la Dipsomanie et de l'Alcoolisme, Archives Gén. de Méd., t. 2ième, 1867. Anstie, F. E.: Alcoholism, Reynolds' System of Medicine, vol. ii, 1868.]

## CHAPTER VI.

### PATHOLOGY OF DISEASE OF THE SPINAL CORD, ITS MEMBRANES, AND THE NERVES.

#### SECTION I.—DISCOVERIES REGARDING THE STRUCTURE AND FUNCTION OF THE SPINAL CORD AND NERVES.

THE doctrines regarding the nature of the diseases associated with the structure and functions of this portion of the nervous system are in a state of transition. The inquiries into the structure of the cord, the arrangement of its minute component parts, and their connections with each other—with the brain on the one hand, and with the different parts of the body on the other—are only yet being investigated with all the care and appliances of modern research, and to some extent elucidated with success. Difficulties of the most formidable kind surround the anatomical, physiological, and pathological relations of the structures, functions, and diseases of the spinal cord and nerves. Much labor is required, in the first instance, to expose the cord in the dead body, and to examine its morbid states; while delicate manipulations and unwearied research, by the most experienced observers during the last half century, have served alike to show how mysterious and difficult is the subject in all its bearings, and how important and interesting the results. In this arduous task the names of anatomists, physiologists, and the busiest of physicians of this country have, in all such recent investigations, borne a distinguished and pre-eminent part.

In 1811 Sir Charles Bell took the initiative in these researches, and surprised the scientific world by his beautiful and interesting discovery regarding the distinct functions of the *anterior* and *posterior* roots of the spinal nerves.

The epoch of another era is marked by the interesting indication of the existence of the property of "*reflex action*," foreshadowed by Unzer and Prochaska, but which the ingenious and important investigations of the late Dr. Marshall Hall so largely contributed to develop. The names of John Reid, Grainger, Swan, Solly, R. B. Todd, and Bowman, stand prominently forward in this line of inquiry.

A third era is characterized by careful records of the history of cases, by the most inquisitive microscopic research into the diseased parts after the death of the patient, and by physiological experiments in living animals, to show the connections and ar-

rangements of the more minute component parts of the spinal cord (see results of a series of microscopic studies of the *medulla oblongata*, by Dr. John Dean, vol. xiv, "*Contributions to Knowledge*," by the Smithsonian Institution, U. S. America). In this field of research the laborers are not few; and our own country is especially distinguished by the observations of Mr. Lockhart Clarke, the late Dr. R. B. Todd, and Mr. Bowman, Drs. Beale, Handfield Jones, Edward Meryon, J. W. Ogle, Roberts, and Bastian. Abroad, the persevering industry of Kölliker, Valentin, Stilling, Remak, Engel, Van der Kolk, Wagner, Brown-Séquard, and Du Bois-Reymond, and many others, have thrown much light on most of the important questions regarding the structure and functions of the spinal cord. But "let not the spark be lost in the flame it has served to kindle." The beautiful discovery of Sir Charles Bell, while it astonished the scientific world at the time, soon expanded in magnitude and importance. From it, as from a mighty tree, the boughs that have dropped from its parent stem have borne to the earth those living blossoms which, germinating in their turn, are now daily expanding their branches into every land where the Science of Medicine is advancing.

It may be useful, in studying the nature of the diseases associated with the structure and functions of the spinal cord and nerves, to have a distinct conception of the more important general points which seem to have been established relative to its anatomy and physiology, and how far these are illustrated and supported by observations on its morbid anatomy. The following is a condensed statement of the general results established by the prolonged labors of Professor Schroeder Van der Kolk, of Utrecht, a more extended detail of which may be seen in *The Medico-Chirurgical Review* for January, 1857; and which are consistent in many respects with the observations of Valentin and Stilling: (1.) The spinal cord (including the *medulla oblongata*) is the instrument through which the power of motion is generated and expressed, and the co-ordination of movements effected, and through which sensation is transmitted to the brain, and to the gray cerebro-spinal matter of the cerebro-spinal centres. (2.) Complete division of the spinal cord abolishes sensation and voluntary motion in all those parts of the body supplied with spinal nerves from below the seat of injury. Any lesion of the nerve-substance which results from a disease-process may do this if it destroys completely the nerve-matter at the seat of lesion. According to the region in which such a lesion may be situated, so are the different forms characteristic of this loss of power. The nearer the brain and *medulla oblongata* the more immediately fatal to life. If, at the junction of the cord with the *medulla oblongata*, such an injury were to happen, immediate death would ensue, as may be seen when an animal is "*pithed*." Generally speaking, from the head downwards the parts of the body are supplied, *seriatim*, by the nerves coming off from the spinal cord, so that according as the injury to the cord is situated lower and lower down, so does the paralysis affect less and less of the body from below upwards to the seat of disease. Thus in the cervical region, below the

origin of the phrenic nerve, when the lesion is throughout a complete segment of the cord, and above the origin of the superior intercostal nerves, breathing is performed only by the diaphragm and abdominal muscles, while the intercostal muscles cease to act, and the ribs cease to rise and fall. In this condition the patient may live a few days, seldom a week, and never a month (WATSON). If the lesion of the segment occurs below the cervical region; in the upper dorsal portion, for example, the breathing is not affected, or but slightly, while the digestive functions become impaired, and paralysis of the trunk and lower limbs is complete. Such a condition is technically called *paraplegia*. It implies palsy and loss of feeling in the lower limbs, hips, loins, and trunk, according as the injury is higher or lower in the dorsal or lumbar region of the cord. A person in this condition may live a long time, depending greatly on the seat of injury; the higher up, generally the sooner fatal. (3.) The nerves which issue from the cord by two roots (motor and sensific) unite and form compound or mixed nerves, whose filaments or strands pass from their origins to their destinations isolated from each other; and everywhere throughout the body the sensific ramifications of the mixed nerve pass to the surface of the part which is moved by the muscles receiving their motor fibres from the same compound nerve; so that, while the former supply sensation to the part, the latter convey the stimulus to excite the act of motion (VAN DER KOLK). (4.) The anterior (*motor*) and posterior (*sensific*) roots of the compound or mixed spinal nerves are now determined to have certain relations with the gray corpuscular elements of the spinal cord, accumulated throughout its central part in such a manner that a transverse section of the cord shows anterior and posterior *cornua* of this corpuscular gray substance. The corpuscular or multipolar cells, constituting the gray matter, are arranged in several distinct vertical columns, extending throughout the whole length of the cord, the anterior column being the principal. The most considerable of these cells constitute the columns of the anterior horns; next, those by the posterior commissure; then those between the anterior and posterior horns; and, lastly, those in the posterior horns, which rank the smallest in size, and it is even doubted by some whether they are really nerve-cells. These columns of corpuscular nerve-cells are larger and richer in nerve-cells at the cervical and lumbar enlargements; and the proportion of cells increases still more at all those points where the roots of the nerves penetrate into the cord to its gray substance. Thus, clusters of cells, occurring more or less apart, are placed above each other longitudinally (CLARKE, VAN DER KOLK). These so-called nerve-cells are extremely simple in structure. They consist of a more or less rounded mass of matter (cells?), having two or more caudate prolongations, with a circular centre or nucleus and nucleolus containing granular molecules. (5.) The anterior roots (*motor nerves*) spring from the cord itself, and take their origin out of the ganglionic cells of the anterior horn, each cluster of which forms a ganglionic plexus. (6.) The anterior medullary fibres of the cord are the channels through which the influence of the Will from the



brain is conveyed to these corpuscular or ganglionic plexuses, whence these motor nerves take their origin. (7.) The posterior roots (*sensific nerves*) have been traced towards groups of ganglionic cells, but have not been shown to communicate with them. (8.) These posterior roots have been traced to subdivide into two portions, which may be called sets of radicles or rootlets. At the posterior part of the cord one set of these posterior nerve-roots ascends immediately in the white substance, and appears to proceed directly towards and into the brain, thus constituting the channel of sensation; the other portion of the posterior nerve-roots transversely penetrates the white substance of the cord towards the posterior horn of the gray matter, through which it passes. Its fibres there mingle, in part, with certain nerve-fibres which are observed to encircle in a transverse direction the posterior horn of the gray substance of the cord; and, in part, they lose themselves amongst the ganglionic cells of the centre of the gray matter, between the anterior and the posterior horns. (9.) These latter rootlet nerve-fibres, of the great posterior roots, thus constitute the apparatus of reflex action, directing their stimulus through the group of ganglion-cells, with which they appear to be connected, into the ganglion-cells of the anterior horn, from whose plexus of cells the filaments of the motor roots arise. The posterior nerve-roots include, therefore, two descriptions of nerve-fibres—namely, those for sensation proper, and those for reflex-action; and hence the greater thickness (more than double) of the posterior roots compared with the anterior. (10.) These different cell-groups of plexuses of corpuscular cells appear to be united throughout the cord by longitudinal connecting-fibres, so that co-ordination of movement is effected. (11.) The roots of the motor nerves thus receive the excitement or stimulus to action from the group of ganglionic cells in which they originate. It is communicated to them, either through the Will anteriorly, and from above downwards, or by the sensific nerves by their reflex filaments posteriorly, and from the peripheral parts of the body with which the sensiferous nerve-filaments are connected. An individual group of corpuscular cells (whence the motor roots arise) thus becomes susceptible to a *psychical* as well as to a *physical* stimulus. (12.) All reflex action takes place by a definite channel; and its operations seem to be regulated by communicating fibres, which bring the different plexuses of nerve-cells into communication with each other. Thus co-ordination and combination of movements are explained; and so also is the diffusion of action over remote regions, especially in great irritation of the cord, as in attacks of epilepsy and tetanus, or while the system is under the influence of the strychnine poison or hydrophobia. (13.) The gray matter of the cord seems chiefly to avail for *motion*; the posterior part being chiefly subservient to *reflex function*, and to the *co-ordination of motion*; while *sensation* is transmitted upwards exclusively by the posterior and lateral medullary columns to the encephalon, and has probably its proper centre in the *medulla oblongata*. Here, also, is probably localized the centre from which the more universal reflex movements and convulsions take their origin. Experience has convinced Pro-

fessor Van der Kolk that the attention of the Physician ought to be directed to the condition of the *medulla oblongata* in cases of epilepsy. He has frequently succeeded, where the disease has not been of long duration, in procuring a recovery through derivative applications to the nape of the neck; while the pathological changes resulting from protracted epilepsy are not unfrequently manifested by induration of the *medulla oblongata*. The morbid state of the spinal cord, which I accurately determined to exist in *four* cases of tetanus, is also consistent with statements regarding its minute anatomy and the relation of its parts. Each of the four cases exhibited one character in common, and pointed out the spinal cord as the seat of lesion in that formidable malady. The lesion referred to was not manifest to the naked eye, but was determined to exist with certainty by an examination of the specific gravity of the cord-substance. For this purpose the cord was separated from its nerves, and divided into parts of a uniform size, and the specific gravity of each determined. Each of the four cases showed that the general specific gravity of the spinal cord throughout is increased in cases of tetanus, the average specific gravity of the healthy cord being 1.036. They showed, also, that a change is *suddenly* indicated about the region of the cord in immediate communication with the wounded part, and that in one case of idiopathic tetanus the change was uniform throughout. In the first case I examined, where the wound was on the occiput, the uppermost three inches of the cord were of the highest specific gravity, and the difference became *suddenly*, and *not gradually*, manifest at the fourth inch. In the third case a very marked difference was apparent when the cervical region was compared with the rest of the cord; and the difference was suddenly marked where the roots of the cervical and first dorsal nerves left the cord to form the brachial plexus. The wound in this instance was on the fingers. "In the last case the difference was suddenly manifested in the lowermost part of the cord, corresponding to the region where the nerves were in communication with the lower limbs, which were the seat of the injury" (*Glasgow Med. Jour.*, No. IV, Jan. 1854). Mr. Lockhart Clarke has since examined the cord in tetanus cases microscopically, and has found peculiar lesions of a most minute kind scattered throughout its substance (*Med.-Chir. Reports*, August, 1865); and more investigation shows a great increase in the growth of cell elements in the implicated portions connected by nerves with the site of injury. (14.) The anterior commissure of the cord is distinguished from the posterior by the decussation of the fibres. After their intersection, when traced downwards, these fibres are observed to be deflected so as to run in part along the margin of the anterior fissure, interlacing themselves within the white substance; and in part to enter the inner edges of the anterior gray horns, where they mingle with the encircling fibres already noticed, which spread themselves thence in the medullary columns of nerve-cells and join the longitudinal fibres. Their function is probably to maintain the motion of the right and left sides of the body. The fibres of the posterior commissure have a parallel course, without any intersection. (15.) Some observations on the

secondary affections of the cord by Dr. Türck seem to show that when disease destroys a certain portion of the nervous centre, the strands, filaments, or cords of nerve-substance which proceed from that centre, or arrive there, subsequently degenerate, having ceased to receive or to convey an impulse. This degeneration takes place in the same direction in which these strands or filaments convey impressions; thus in the centripetal fasciculi (posterior columns) the secondary affection occurs always in the centripetal direction; while in the centrifugal fasciculi (anterior columns) it shows itself in the centrifugal direction; and in the mixed fasciculi (lateral columns) it shows in both directions. Thus it happens that secondary affections of filaments or strands of fibres in the spinal cord may result from a lesion in one of the hemispheres of the brain; generally in about five weeks after the primary injury.

This observation may be regarded as a rule; and to some extent it may also explain cases of so-called "*paralysie musculaire progressive*" which have been described by Meryon, Cruveilhier, Aran, Valentiner, and Roberts; although the nervous lesion has only been demonstrated in some of the cases.

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## CHAPTER VII.

### DETAILED DESCRIPTION OF THE DISEASES OF THE SPINAL CORD.

#### SPINAL MENINGITIS.

**LATIN**, *Meningitis spinalis*; **FRENCH**, *Méningite spinale*; **GERMAN**, *Entzündung der Häute*—**Syn.**, *Meningitis spinalis*; **ITALIAN**, *Meningitide spinale*.

**Definition.**—*Inflammation of the membranes of the spinal cord.*

**Pathology.**—The inflammatory states of the membranes of the cord, and the morbid effects they produce, are the same as those of the membranes of the brain—namely, serous effusion, which may terminate in suppuration or softening.

The rachidian *dura mater* may be inflamed either at its free or at its adherent surface. On examining the spinal canal, after caries of the vertebræ, the areolar tissue uniting the dura mater to the walls of this cavity is often found greatly loaded with venous blood, and in some instances is broken down, so that the dura mater is entirely detached—a state most probably due to inflammation. This inflammation may terminate by resolution, or it may advance, and serum be effused between the osseous structure and the dura mater. In this site the effusion has no communication with the cavity of the cranium, because the dura mater of the cord, while it is but loosely attached by areolar tissue to the vertebræ, is very firmly attached round the margin of the *foramen magnum*, and especially to the basilar portion of the occipital bone.

The *dura mater* of the cord also appears liable to the ulcerative process and to gangrene. In a case given by Ollivier (vol. ii, p. 569), of a druggist who died on the twentieth day, after suffering from lumbar pains, with rigidity of the trunk and lower extremities, together with tetanic spasms, there was found, on cutting through the muscles of the lumbar region, half an ounce of pus, or more, which was traced to the cavity of the arachnoid, the rachidian *dura mater* having ulcerated and ruptured.

The spinal *arachnoid* and *pia mater* are liable to inflammations similar to the corresponding membranes of the brain.

Diffuse inflammation of all the folds of the *arachnoid* has often been observed; those membranes being red and injected for a greater or less extent.

Effusion of serum, both into the cavity of the *spinal arachnoid* and between the *dura mater* and *pia mater* of the cord, is not uncommon. Such effusion communicates freely with the cavity of the cranium, so that fluid may pass easily from the one to the other.

Suppurative inflammation of the spinal membranes also occasionally takes place in the cavity of the *arachnoid*. Frequently, if the inflammation is acute, it is associated with disease of the *cerebellum*, or of the intracranial membranes; and in the chronic form it rarely exists except in connection with caries of the vertebræ.

Effusion of fluid within the cavity of the spinal cord is analogous to fluid in the ventricles of the brain. It leads to compression of the cord, and paralysis as a symptom of such compression. *Hydro-rachis*, as it is termed, is usually a congenital state, associated with a defective development of the vertebral arches. The paraplegia it induces generally extends to the sphincters and the bladder, producing incontinence of fæces and urine. The condition is indicated by a tumor at the lower end of the canal, over the region of the sacrum; and the cavity of such tumors communicates with the cavity of the vertebral canal; and the paralyzing influence is occasioned by the pressure of the fluid on the spinal cord. Congestion of the membranes may occur as a stage preliminary to all of these conditions.

**Symptoms.**—The symptoms of *rachidian arachnitis*, or *meningitis* of the cord, are often obscure at the commencement; but once formed, the disease is characterized by pains in the back, with affection of the muscles, and retention of urine. Paralysis does not occur, except by pressure produced by exudation of fluid, or by extension of inflammation and disorganization of the cord itself (MERYON). Ollivier, quoted by Meryon, gives a case of spontaneous *spinal meningitis*, in which the symptoms closely resembled those of structural lesion of the cord itself. The patient, a man aged twenty-four, was admitted into the Hôtel Dieu with obscure symptoms. On the fifth day of the disease he had paraplegia, with hyperæsthesia of the lower extremities. Three days afterwards the arms became partially paralyzed, semiflexed, and stiff; the right pupil was more dilated than the left; the association of ideas was slow. Two days subsequently he died. The cellular tissue which surrounds the *dura mater* of the spinal canal was filled with

vessels injected with blood. The spinal cord was enveloped in a layer of gelatinous yellow matter, between the arachnoid and pia mater. It was most abundant over the lumbar enlargement, and extended as high as the third cervical vertebra. It was thickest where it covered the posterior columns of the cord (*Traité de la Moelle Epinière*, tome ii, p. 551, 2me edition).

One marked symptom of congestion of the membranes, referred to in a case related by Dr. Meryon, is the difficulty experienced in walking on first arising after a night's rest. I have observed this to be a constant indication of spinal congestion; and it may be temporarily induced by *strychnine* or *nux vomica*.

The affection of the muscles varies from simple stiffness of the part to *opisthotonos*. This latter symptom is often limited to the neck or trunk, without the limbs participating, as in a case given by Rayer, in which the trunk and neck were drawn backwards, while the patient walked freely till the time of his death. In the case of a waggoner thrown off his cart, and pitched on his neck and shoulders, the neck was stiff, the jaw was locked, the body convulsed, and the patient delirious. It was not till the twelfth day, however, that the lower extremities became affected and palsied, when the patient sunk into a typhoid state and died. A large quantity of pus was likewise found in the spinal arachnoid cavity. Neither the pulse, nor the tongue, nor temperature, is much affected at the commencement of *spinal meningitis*, but towards its close the pulse becomes rapid and feeble, the tongue brown and dry, and the teeth fuliginous. The patient is now said to be "*typhoid*," and he dies delirious or comatose. Retention of urine generally persists from the beginning to the termination of the disease. Constipation often exists to a great degree at first, but afterwards the bowels act regularly, or even suffer from diarrhœa.

**Diagnosis.**—The symptoms which distinguish *spinal arachnitis* from inflammation of the substance of the cord are *pain* and *contraction* or *convulsions* of the limbs; for in pure *myelitis* there is seldom any severe or constant pain, while the limbs are generally palsied, and their sensation benumbed or lost. It is distinguished from *rheumatic lumbago* or *psoas abscess* by the affection of the limbs and of the bladder.

**Prognosis.**—Many authorities considered *spinal arachnitis* to be incurable, but numerous cases marked by the characteristic symptoms in a mild form do recover.

**Treatment.**—*Spinal arachnitis*, seldom depending on a morbid poison, is perhaps in all cases best treated by bleeding and mild purgatives. General bleeding is sometimes necessary; but local bleeding, either by cupping or leeches, along the vertebral column, is most useful, and cannot be omitted with safety. The medical treatment consists in moderate purging by the neutral salts, as the *sulphate of soda* or the *sulphate of magnesia*; for, as these act on the bladder as well as on the bowels, they are probably the best remedies. But whatever purgative may be selected, it will be proper to combine it with the *tincture of hyoscyamus*, or other mild opiate, to procure the patient some relief from his sufferings. Ergot



of rye (*secale cornutum*) has been very much used in France; and Dr. Meryon speaks favorably of its effects, combined with *iodide of potassium*, in a case which manifested no complication of spinal effusion (*Medical Times and Gazette*, Aug. 31, 1863). The warm bath is an excellent adjuvant in the earlier stages of the disease; whilst in the latter stages *blisters*, *setons*, *moxæ*, or the ointment of the *tartrate of antimony*, are more beneficial, or at least are deserving of a trial. The paralyzing effusion may disappear during their use, combined with the action of diuretics. The *external* application of *belladonna* and *chloroform* will be found of essential service in diminishing the violent pain which accompanies meningitis of the cord (MERYON). Cold, in the form of bladders filled with ice, applied along the spinal cord, may be of service where congestion prevails.

Abstinence from all animal diet should be imperiously prescribed throughout the whole course of the disease.

In cases of *hydrorachis*, where a tumor is associated with *spina bifida*, iodine injections have been proposed (VELPEAU, DEBOUT, BRAINARD, GROSS, MERYON). In operating, the puncture should be very small, by a small, flat, curved needle, directed subcutaneously into the sac. A drain of the fluid may then be allowed to escape through a canula, and the injection used must be very weak at first, the object being to excite a slow process of inflammation in the cyst. One-eighth of a grain of *iodine*, and a quarter of a grain of the *iodide of potassium* in solution, is the quantity prescribed for injection by Dr. Gross. He then closes the puncture with a twisted suture, and coats it over with collodion. An anodyne should be administered, and the child kept lying on its face. If the life of the child be saved, paraplegia is still apt to remain, and perhaps involuntary defecation and micturition (Meryon in *Brit. Med. Journal*, July 11, 1863, p. 28; also *Practical and Pathological Researches on the various Forms of Paralysis*, p. 25).

#### EPIDEMIC CEREBRO-SPINAL MENINGITIS—SYN., CEREBRO-SPINAL FEVER, OR MALIGNANT PURPURIC FEVER.

LATIN, —; FRENCH, *Fièvre cérébro-spinale*—Syn., *Fièvre purpurée maligne*; GERMAN, *Epidemische meningitis*; ITALIAN, *Febbre cerebro-spinale*—Syn., *Meningitide cerebro-spinale*.

**Definition.**—“A malignant epidemic fever of an acute specific character, of sudden invasion, attended by painful contraction of the muscles of the neck and retraction of the head. In certain epidemics it has been frequently accompanied by a profuse purpuric eruption, and occasionally by secondary effusions into certain joints. Lesions of the brain, the spinal cord, and their membranes, are found on dissection. The course of epidemic cerebro-spinal meningitis is rapid and very fatal, attended with great prostration of the powers of life, severe headache, and pain along the spine.”

**Pathology.**—During the early part of 1865 a disease of this nature prevailed epidemically about the Lower Vistula; and in

other parts of North Germany. It had some characters of being a specific fever like *Typhus*, but its characteristic lesions were chiefly on the surfaces and enveloping membranes of the cord. The *pia mater* was found in fatal cases to be swollen and infiltrated, or perhaps suppurating and disorganized. But these changes of the surfaces are also *indicia* of changes more or less destructive within the nerve-centres. Lymph is more rarely effused, yet has occasionally been found organized, uniting the opposite sides of the serous sac together. The *pia mater* and the *arachnoid* have also been found adherent after effusion of lymph; and instances have occurred in which the layers of the spinal membranes have been found united to each other. There is reason to believe that this disease has prevailed as an epidemic in Europe at different periods and different places, during the fourteenth, sixteenth, and seventeenth centuries, and in the eighteenth century in France, Germany, Italy, England, Ireland, and Scotland. In the present century it has prevailed at Geneva, in Switzerland, and in some parts of North Germany, and in Ireland, and in the United States of America. The name of the disease is apt to fix the attention on local structural changes (which are probably only secondary, and may not always be present) to the exclusion of a consideration of the real nature of the malady. It has been regarded, therefore, by some, as a form of simple acute cerebro-spinal meningitis; but its rapid course and greater severity, and its clinical history, clearly show that it is more than a simple meningitis. The whole of the nervous system is gravely implicated from the first, so that it kills at an early period, leaving no evidence of local structural change. On the other hand, there seems to be no constant relation between the severity and duration of the illness and the extent of the lesions seen after death. Again, there are not a few who regard the disease as a variety merely of typhus fever (BOUDIN, MURCHISON, UPHAM); and cases are met with, particularly when cerebro-spinal meningitis and typhus prevail together, which favor this view. But it is believed that the suddenness of the invasion, the rapid course, the absence of the mulberry rash of typhus, the early appearance of the purpuric eruption or hæmic spots, are sufficient to distinguish epidemic cerebro-spinal meningitis from typhus fever. Each seems due to some specific poison, which some believe to be of malarious origin; others, again, of the nature of influenza, or of typhoid pneumonia. The exact nature of the disease seems, therefore, to be still undetermined; and pathologists have rather stated what it is *not* than what it is. So far it can be said that it seems to be a substantive specific disorder, with constant symptoms, and therefore ought, perhaps, to be considered under the general diseases of a specific febrile nature.

**Symptoms.**—The invasion is sudden; or weariness and a general sense of uneasiness may be felt for a day or two before more acute symptoms set in. Most usually shivering prevails, intense vertigo, headache of intolerable severity, violent obstinate vomiting, and painful muscular stiffness (which soon develops into tetanic con-

traction), particularly of the neck and back. While consciousness lasts, the distress in the head is incessant, and even during delirium or stupor the patient's instinctive movements show that the head is the chief seat of pain. The eyes are expressive of wild distress, the face generally pale, pupils contracted, and conjunctiva red. Terrible restlessness and general muscular agitation are soon added to other symptoms. Often the sensibility of surface is so great that every touch or movement causes agony. The neuralgia increases, and muscular contractions become more and more uncontrollable and convulsive, affecting, like tetanus, all parts of the body. Deglutition is affected; respiration becomes irregular and imperfect. The head is dragged tightly backwards upon the neck, and the features are fixed in the characteristic grin of lock-jaw. Consciousness becomes effaced, and delirium tends towards stupor, the patient passing into coma, or into a depression on the confines of death. By this time emaciation has greatly progressed, and goes on, from the profuse sweating which is apt to attend the typhoid state, into which he rapidly passes. Anæsthesia, muscular paralysis, drooping of the eyelids, or squinting, are not unfrequent phenomena; the pupils being dilated, or motionless, or unsymmetrical. When death ensues, it is generally from the fifth to the eighth day; otherwise the duration of the disease may extend over three or four weeks, and convalescence may be of many months' duration. After a few hours, to one or more days, an eruption or marking becomes visible, upon the skin of the neck, abdomen, back, arms, legs, and face. It is of a hæmic or purpuric appearance, and made up of distinct dark-red or purple spots, about the size of a pin's head or a little larger. The spots are not raised, nor do they fade on pressure, and in some cases are not visible till after death. The tongue remains moist and creamy till the spasmodic stage becomes developed, when it becomes dry, dark-colored, or even black, fissured and swollen, or covered with sordes. Herpetic eruptions on the lips are not uncommon. The respirations become slow and labored, and as a fatal issue approaches they become hurried, irregular, infrequent, or stertorous; the heart-beats and the pulse become quick, feeble, and tremulous, or weak and slow, with cardiac blood-murmurs (DA COSTA). The body-temperature rarely rises above 100° Fahr. Death is generally by coma, or from paralysis of the heart by lesion of the *medulla oblongata*.

The duration of attack varies from a few hours to many weeks. A fatal issue is recorded in as short periods as three, four, five, six, twelve, and thirteen hours; but more than half the fatal cases die between the second and the fifth day. Convalescence may begin from the fifth day to the fourth week, or later, and is always tedious. Health is not restored for many months. Relapses are not infrequent, and often mortal. The most frequent complications are—congestion of the lungs; sero-purulent effusions into serous cavities, especially the pleura and joints; erysipelas; sore throat; swelling and suppuration of the parotid gland.

**Prognosis.**—The disease has been marked by great fatality; and no case can be regarded without great anxiety.

**Treatment.**—At the outset, stimulating embrocations to the spine and extremities, with moist or dry heat to the limbs, and wrapping in blankets, give some relief. Opium in large doses is the only drug which has appeared to be of any service in subduing the virulence of the symptoms. Quinine, in cases where malaria aggravates the disorder, is also well spoken of. No evidence appears favorable to the use of bloodletting, veratria, aconite, strychnia, alcoholic stimulation, ammonia, camphor, valerian, musk, or mercury—all of which have been tried. Ether and chloroform inhalation have been of use as sedatives; and tincture of cantharides has been of service in cases marked by extreme depression. Counter-irritation, by the actual cautery applied along the spine, or by blisters, has been followed by alleviation of the pain and other symptoms.

[The foregoing article of the Author being chiefly a brief of the Editor's article upon Epidemic Cerebro-Spinal Meningitis, published in the first American edition of this work (1866), the Editor reprints the original article, with a few additions, which are marked thus : [ ].]

#### [EPIDEMIC CEREBRO-SPINAL MENINGITIS.\*

(DR. CLYMER.)

**Definition.**—*An acute specific disorder, commonly happening as an epidemic, general or limited, and, rarely, sporadically; caused by some unknown external influence; of sudden onset, rapid course, and very fatal; its chief symptoms, referable to the cerebro-spinal axis, are great prostration of the vital powers, severe pain in the head and along the spinal column, delirium, tetanic, and, sometimes, clonic, spasms, and cutaneous hyperæsthesia, with, in some cases, stupor, coma, and motor paralysis; attended frequently with cutaneous hæmic spots; its morbid anatomical characters being congestion and inflammation of the membranes of the brain and spinal cord, although there is reason to believe that the evidence of these changes may be wanting, even in cases of long duration.*

**History and Geographical Distribution.**—Dr. Tourdes, who has shown great industry in the study of the history of cerebro-spinal meningitis, is of the opinion that it prevailed epidemically in Europe at different periods of the fourteenth, sixteenth, and seventeenth centuries. Dr. Valleix remarks, that owing to the descriptions being too incomplete, and the absence of post-mortal examinations, not much weight can be given to these researches. During the eighteenth century it is probable that there were outbreaks of it in France, particularly in the French fleet at Brest (1758), Germany, Italy, England, Ireland, and Scotland. In the present century (1805), it is said to have prevailed at Geneva, Switzerland; but the recorded post-mortal examinations do not prove the assertion, though the

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\* The name cerebro-spinal meningitis is not a proper one for this affection, even with the prefix "epidemic," for, as Dr. Valleix remarks, it is "begotten of anatomical bias and an incomplete appreciation of the facts." It gives no accurate notion of the real nature of the disorder, and takes heed only of the local structural changes, which are, probably, secondary, and may be wanting. It has been also called *typhoid meningitis*, *malignant meningitis*; but to these names the same objections lie. *Spotted fever* is hardly distinctive enough, there being so many other essential disorders in which spots on the skin appear. *Petechial fever*, has been proposed by Dr. G. B. Wood, of Philadelphia. [Dr. W. Stokes calls it *malignant purpuric fever*; and Dr. R. D. Lyons, *febris nigra*.]

symptoms of a short endemic there resembled those of cerebro-spinal meningitis in some respects. While occasional cases, symptomatically like the disease, had happened in the United States towards the close of the eighteenth century, it was first distinctly seen in the epidemic form at Medfield, Mass., in 1806, and from that time until 1816 it was constantly epidemic throughout this country, particularly in the New England States, upon which it seemed to fasten. It is, however, quite evident that all the epidemics described as cerebro-spinal meningitis during that period cannot properly be classed as such. From 1816 to 1860, casual endemics, with sporadic cases here and there, seemed to have occurred in several of the States, particularly the Southern and Western, the Middle and Eastern having been comparatively exempt. "In 1816-17 the disease appeared in various parts of South Carolina; from 1818 to 1822 it was seen in Mecklenburg, Lunenburg, and Brunswick, Va.; in some parts of the Western States during the spring of 1819, and also in North Carolina, and the mountainous parts of Virginia; in 1821 in Franklin County, Pennsylvania; in 1823 it was recognized at Saco, Maine, Berlin, Connecticut, and in the Shenandoah Valley, Va. In 1823-4-5 it broke out in the vicinity of Middletown, Conn., and also in some parts of that State in 1826, and likewise at New Orleans, and Fort Adams, and in 1827, in Trumbull County, Ohio. In 1832, sporadic cases occurred in New London, Connecticut, which in many respects resembled this disease. . . . There is no mention made of it from that date till in 1845-46, when an epidemic prevailed in Clark County, Illinois, called 'black tongue,' thought by Dr. McCoy to be cerebro-spinal meningitis, on account of the post-mortem appearances. In the early part of 1847 it was seen in Mississippi, Tennessee, Missouri, and Arkansas, [?] and resembled a modified pneumonia; and in the winter of 1847-48 at Washington, D. C. Dr. Ames has given an account of it as it appeared at Montgomery, Alabama, during the winter and spring of 1848. Dr. Sargent read a paper before the Massachusetts Medical Society on this disease as it showed itself in Millbury and Sutton during 1849; it was also noticed, the same year, at Mecklenburg, New York. Again a few cases happened in New Orleans during the last days of January, 1850, and in Central and Western New York during 1857. In October, 1859, there commenced an epidemic at Castlecraig, Virginia, which continued nearly a year" (WEBBER, p. 61-2).

From 1814 to 1857 circumscribed epidemics of cerebro-spinal meningitis seem to have been constantly prevalent in various parts of Europe,—in the garrisons at Grenoble and Metz, France, in 1814 and 1815; in Ireland in 1813, 1814, and 1815; in Italy and Germany in 1817; at the Millbank Penitentiary, near London, in 1823; in parts of France in 1832, and again, in a number of the garrisons, from 1837 to 1842. The disease was chiefly confined to the military, but in a few instances extended also to the civil population; notably at Strasburgh, in 1841 (C. BROUSSAIS). It appeared at Civiano in the Neapolitan Kingdom in 1837, and various districts of Naples were invaded in the winter and spring of 1840. In 1840, and again in 1845, it appears to have occurred in Algeria. From 1843 to 1849 it prevailed in more or less of the departments of France, but chiefly amongst the soldiers. In 1844, there was an outbreak of undoubted cerebro-spinal meningitis at Gibraltar. In 1846, a disease, called and described by Dr. Robert Mayne (*Dub. Quart. Journ. of Med. Science*, vol. ii, 1846), as "cerebro-spinal arachnitis," broke out in the workhouses and hospitals of Dublin, the spinal membranes suffering more than the cerebral. Spain was visited in 1849, and Sweden, from 1854 to 1861. In



1863, '64, '65, it prevailed as an epidemic of wide range, and with great mortality, in West Prussia, and reaching as far as the Grand Duchy of Baden. About the same time, a very fatal disease devastated parts of Russia, and which, from Dr. Burdon Sanderson's Report, was epidemic cerebro-spinal meningitis.

[A severe and extensive epidemic broke out in Dublin, in March, 1866, and reached its height during the following year, extending to other towns in Ireland, and some cases happened in England, Lincolnshire, and in London. The British forces in Ireland suffered much in proportion to their average strength.]

The rise and progress of the late epidemic of the disease in this country has been described by Dr. Webber. It prevailed "to a considerable extent amongst the troops [of the United States Army] in camps and barracks during the late war" (WOODWARD), and also in the Confederate Army (GAILLARD). It was first seen during the winter of 1861-2 in the Army of the Potomac [?] and in Livingston Co., Missouri, amongst the United States troops. "In the fall of 1862, it appeared among the negroes who were taken to Memphis, Tennessee, by the Union Army; and one or two cases were met with among the soldiers in the vicinity of Newbern, N. C.; during the winter of 1862-63, and the spring of 1863, it appeared in La Grange County and other portions of Northern Indiana; at Newbern, N. C., during January, February, and March; and during these months, and also April, at Newport, Rhode Island, among the midshipmen of the Naval Academy; in February and March it was seen at Philadelphia, and during the latter part of the year at Cambridge, Ohio. During the two successive winters of 1862-63 and 1863-64, it was epidemic in Morgan County, Illinois. During the winter of 1863-64, the negroes at Memphis were again visited by it; and in the same winter and succeeding spring, parts of Clark and Crawford Counties, Illinois; in the northwestern parts of Pennsylvania, and parts of New Jersey, it was noticed during this year, and also in 1862 and 1863. Only a few cases occurred around New York. In January it was in Brattleboro', Vermont; and in January and February, in Philadelphia, and at Benton Barracks, Missouri. Through March it was seen at Brandon, and St. Albans, Vt., and Louisville, Kentucky, and during January, April, and March, there were cases in Boston; in May, at Chicago, at Leland, and in Williamson County, Illinois. In the latter part of July, cases occurred at the Stanton General Hospital, Washington, D. C. In October, Mechanicsburg was visited by it, and in November, Marshall, Ill., and during the latter part of the year, St. Paul's, Indiana. During the winter of 1864-65, a few cases were seen at the City Hospital, Boston; in January, 1865, at Greenwich, and in April, at Palmer, Mass.; in the latter part of the month at Kewana, Ind., and early in the year at Palestine, Ind.; in May, at Nittany Hall, Pa. From September, 1864, to May, 1865, the disease appeared amongst the troops at Gallop's Island, Boston Harbor" (WEBBER, *l. c.*, pp. 63-4). [In April, 1863, four cases occurred in a single regiment of the 22d North Carolina (Confederate), of which three died; and eight cases and six deaths in the 3d Alabama (Confederate), February, 1863. Dr. Sandford B. Hunt saw several cases in the camp of a Missouri regiment, at Little Rock, Arkansas, during the winter of 1864-65 (*San. Comm. Med. Mem.*).]

That cerebro-spinal meningitis may happen as an endemic, or epidemic limited to one place or district, or even institution, or family, there is abundant evidence. Well-marked sporadic cases, settled beyond doubt by post-mortal examinations, have been reported. One of the most in-

teresting, is that of a newly enlisted man in the Mississippi Squadron in 1864—the only one—in which the meningeal lesions were well marked. Dr. Gilbert has reported two sporadic cases which he saw at Gettysburg, Pa., in 1844, and another in Philadelphia, in 1846. Many others are upon record. In the winter of 1863–64, the writer saw a single case, in one of the U. S. A. General Hospitals at Beaufort, S. C., and several more in the spring of 1865, in the Army Hospitals at Savannah, Georgia. [Dr. Samuel Wilks saw one case in each of the years, 1856, 1858, 1859, in London. Dr. Day, a fatal case at Stafford, in 1859, and another in 1865.] Dr. Geddings, of Charleston, S. C., mentioned to the writer in 1865, he had met with occasional cases amongst the negroes of that city, and related one of a negro laundress, in which the disease proved fatal within twenty-four hours after seizure.

[Notwithstanding its wide geographical distribution, there is no corresponding diffusion amongst any one population during its prevalence, it, as a rule, being limited during an outbreak to certain localities and to certain portions of the population of such localities. The distribution of the disorder seems to be by a series of isolated eruptions rather than by general spreading.]

**Morbid Anatomy.**—The morbid appearances found in the cerebro-spinal axis and its membranes are: when death has happened within two or three days after the invasion, opalescence of the upper surface of the cerebrum, seemingly in the subarachnoid fluid; an abundant vascularity of the membranes of the brain, chiefly of the pia mater; a large increase of serum in the subarachnoid space and in the ventricles, clear or turbid, and mixed with flocculi of lymph, and as often as otherwise, even in cases of the briefest duration, an abundant exudation of thick, yellowish, apparently semi-organized lymph on the base of the brain and the medulla oblongata (UPHAM). The membranes at this stage are sometimes remarkably dry, without injection; or they may be adherent to the surface of the brain, or among themselves. The cerebro-spinal fluid is much increased, and of a yellowish and milky hue; the spinal meninges congested like those of the brain; and the cord has been found softened. Where the disease has lasted for any time, the cerebral exudation is soft, opaque, yellowish, two or three lines in thickness, and has been compared by Dr. Tourdes to a layer of butter spread over the brain; or it may be denser and have a pseudo-membranous look. It is found chiefly along the course of the vessels, and, when small in quantity, limited to, and ramifying with, them; or it may be in little, irregular patches of variable size, scattered over the brain surface, or covering the pons or medulla oblongata, or cerebellum, or parts of the cerebrum; or, it may completely envelop the cerebrum, cerebellum, and the intracranial cord. Commonly superficial, it may dip down with the pia mater amongst the convolutions; or be found in the ventricles, “in the posterior cornua of the lateral ventricles, in its concrete form particularly, or else tingeing and thickening, with an opaque greenish pus, the serous fluid of the whole cavity” (UPHAM). According to Dr. Tourdes this happens in about one-half the cases. Purulent infiltration of the choroid plexus, and superficial softening of the walls of the ventricles, have been seen. The substance of the brain is occasionally softened. The spinal membranes are affected in like manner, and the cord itself, like the substance of the brain, may be injected, or even softened.

[Dr. Burdon Sanderson found the gelatinous substance under the microscope to consist of cell-like bodies, either adhering to each other so closely that they could not be completely separated, or embedded in a

transparent interstitial substance, while the sero-purulent fluid which occupied the spinal subarachnoid space, and in some cases the ventricles, exhibited corpuscles and granules floating freely. The cell-like bodies, although in general resembling pus-corpuscles, did not present that uniformity of size and character which are met with in normal pus. They were usually, but not always, of regular circular contour, and varied in diameter from  $\frac{1}{1500}$ th to  $\frac{1}{1200}$ th of an inch. Occasionally they exhibited the appearance of an external cell-membrane, but in most instances this could not be made out even in perfectly fresh exudations—cases that were examined as early as eight hours after death. They invariably contained numerous granules, some of which were cleared away on the addition of acetic acid. Those which remained were highly refractive, but did not assume any special form of arrangement. The interstitial substance was beset with granules, some of which were albuminous, and others fatty. It was most abundant and distinct on the surface of the spinal arachnoid, where it infiltrated the fine connecting tissue and minute bloodvessels of the pia mater.]

In all epidemics of this disease cases have occurred in which no appreciable changes have been found in the cerebro-spinal membranes. Two interesting instances have been reported by Dr. Levick, of Philadelphia (*Am. Jour. of the Med. Sciences*, July, 1864, and July, 1865)—one an adult female, and the other a child, aged eighteen months, both of whom died in twelve and fourteen hours after seizure in the midst of health—in which the cerebro-spinal meningeal vessels were filled with black blood, but there were no traces of inflammation, and the substance of the brain and medulla oblongata was natural in appearance and consistency.\* The late Dr. Valleix remarks, that when there is more or less absence of the meningeal changes, it is among those who have been struck down by the disease as by a thunderbolt; and there is certainly much testimony to this effect; but Dr. J. J. Woodward, speaking of cerebro-spinal meningitis as observed in our armies during the rebellion, in this connection writes: "There were at least two classes of cases brought under the observation of this [the medical] department [of the army]. In the first, the autopsy disclosed grave anatomical lesion of the cerebro-spinal axis, accumulations of serum, sero-pus, pus or tough yellow lymph, especially in the ventricles about the base of the brain and in the upper part of the spinal canal. In the second class of cases, no perceptible anatomical lesion in the cerebro-spinal axis was observable. These two groups of cases rest upon equally reliable evidence, and are not to be disposed of on the supposition that the latter represent merely an early stage of the former, since it is to be remarked that *both* anatomical conditions appear to have been found indifferently in protracted cases as well as those which proved suddenly fatal."†

The blood is usually very dark-colored and fluid, even in the briefest cases; [but in some instances firm fibrinous clots have been found in the heart after death (TOURDES, STILLÉ).] The coincident lesions are: congestion and œdema of the lungs; pleural, pericardial, and articular sero-purulent effusions; and, occasionally, enlargement of the glands of Brunner and of Peyer, without ulceration.

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\* [Dr. Parks, in quoting these cases, calls attention to the omission of stating whether or no there was opalescence, or unnatural dryness of the membranes (*loc. cit.*, p. 47).]

† Letter to Dr. L. Parks, quoted in *Report of a Committee of the Massachusetts Medical Society, on Spotted Fever or Cerebro-Spinal Meningitis in the State of Massachusetts*, May, 1866. Boston, 1867.]

**Symptoms.**—As a rule the invasion is sudden, without warning indisposition, except, perhaps, general weariness, or aching of the whole body, or shivering, which latter is often the initial symptom, and may amount to a sharp chill. In some cases prodromata happen. Gilchrist states that in the Gibraltar epidemic, weariness and a sense of general uneasiness were frequently felt for a day or two before the onset of the acute symptoms. Dr. Tourdes says that sudden invasion was the exception, not the rule, and that it happened in at most one-quarter of his cases. Dr. George G. Tucker, of Westfield, Mass., in reporting sixteen cases, remarks: "The mode of invasion varied considerably in different cases. In some there were the usual symptoms of febrile disturbance; in others there were no formative periods or premonitory symptoms, the patient being suddenly insensible and convulsed, with the rapid form of opisthotonos" (*Mass. Med. Society's Report*, p. 104, 1066). But the "formative," or premonitory symptoms given by Dr. Tucker really mark the actual beginning, and this is nearly true of those mentioned by Dr. Tourdes as "precursory." The onset of the disorder is almost constantly marked by severe headache, usually occipital, sometimes frontal; patients speak of it as acute, excruciating, and unbearable, and, in some instances, as if the brain were bound, or stretched, by a band, or by a bar of metal. Dizziness may be complained of. Excruciating pains in the nape of the neck, limbs, calves of the legs, and joints, particularly the knee-joint, and likened to those of rheumatism, singly or collectively, are felt at an early stage, along with stiffness of the jaws and neck. A sudden and acute pain in a joint, or in a finger or toe, compared to the stinging of a bee, has sometimes marked the access. Stinging pains in the arms and legs later in the attack have been noticed as sometimes occurring in the late epidemic in this country. Sore throat, in some epidemics, has been a common initial symptom. Dr. Upham speaks of cases which began like a severe cold, and in such he found a tendency to palsy of the muscles of the tongue and face; and the same has been noticed by some of the German writers. There are often soreness and tenderness at the back of the neck and along the spine. Nausea and vomiting may happen at the outset. Generally there is great weakness, from the beginning; the pulse is quick and feeble, or it may be little changed; the breathing may be natural, or slow and oppressed, or sighing; the surface is cool and moist, the face suffused, and eyes bloodshot. Delirium may set in soon, instances in which raving delirium has begun within half an hour of the onset, having been recorded; or may happen later; it is generally not violent, but wandering, or talkative, or muttering, with perversion and dulness of intelligence, and great apathy, though the patient may be readily roused to consciousness; at other times there may be initial stupor, or even coma. The intellect may be unimpaired throughout the attack. From a few hours to one or more days, the eruption is seen upon the neck, abdomen, back, arms, legs, and sometimes the face; it is hæmic and distinct, made up of dark-red or purple spots of the size of a pin's head to that of a dime, not raised, nor fading on pressure. [In the late epidemic in Ireland (1866-67) the common form of the eruption was *purpura*. Sometimes there are only dark mottled spots scattered here and there over the skin. The eruption is not constant.] In some cases the hæmic spots have not appeared until after death. [Of 98 cases admitted into the Philadelphia Hospital in 1866, 36 had petechiæ; 13 mixed petechiæ and erythema; 9 erythema and urticaria; 3 indistinct petechial mottling; and 37 no eruption at all (GITHENS). In the Lower Vistula epidemic hæmic spots were comparatively rare; in the



Irish epidemic nearly constant. Rubeoloid patches have been occasionally observed.]

In some epidemics herpes first on the lips, and then extending to other parts of the face, and to the ears and neck, has appeared about the second, third, or fourth day. Dr. Phelps, of Brattleboro', Vermont, mentions having seen them in the late epidemic in this country, [and Stillé says of the Philadelphia outbreak, "herpes labialis was noticed in a few cases." Hirsch, describing the eruption in the Lower Vistula epidemic, (1865) calls it "A vesicular eruption (eczema), sometimes herpetic in character, chiefly appearing in the lips, but occasionally extending over the sides of the face, diffused more or less on the trunk, or showing itself in patches on the limbs. It has occasionally taken the form of shingles. When associated with purpura the vesicles may be flattened and rest upon a livid base." The tongue is moist and creamy. To these symptoms others are soon added: cutaneous hyperæsthesia, local or general, is very constant, and sometimes to such a degree, that slightly touching or brushing the skin with the hand will bring on reflective muscular contractions; anæsthesia is very rare, and generally at a late stage. The muscles of the nape of the neck become rigid and retracted, and the head is thrown back; this is one of the most constant and persistent symptoms. This rigidity and retraction may extend to the muscles of the back, and the flexors of the forearm, and the muscles of the jaws, and have been noticed in those of the abdomen, and in the flexors of the legs; they are tetanoid, and may amount to opisthotonos and trismus. Sanderson says that in the Prussian epidemic 1864-65 there were many cases in which there was no stiffness or retraction of the muscles. Twitchings in the muscles often precede them. Epileptiform spasms, which may be general, or local, unilateral, or limited to a limb, have been occasionally noticed. The tongue may now become dry, dark-colored, or even black, fissured and swollen, or covered with sordes. The bowels as a rule are constipated, but there may be diarrhœa and constipation by turns; nausea and vomiting may recur, after having disappeared, or they may not set in until the second or third day; the urine is scanty, and retention and strangury are not infrequent. The respirations are slow and labored, and towards the close of fatal cases hurried and irregular, or infrequent, or stertorous. The heart-beats and the pulse are often quick, feeble, and tumultuous, though sometimes weak and slow; and well-marked cardiac blood murmurs have been heard (DA COSTA). [In 6 adult cases, Burdon Sanderson found the pulse to vary from 56 to 98, the average beats being 85. In 98 cases observed by Githens, the pulse varied from the natural standard to 150 beats per minute. In all it was very weak, with a dicrotic tendency, sometimes entirely imperceptible in the radial artery, and always interrupted by slight pressure.] There would seem to be direct correlation between the frequency of the pulse and the body-temperature; the latter, according to the German observers, rarely rising above 100° Fahr. [The highest temperature in different cases noted by Githens varied between 100° and 105° Fahr.; while in two it was below 100°.] The skin may be dry or moist; and profuse and irregular perspirations, sometimes of the head and face only, have been mentioned by a few writers, and their sickly smell noted. Disorders of the organs of sight and hearing, though happening, are by no means constant; squinting, and some degree of deafness, and buzzing in the ears, are the most common. The state of the pupils is very variously reported as being dilated, contracted, irregular. Notwithstanding the great debility, decumbency is most generally on the side, at least until towards the last, though there are usually much restlessness and tossing about.



When the issue is happy, there is a gradual or sudden abatement of the symptoms, with reaction, and sometimes, the supervention of mild fever. Death happens by coma, or by paralysis of the heart and lungs from damage to the medulla oblongata, or, possibly, by asthenia.

The chief and more constant symptoms of this disease, found on a careful analysis of a large number of undoubted and uncomplicated cases, have been grouped in the description; but they are rarely all seen together in the same case, or in all epidemics of cerebro-spinal meningitis; so various are its manifestations in the same epidemic, and at one time and at another. In some instances, the progress is so rapid that there is no time for the development of many of the symptoms, the patient being struck down, and dying quickly, as in blasting cases of scarlet fever, diphtheria, and typhus, [the fulminant form of recent writers.] Or one or more of the common symptoms may be absent during a whole epidemic. There are properly no stages of this disorder; all the essential symptoms noticed in its course may be present at the outset, and with initial severity, or they may follow each other in close sequence. Abatement in the intensity of some symptoms is occasionally seen, and others disappear and return, or pass away entirely; but marked remissions, except, perhaps, where there is malarial complication, are not common.

The *duration* of an attack is from a few hours to many weeks. Professor J. S. Jewell has recorded a case which ended fatally within three hours from the onset; Dr. Lidell another, in five and one-half hours; the German authorities, many within four hours; and Dr. J. J. Levick, two in twelve and thirteen hours respectively. [Dr. S. Gordon reports a well-marked case of death within five hours of seizure.] More than one-half the deaths happen from the second to the fifth day. Rummel says that the disease runs its course more quickly at the beginning than at the close of an epidemic. [In the Lower Vistula outbreak the most acute cases terminated fatally in from twelve to seventy-two hours. Of the cases in the Philadelphia Hospital (1866), the deaths were from forty-eight hours to fourteen days, while the cases which ended happily lasted from twenty to thirty days; the acute symptoms rarely continued beyond two weeks (GITHENS). In the late Irish epidemic, a large proportion of the fatal cases died in from ten to forty-eight hours; in others, death happened at the end of the second and during the course of the third week (RADCLIFFE). In 95 fatal cases analyzed by Parks, the duration in 66 was five days or less, in 1 eight days, in 28 ten days or over.]

*Convalescence*, taking place from the fifth day to the fourth week, and often later, is tedious, and full health is sometimes not regained for months. General weakness and lassitude are long complained of, and one or both legs may remain feeble for awhile; or any impairment or perversion of the senses which happened in the attack may last for some weeks; and occasionally boils or abscesses appear. *Relapses* are not infrequent, and often prove fatal; though in the Massachusetts epidemic of 1810, recovery seemed to be the rule.

Several *forms* or varieties of epidemic cerebro-spinal meningitis have been described by writers. There is no doubt, as Dr. N. S. Davis remarks, that "in regard to the disease promiscuously styled 'spotted fever' and 'cerebro-spinal meningitis,' as reported in our literature, no less than three or four diseases have been confounded together." (*The Transactions of the American Medical Association*, vol. xvii, 1866.) The force of the ætic poison may be directed chiefly to the brain, or the spinal cord, or lungs, causing various symptomatic expressions; but in most of these cases there are certain typical traits which mark the real nature of the disorder.

In *blasting* [*fulminant*, or *siderant*,] cases, the nervous symptoms are less objective; there is great prostration of the vital powers from the outset; the pulse is small and feeble, and gets hourly weaker; the skin is cold, pale, and mottled; the face and extremities livid; the features pinched, and the expression anxious; the respiration hurried, sighing, or irregular; the eyes staring and glazed; and there is deep lethargy, with death by coma. Yet very rapid cases happen, in which acute symptoms persist almost to the last. The "epidemic influence" is seen in the occurrence of cerebro-spinal symptoms in other contemporaneous disorders, as pneumonia, erysipelas, diphtheria, and typhous and typhoid fevers. (See vol. i, p. 351.)

The most frequent *complications* are: (1.) Congestions of the lungs; (2.) Affections of serous membranes with sero-purulent effusions, particularly of the pleura and joints; (3.) Malarial toxæmia; (4.) Erysipelas; (5.) Sore throat; and (6.) Swelling and suppuration of the parotid gland.

**Mortality.**—Most epidemics and endemics of cerebro-spinal meningitis have been marked by great fatality: in some instances throughout their whole course, in others only for awhile after the outbreak. It decimated one of the United States volunteer regiments in the West during the late war, one man in every ten dying from it. In one of the French towns, of 240 taken, 120 died. In one Swedish epidemic, of 3051 persons attacked, 1387 died. In 1035 cases in several towns of France, there were 592 deaths, or a mortality of 1 in 1.76 (C. BROUSSAIS). In the Southern States during the war, in one instance there were 66 deaths in 154 cases, and in another 24 deaths in 40 cases (JEWELL). The mortality from the disease in that section of the country during the rebellion has been estimated at from 60 to 80 per cent. (GAILLARD). In the first winter of its visitation at Memphis, there were no recoveries. In 35 cases reported by Dr. S. C. Young, of Grenada, Miss., not one got well; and Wunderlich, Stonone, and others, have given instances where every case was fatal. [The death-rate in the several epidemics between 1838 and 1865 varied between 75 per cent. and 20 per cent. (HIRSCH). In the Massachusetts epidemic (1866) there were 278 cases and 170 deaths, or a mortality of a little over 61 per cent. In the Philadelphia Hospital (1866) there were 43 deaths and 130 cases, or a mortality of 33 per cent. (GITHENS). Dr. Stillé remarks that while ten epidemics, in various places, between 1838 and 1848, gave an average mortality of 70 per cent., a like number during the decade from 1855 to 1865 gave only about 30 per cent.]

**Prognosis.**—No case of this disorder can be regarded without anxiety by the attending physician, particularly during the first three or four days; for one-half the deaths happen before the fifth day. After the fourth or fifth day, if fatal symptoms are not present, the prognosis is more hopeful. But the patient is not safe even when convalescence has fairly set in, for there is always a risk of relapse, which in some epidemics has been constantly mortal. In *blasting* cases attended with collapse or coma, no recoveries have been reported.\* The influence of age and sex upon the issue of the disease has not been determined. It is asserted to be more fatal between ten weeks and seven years than at any later period of life of equal length. The favorable signs are, an abatement of the symptoms referable to the cerebro-spinal axis; general reaction, shown by the state

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\* [Mr. J. Netten Radcliffe says, without giving, however, instances or authority. "Recovery from this form of epidemic cerebro-spinal meningitis is not unknown, but is an exceedingly rare event" (Reynolds' *System of Medicine*, vol. ii, p. 680).]

of the heart, pulse, and skin ; and a more natural respiration. There are many instances on record in which pregnant females have got well.

**Diagnosis.**—Though the groups of symptoms vary in different epidemics and in different cases of the same epidemic, and are rarely all present in any one case, there is generally such a combination of them, sufficiently peculiar and typical, that a diagnosis is readily made. The sudden onset and rapid development of the disorder, with morbid phenomena clearly referable to the central nervous system, overwhelming prostration, and the characteristic eruption, are all distinctive. In some cases the symptoms are imperfectly developed, or are more or less marked by coincident disease in other organs ; in such instances the circumstances of the access, the prevalence of epidemic cerebro-spinal meningitis, and exclusion, will serve to point out the real nature of the case. It should be remembered that sometimes the pathogenetic force seems to be spent at one time upon the brain, at another upon the spinal cord ; and, setting aside all doubtful cases in which the primary disorder was probably pulmonary, occasionally upon the lungs. Many diseases, as erysipelas, diphtheria, scarlet, typhoid, and typhous fevers, especially the two latter, are, unquestionably, very liable to be, in some degree, influenced by the prevailing epidemic. When typhus or typhoid fever are accompanied with spinal symptoms, the diagnosis may be embarrassing ; but as a general rule, such is not the case. (See article on *Spinal Symptoms in Typhoid Fever*, by the Editor, vol. i, p. 350.) The invasion of typhoid fever is marked by more or less illness for several days ; subsequently its symptoms are characteristic ; and whatever spinal epiphenomena may happen, they cannot with any care lead to a confusion of the two disorders, except in very exceptional cases. The differential diagnosis of epidemic cerebro-spinal meningitis and typhous fever, will be considered when the nature of the disease is discussed.

The cerebral variety of congestive malarial fever, beginning with intense headache, and soon followed by deep coma, may very closely resemble the blasting form of epidemic cerebro-spinal meningitis with early coma. The writer has seen several cases of this kind. When happening in a paludal district, or during the prevalence of an epidemic of the latter disorder, they may be very puzzling. Fortunately the treatment is the same, and its success or failure will materially assist in clearing up any doubts. Simple acute cerebro-spinal meningitis, as a primary disorder, is probably a nosographic myth, and never a morbid entity. When it happens as a secondary, or traumatic, affection, its previous clinical history and its behavior are so widely different from the epidemic disorder, that to confound them would seem impossible.

**Etiology.**—Of the host of predisposing and exciting causes of this disease which have been catalogued by writers, all have been named in connection with other disorders, endemic and epidemic, and most or all have been wanting in some outbreak of cerebro-spinal meningitis, or in the several localities where it has prevailed, or in individual cases. In this respect nothing constant has been noticed. Dr. Jewell writes: "The disease, as regards cold, has often been prolonged into the hot season, and has often begun as an epidemic and prevailed extensively in midsummer, as many as eight times in France. It has prevailed in rural districts as well as civic districts, and has even seemed to prefer rural districts in some cases, as in our own late epidemic. It has appeared among both rich and poor ; on the uplands as well as lowlands ; among those well fed as among those poorly fed ; among those who have enjoyed thorough ventilation, as well as those under an opposite condition ; among the civil as

well as the military population ; and so in relation to the whole of these known assumed causes, of which the malady seems to be wholly independent, except that they may, in a general way, predispose to or intensify it ; for *not one* of them has been observed to be constantly associated with the disease" (*loc. cit.*, p. 34).

[Of one hundred and eighty-two European epidemics, twenty-four were in October and November, forty-six in December and January, forty-eight in February and March, thirty in April and May, twenty-four in June and July, and ten in August and September (SIMON). The outbreaks in this country have been chiefly during the winter and early spring. In Sweden, of 417 local outbreaks, 311 were in winter, and 106 in summer. Of 85 epidemics in Europe and the United States, noted by Hirsch, 33 prevailed in winter, 24 in winter and spring, 11 in spring, 1 in spring and summer, 2 in summer, 1 in summer and autumn, 1 in autumn, 1 in autumn and winter, 3 in autumn, winter, and spring, and 6 throughout the whole year.]

It has been very capricious with regard to *sex*, in various epidemics and localities, sometimes males being chiefly its victims, at other times females. All *ages* have suffered, from the infant a few weeks old to the octogenarian. In the Belfast workhouse it was almost confined to boys between the ages of seven and twelve years (MAYNE) ; most of the cases seen by Hirsch, Niemeyer, Rummel, and others, were under fifteen years, and the same was remarked during the Danish and Swedish epidemics, and those at Stettin and Bromberg. In Berlin, the disease was almost limited to the adult population. Pleiffer says it prefers winter, soldiers, and children. The young and vigorous would seem to be more often attacked than the feeble, the sickly, and the aged. In 116 cases, in which the age is given, there were : between 1 and 15 years inclusive, 39 ; between 16 and 30 years inclusive, 64 ; between 31 and 46 years inclusive, 12 ; and 1, aged 68 years (WEBBER). In France, it is looked upon as a military disease, soldiers in garrison, and particularly recruits, being the chief sufferers. Dr. J. J. Woodward writes : " Recruits have not escaped, and those have especially suffered who were crowded in barracks and draft rendezvous."

[Dr. John Simon writes : " Epidemics have seemed particularly apt to occur in establishments where masses of special population have been living in common domicile—as in workhouses, convict prisons, schools, and (above all) barracks. And in several such cases the epidemic has seemed to confine itself to one section of the establishment—to one block of building, to one floor, or to one room. It is asserted that, as a general rule, the affected segment of population has been in over-crowded and ill-ventilated quarters. And when the disease has spread from such centres, or has independently arisen among common populations, this, almost always, has been said to have been under similar unwholesomeness of circumstances. Where the epidemic has been among soldiers, officers have enjoyed almost entire immunity ; and where common populations have been suffering, the disease has shown great, if not exclusive, preference for the worst lodged classes of the community. . . . In some cases, according to local reports, the distribution of an epidemic has very decidedly not been governed by conditions of over-crowding and ill-ventilation" (*Eighth Report of the Med. Officer of the Privy Council*, 1865).]

The mass of testimony is against its being contagious ; [but Drs. Hirsch and Stokes have reported cases of apparent communication of the disease from the sick to the well ; and Boudin gives instances of its appearance in garrisons, and among the civil population of towns, after the introduction of detachments of troops among whom the disorder had prevailed or was prevailing at the time.]



There is reason, therefore, to believe, that the effective cause of epidemic cerebro-spinal meningitis is to be sought for beyond physical and bodily conditions; that it is outside of the degree of heat, moisture, &c., and the constitutional state of the individual; and we are forced to take refuge in the assumption of an unknown special morbid agent as the ætiologic factor.\*

**Nature.**—It has been well said by Dr. O. W. Holmes, with respect to the disorder as it prevailed in this country from 1810 to 1816, that “it is easier to say what it was *not* than what it *was* ;” and the same remark holds good of every epidemic and endemic of the disease which has since happened, both in Europe and the United States. Many have regarded it as a form of simple acute cerebro-spinal meningitis. Aside from the great infrequency of this affection as a primary or idiopathic affection, and the improbability of epidemic or endemic outbreaks of it, it may be urged that the behavior and the results of treatment of the disease under consideration are not explained upon the supposition of its being a simple meningeal inflammation. Those pathologists who place it nosographically by the side of inflammations of serous membranes are forced to assume that “the epidemic constitution imposes upon these phlegmasiæ a more rapid course, and a greater gravity than obtains in sporadic inflammation of the meninges; that is the only point they appear to differ from the latter” (MONNERET); which is pure hypothesis, and gives no rational explanation of the observed differences, but in reality concedes the agency of an unknown external cause. Besides, the clinical history of epidemic cerebro-spinal meningitis shows that the whole system is implicated from the outset, and it often strikes down its victims without leaving any trace of local damage. Nor is there any constant relation between the severity and duration of the symptoms and the degree and extent of changes in the cerebro-spinal membranes; they are present or absent alike in protracted cases as in those which have proved suddenly fatal, writes Dr. J. J. Woodward, as the result of the observations made by the medical officers of the army during the late war. Granting, however, that they are always present, it would not prove that the disorder was a modified idiopathic meningeal inflammation, any more than the invariable changes of the agminate glands of the intestine in typhoid fever would entitle that affection to be regarded as a dothineritis. There is a great difference between a pathogenetic cause and an anatomical character. Those who hold to the localization of the disorder, and regard the meningeal lesion as primary and essential, seem to have taken as hasty and limited a view of its manifold morbid phenomena, as Ploucquet, Marcus, and Clutterbuck did of continued fever, when they referred it to inflammation of the brain.

On the other hand, amongst those who believe that epidemic cerebro-spinal meningitis should be classed with the so-called zymotic diseases, there are many who consider it but a variety of typhus fever. Boudin, Murchison, Upham, Baltzell, and others, have ably argued this question.

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\* Dr. Robert Law, of Dublin, in reporting several sporadic cases of cerebro-spinal meningitis, observed by him in 1865, writes: “It is a fact worthy of recording, that at the time we were attending this lady [suffering from cerebro-spinal meningitis], nine rabbits out of eleven, which her son had, died all in the same way; their limbs seemed to fail them, they fell on their side, and then worked in convulsions, and died. Two hens fell lifeless from their roost.” Three of the rabbits were examined; in two there were congestion of the vessels of the base of the brain, and in the other vascularity of the membranes of the spinal marrow.—(*Dub. Jour. of Med. Science*, May, 1866.)



That there is a certain analogy between the two disorders, and that cases occasionally are met with, particularly when cerebro-spinal meningitis and typhus are coincidentally prevalent, which would seem to favor this theory, must be admitted (see vol. i, p. 351, and *Dublin Quar. Jour. of Medical Science*, 1849); but there are so many striking points of difference in the history of the two affections, that it is difficult to understand how they have ever come to be looked upon as one. The identity of the two diseases has been supported by some of the highest authorities in the profession, who have had large means of clinical observation, and their views are entitled to consideration; but the evidence upon which they are founded cannot here be examined, or the subject properly discussed. The suddenness of the onset in epidemic cerebro-spinal meningitis, its rapid course, the absence of the mulberry rash of typhus, the early appearance of the hæmic spots, and its non-contagiousness, whilst many of the characteristic symptoms of the continued fever are wanting, are all diacritic traits, which should, it is believed, prevent any confusion between the two diseases. Dr. Burdon Sanderson says, that the facts observed at Dantzic afforded no proof of there being anything in common between epidemic cerebro-spinal meningitis and typhus, except so far as each was due to a specific poison (*Med. Times and Gazette*, May, 1865). Dr. Luther Parks writes: "We can conceive that on the negative side of the question of the connection of 'spotted fever' with typhus, the same line of argument may be used as that of Dr. Holmes in speaking of the epidemic of 1806 to 1815—that a disease which is sometimes almost as sudden in its invasion as a stroke of lightning; which is rarely suspected of being contagious; which gives us a solitary case in a ship-of-war, a single case in a boarding-school, two cases only in an almshouse; which in civil practice affects the villages and isolated farm-houses of the interior (where typhus 'running the ordinary course' is unknown) as much, at least, as the large cities; which, in a great majority of cases, is fatal in a few days or even hours; the mortality of which is very variable: such a disease presents so many points of difference, when compared with British typhus, that we should hesitate before pronouncing the two identical."

[Tourdes, Levy, Lebert, Niemeyer, Hirsch, Stokes, Gordon, and other Irish and English authorities, clinically familiar with the disorder, protest against confounding, pathogenetically, typhus and epidemic cerebro-spinal meningitis. Hirsch says: "Apart from its very obscure pathological essence, there is hardly anything in its symptoms or lesions which brings it [epidemic meningitis] within that comprehensive and elastic term—*typhus*." Dr. Stillé, whose opportunities for examining both diseases, at about the same period, were large, writes of Hirsch's remarks: "We fully adopt the language just quoted, as faithfully representing what we conceive to be the truth" (p. 116). Dr. J. Netten Radcliffe answers the question, *Is epidemic cerebro-spinal meningitis a form of, or allied to, typhus?* in these words: "It differs from typhus in the aspect of the patient, rhythmical progress, range and course of temperature [lower and irregularly fluctuating,] form of cerebral affection, character of eruption, sequelæ, rate of mortality, anatomical lesions, and manner of dissemination. Differing in all essential particulars, doubt can only arise when the two diseases prevail together" (Reynolds' *System of Medicine*, vol. ii, p. 699).]

It has also been regarded as a form of pernicious paludal fever, [but there are no sufficient grounds to believe it to be of malarious origin.]

Dr. Levick believes that there is an epidemic influence which shows it-

self in its mildest form as influenza; again as typhoid pneumonia; once more as cerebro-spinal meningitis; whilst in some cases the blood itself appears to be greatly affected without presenting at once the phenomena of disease of any special organ (*Am. Journal of the Med. Sciences*, vol. xlviii, 1864).

The pathogeny, then, of epidemic cerebro-spinal meningitis is still unsettled. While there is reason to say that it is not a variety of simple or idiopathic inflammation of the membranes of the brain and spinal cord, nor of typhus fever, nor of pernicious paludal fever, but a substantive disorder, consistent with itself in all material points, with constant symptoms produced by a constant cause, and hence entitled to be described and regarded as a distinct disease [whose proper nosological place is amongst general diseases born of an external morbid poison,] it must be owned that its pathogenic nature remains unknown.

**Treatment.**—There is no antidote to the specific ætic poison, nor can it be expelled by elimination. The indications are to stay if possible the progress of the disorder, and sustain the vital powers. A hot bath [102°–106°,] in which the patient is to remain a short time only, and to be immediately wrapped in blankets, often gives some relief. When the surface generally, or the extremities are cold, friction with turpentine and chloroform may be used, or sinapisms put on the arms and legs. A moderate quantity of blood may be drawn by cups, or leeches, applied to the back of the neck, followed by counter-irritation, or a blister. Brandy, or ether, or chloroform, in small quantities, may be administered both as stimulants, when indicated, and to allay the nausea and check the vomiting. Two or three large doses of quinia with opium should be given, at proper intervals; or hypodermic injections of morphia about the seat of pain. The bowels ought to be kept free by purgative enemata, containing croton oil, turpentine, &c. Nutritious and suitable food must be taken, when possible, at short intervals, and through the night as well as the day. During convalescence fresh air, good diet, and tonics are required.

Various plans of treatment have been suggested and adopted in this disease; some agreeing with the pathogenetic notions of their partisans, others being purely empirical. The results have not been very happy, though it is difficult to justly estimate the true value of each method, from the varying severity of individual cases, and of different epidemics. General bleeding has had its advocates in all countries and in every epidemic; but it is believed that an honest examination of the testimony, both for and against it, will show that it is harmful. Veratrum, aconite, and digitalis, have been tried, and proved unsatisfactory, though Rummel recommends digitalis in the early stage. Opium has been greatly extolled, and the late Dr. Valleix says that, in large doses, it is the only drug which has appeared to exert any real influence over the disease; and this good opinion of its effects is confirmed by Minor, Tully, Hirsch, Boudin, Forget, Levy, Boutin, C. Broussais, Lidell, W. H. Draper, Levick, Stillé, and others. [One grain of opium may be given every hour in very severe cases, and every two hours in less severe ones (STILLÉ).] Niemeyer speaks well of subcutaneous injections of morphia. The evidence in behalf of quinia is very conflicting. Hirsch, Rummel, Forget, Tourdes, J. S. Jewell, and Upham, believe that it does no good; whilst Gassaud, who states that he lost but 2 cases out of 126 treated, attributes this wonderful result to the free and early use of quinine; and Gerhard, Wales, Ottmar, Burr, Durand, Leonard, and Levick, have found it decidedly beneficial. It is, however, generally admitted, that when there is malarial complication, or the disease happens in paludal districts,

it should be given. Nothing appears in favor of brandy, ammonia, camphor, valerian, or musk; and the same may be said of mercury, strychnine, and ergot. Rummel, Hirsch, and others praise the iodide of potassium in the later stages, but no real proof of its utility has yet been brought forward. The bromide of potassium has been given, and it may possibly prove a valuable adjuvant in controlling some of the symptoms, and, as it cannot do harm, merits a fair trial, but hardly to the exclusion of other remedies. Inhalations of ether and of chloroform have been found by some of the French physicians useful as sedatives. The tincture of cantharides has, in the experience of Professor Allen, of Chicago, yielded good results, particularly in cases marked by great depression (JEWELL); and from its happy stimulant effects, in cases where there is great depression of the nervous system, its employment is rational. The sulphites and bisulphites, and permanganate of potash, have been proposed, and used during the recent epidemic in this country, on account of their supposed antiseptic properties; but no reliance should be placed upon them as nullifiers of the blood-poison. There is much strong testimony in behalf of counter-irritation and blistering along the spine. The actual cautery freely applied to the back, has been followed by great alleviation of the pain and other symptoms (ROLLET). At the outset of an attack, stimulating embrocations to the spine and extremities, and moist and dry heat to the limbs, have been much employed and praised by physicians of all countries, and are without doubt often very relieving. Dr. J. Burdon Sanderson recommends the application of ice-cold to the spine during the first day. [Dr. J. Netten Radcliffe says: "The application of *cold* to the head and spine, either by means of ice or a freezing mixture, in Esmarch's (or Chapman's) India-rubber bags, has furnished by far the most satisfactory results of all direct treatment" (*l. c.* p. 702). If there is much prostration during the local use of cold, the trunk and extremities should be kept warm by cotton-wool, hot sand-bags, or hot-water bottles.

When this disorder appears, either periodically or as a local outbreak, all the sanitary measures, commonly used in other affections of a common origin, should be put in force. Mr. J. Simon says: "I am strongly of opinion that the best sanitary precaution which, in the present state of knowledge, can be taken against the disease, must consist in care for the ventilation of dwellings."]

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The medical journals of this country, published during 1864, '65, '66, and those of Dublin and London, during 1866-67, contain valuable articles, but too numerous to mention separately here.]

## MYELITIS.

LATIN Eq., *Myelitis*; FRENCH Eq., *Myélite*; GERMAN Eq., *Entzündung des Marks*—Syn., *Myelitis*; ITALIAN Eq., *Mielitide*.

**Definition.**—*Inflammation of the substance of the spinal cord.*

**Pathology.**—As the spinal cord is a continuation of the brain, and similarly composed of medullary and cineritious matter, it is reasonable to expect that its diseases will be similar. Such is observed to be the case. Inflammation of the cord may be diffuse. It is characterized, post-mortem, by a few more bloody points than usual, or by a slight red or rose-color suffusion throughout its substance. There is reason to believe, writes Dr. Abercrombie, that inflammation of the substance of the cord, like the corresponding affection of the brain, may terminate fatally, either—(1.) In the *acute inflammatory stage*; (2.) By *ramollissement*; (3.) By *undefined suppuration*; or (4.) By *abscess*.

The most common affection, however, is ramollissement or serous inflammation, in which the substance of the cord is greatly broken down and softened, so as to be sometimes reduced to a mere pulp; or so diffuent as to give the sensation of fluctuation under the finger. The disorganization may embrace the whole thickness of the cord, sometimes only one of its columns, so that it is of variable extent. It is constant, however, and the centre or *gray* substance of the cord is more softened than that of the circumference or white substance. The ramollissement may exist in the cervical, dorsal, or lumbar portions; but it is most common in the lumbar, and after that in the cervical portions, or in those parts which contain the greatest quantity of *gray* substance, and the greatest number of bloodvessels. The part affected is generally swollen—a circumstance more striking than in similar diseases of the brain,



because the spinal canal is large in proportion to its contents, compared with the cranium. The softened part is also generally ash-colored or white. Some pathologists have regarded ramollissement of the cord as a particular alteration of the nervous system, resembling the effects of a contusion of soft parts, and the result of shock. It often occurs, however, when no shock has been received, and has not the least resemblance to a contusion of soft parts.

Induration of the spinal substance is another result of *myelitis*, and probably depends upon a form of inflammation in which fibrinous exudation becomes consolidated. Portal states he has found the cord of a cartilaginous hardness, while the membranes were red and inflamed; and Abercrombie gives a similar case.

The substance of the cord may likewise become infiltrated with pus, or it may be collected into an abscess. The fact of infiltration is perhaps questionable; but there can be no doubt of an abscess having occasionally formed in the substance of the cord. Velpeau gives a case, quoted by Dr. Abercrombie, in which an abscess was formed in the right column of the cervical portion of the cord, three inches long and two lines broad, while a smaller one existed in the left column (*Revue Méd.*, vol. ii, p. 217).

**Symptoms.**—The symptoms of *myelitis* are in general limited to the parts below the injury. In a few cases, however, the effects of the accidents are reflected from below upwards. In general, both upper or both lower limbs are affected; but in a few instances only one limb. The earliest symptoms are recognized in the fingers and toes, in the feeling of numbness, with a sensation of coldness extending up the limb. Shortly afterwards the patient complains of pain in the back, corresponding to the seat of greatest intensity of the inflammation. This is not constant; but when we make pressure with the finger over the spinous processes of the affected part, it may be augmented or only then felt. These symptoms are succeeded by impaired motion, and often likewise by diminished sensation of one or more limbs, followed by paraplegia or other form of palsy. If only one side of the cord be affected, the paralysis which results is confined to one side of the body. When the anterior columns chiefly are the seat of the inflammation, the paralysis which follows is that of muscular motion, but of sensation if the lesion exist in the posterior columns; and if a careful analysis be made of the several cases in which the gray substance of the cord has been implicated, it will be found that the function of reflex action has been deranged (MERYON). In the early stage, when congestion prevails, there is exaltation of tactile sense and of muscular contraction. Another marked symptom may be often distinguished—namely, a difficulty experienced in walking on first rising after a night's rest—a feature more or less constant in cases of spinal congestion. The palsied limbs may be either relaxed or permanently contracted: thus, the hand may be bent on the upper arm, or a leg be flexed upon the thigh, or the affected limb may be attacked with convulsive twitchings, or may beat incessantly. As the disease advances, the bladder becomes affected, and the patient is incapable of retaining his urine, from the sphincters being palsied. The



action of the bowels is slow in the first instance ; but towards the close of the disease the patient may be purged, and the stools pass involuntarily. If the disease be the result of an accident, the pulse is at first rapid and full ; but if the disease be spontaneous, the pulse is generally natural, until the powers of life are broken down by the continuance of the affection. As death approaches, the nates and the prominent parts of the pelvic region, on which the body rests, ulcerate extensively, so that deep sloughs form ; and although the patient, from anæsthesia, suffers no pain, he nevertheless ultimately sinks exhausted.

In *myelitis*, and in injuries of the spine from wounds and contusions, some differences in the symptoms have been observed, according to the seat of the injury. The disorganization of the substance of the cord entails a condition of paralysis more or less extensive, according to the seat and the extent of the inflammation. Every part of the body which receives its nerves from the spinal cord below the upper level of the structural disorganization is paralyzed ; consequently, when destructive *myelitis* extends throughout the cord to the *fifth* pair of cervical nerves, the upper extremities are paralyzed, and all those parts which receive their nerve-power from a lower level of the cord are paralyzed too. If, again, the spinal cord be lacerated or divided above the origin of the phrenic nerves, or above the third cervical vertebra, death is the immediate consequence, the nervous influence being no longer transmitted with sufficient completeness to the diaphragm and other muscles of respiration. Petit gives two remarkable instances of this. The only son of a working man went into the shop of a neighbor, who in play raised the child from the ground by putting one hand under his chin and the other at the back of his head. The child, only six or seven years old, struggled, dislocated his head, and died immediately. There are a few cases, however, in which disease of these parts has not been immediately fatal. Thus, the *odontoid process* has been destroyed by caries, or the second cervical vertebra has been dislocated, and yet the patient has continued to live for some months, or even some years. A remarkable case of a diminished area of the occipital foramen, whence resulted great pressure on the cord, is related by Mr. Holberton in *The Medico-Chirurgical Transactions*, vol. xxiv, p. 180. The patient lived more than two years, the most remarkable symptom being an extremely slow pulse. In these chronic cases the formation of the disease is slow, so that the cord becomes accustomed to the gradually increasing pressure, and the respiration consequently still continues to be carried on principally, though feebly, by the muscles of the neck and shoulders, the diaphragm and intercostal muscles being more or less palsied.

When the injury, however, is below the origin of the phrenic nerves, or at the level of the fifth and sixth cervical vertebræ, the inspiration is free, but the expiration is laborious, for the intercostal and abdominal muscles are paralyzed, and incapable of assisting in that process. The patient can yawn, for that is an act accompanied by inspiration ; but he cannot sneeze, for that is an act accompanied by expiration. At this point, also, the upper extremities are still

palsied, both as relates to motion and to sensation. When the palsy of motion and of sensation is complete, the patient, during the short remaining period of his life, presents the extraordinary phenomenon of a living head, with its sensibility and muscular powers unimpaired, attached to a trunk and extremities of whose existence he is only conscious by the sense of sight (BRÖDIE).

The circulation of the blood is affected, and the action of the iris of both eyes, through the medium of the sympathetic nerves (MERYON). Another very common symptom connected with injuries of the upper portion of the cord is *priapism*, which may show itself about the second or third day after the accident, and generally subsides after the first fortnight. It sometimes occurs even when all sensation in the part itself is destroyed, so that the patient is not sensible of the introduction of the catheter.

If the injury be in the situation of the sixth and seventh cervical vertebræ, the palsy of motion and of sensation of the upper extremities is frequently imperfect, while it is complete in the trunk and lower extremities.

When the spinal cord has been injured in the part corresponding to the first dorsal vertebra, the upper extremities may still suffer from an incomplete palsy either of motion or of sensation, or both. When, however, the seat of the lesion is in a line with the second dorsal vertebra, the sensation and motion of the upper extremities remain unimpaired, but the respiration is still difficult, from the palsy of the intercostal and abdominal muscles. If the paralyzing influence do not extend through the entire thickness of the cord, then the lower extremities may preserve their sensation and motion, although the arms hang powerless, owing to the disease having dissected out, as it were, the groups of ganglionic cells which determine the action of certain sets of muscles, whilst the conductors of the Will for the movements of the legs pass by unscathed (MERYON). An illustrative case, occurring in the practice of M. Broussais, is recorded by Ollivier. The patient was a medical student, aged twenty-one, who had, as a result of acute myelitis, complete paralysis of the upper limbs, while the legs, as well as the bladder and rectum, retained their healthy power. He died on the eighth day after the attack. There was some increased vascularity of parts of the encephalon, considerable congestion of the sinuses of the cord with fluid blood, and much sanguineous effusion between the dura mater and the vertebral arches opposite the brachial enlargement of the cord, as well as a considerable quantity of red serum between the pia mater and arachnoid at the lower part. Four minute cartilaginous laminæ were found about the centre of the dorso-lumbar enlargement; and the opposite surfaces of the arachnoid were adherent at several points over the brachial enlargement, while part of the cord, especially the gray substance, was remarkably soft for about two inches. The remaining part of the cord below was somewhat softened.

When the disease occurs in the dorsal region between the two enlargements of the cord, the respiratory muscles, which are under the influence of the dorsal spinal nerves, obeying the laws of irrita-

bility, are frequently agitated by violent spasms, and the breathing is accomplished by short and painful efforts. If the disease extend to either enlargement, the arms or legs may participate in the spasmodic movements. But, as the work of disorganization goes on, anæsthesia of the surface and paralysis of the muscles, above alluded to, follow in the train of symptoms;—abdominal respiration, disturbed circulation, embarrassed digestion, difficult defecation, inefficient micturition, and all the consequences of these respective functional disturbances, ensue.

The symptoms, when the injury is in the lumbar region, are not dissimilar to those of the dorsal region, except that the respiration is unaffected. When the lumbar region is the seat of the disease, the sound introduced into the bladder is more frequently covered with incrustations, and the patient also more commonly suffers from ulceration of the nates; but these symptoms, perhaps, result only in consequence of the patient surviving for a longer period than when the superior portions of the cord are affected. When the *myelitis* is limited to the lumbar enlargement of the spinal marrow, the convulsive movements occur at an early period of the disease, and cease *pari passu* with the disorganization of the cord. For a time the electro-muscular contractility is retained; but eventually it is almost always lost. The urine generally becomes alkaline, from retention by spasmodic contraction of the sphincters of the rectum and bladder; and *priapism* not unfrequently results as a reflex action from a distended bladder—a state which soon gives place to a negative condition, ushered in by reflex spasms of the legs during defecation and micturition (MERYON, *l. c.*, p. 35).

In chronic affections of the cord the palsied limbs usually waste, and become atrophied.

In cases in which a limb has suffered from palsy, both of sensation and of motion, some singular phenomena of reflex action still remain. When a stimulus has been applied to the palsied limb, it occasions involuntary contraction of the muscles of that limb. Thus, when a feather is passed lightly over the hollow of the foot, as in tickling, convulsions occur in the limb, although the patient is quite unconscious that anything is touching his foot. These movements are quite independent of Volition, and vary in extent and force inversely with the degree of voluntary power possessed by the affected limb, being most forcible when the loss of voluntary power is most complete, and diminishes gradually in extent and force as that power is increased. In some instances, by irritating one leg, movements were caused not only in that leg, but also in the other leg; and similar phenomena have been observed by Sir G. Blane and others in decapitated animals, showing that (consistent with the anatomical observations already referred to) a portion of the cord may furnish a supply of nervous energy after disease has interrupted its connection with the brain.

In all cases where the lesion of the cord is of such a nature as to intercept the transmission of the influence of the Will from the brain, convulsive movements are apt to occur in the legs, and to continue for a long time even after the arms have become com-

pletely paralyzed. These phenomena, says Dr. Meryon, are doubtless owing to the excitement of disease reflected from the spinal marrow to the motor nerves of the lower extremities. Similar involuntary movements may be produced artificially by tickling the soles of the feet, whose nervous connection with the brain is cut off by the destruction of a portion of the cord. The influence of the stimulus is transmitted to the spine by the incident nerves, and is reflected back by the motor nerves, thereby producing spasmodic contractions of the limb (Budd in *Med.-Chir. Trans.*, vol. xxii).

**Diagnosis.**—Diseases of the spinal cord and diseases of the brain are often followed by nearly similar symptoms; and, consequently, the one may be confounded with the other. But the history of the case, whether it has or has not been preceded by a fit of apoplexy or of epilepsy, will often enable us to determine the particular seat of the disorder. The antecedents and concomitant circumstances of every case must be carefully inquired into, and judged of upon their own merits. Myelitis is distinguished from lumbago, psoas abscess, and hip disease, by the absence of pain, and by the existence of palsy.

The characteristic symptoms of paralysis, as induced by destructive myelitis, are as follow (MERYON, *l. c.*, p. 35): (1.) Pain over that portion of the back which corresponds to the seat of inflammation. (2.) Lesions of sensation, giving rise to feelings of formication, creeping, prickling, tingling, heat, or cold, to numbness or complete anæsthesia. (3.) A gradual and progressive diminution of muscular power, distinguishing it from the paralysis which the French have denominated "*ataxie locomotrice progressive*." (4.) An equable degree of paralysis in all the muscles which are implicated; for as in health the nerve-force is distributed to whole groups of muscles in an equal degree, so likewise is it annulled when the nervous centre is disorganized. (5.) Convulsive and reflex movements of the paralyzed muscles. (6.) Spasm or paralysis of the rectum and bladder. (7.) Alkaline urine. And, finally, (8.) The loss of electro-muscular contractility.

**Prognosis.**—There seems no reason to doubt that, as many perfectly recover from superficial inflammatory lesions of the brain after fever, so also many slight inflammatory affections of the substance of the cord may subside, and the patient do well. Many cases, indeed, even when the bladder is slightly affected, recover. If, however, the disease be of more than a few weeks' continuance, the prognosis is always grave. Still, some few cases recover, the palsied limb becoming withered. But more commonly the disease runs on, and the patient at length dies after a long illness. When paralysis has once supervened, there is great reason to fear that the inflamed portion of the cord has passed into a state of disorganization, and that the disease is incurable; but the prognosis also eventually depends in some degree on the precise seat of the lesion. If it be in the cervical region, the immediate danger is greater than when the lesion is in the dorsal region; in this latter, again, the prognosis is more unfavorable than when lower portions of the spinal marrow are affected; and when the patient retains the command

over the motions of the rectum and bladder, and the acid character of the urine is persistent, the case is still more hopeful (MERYON).

**Causes.**—The more common causes of disease of this portion of the nervous system are accidental violence, as blows or falls. Affections of the cord, however, sometimes occur idiopathically, and the constitutional causes producing it are exceedingly undetermined. They have been referred to a suppression of the menses in the female, and to the suppression of a hemorrhoidal flux in the male, while others attribute them to sitting in damp or wet clothes, to onanism, or to venereal excesses, and prolonged exertion in the erect posture without active movement, inordinate muscular exertion, the action of cold, and the development of tubercle.

No age is exempt from *myelitis*, but it occurs more frequently from ten years old and upwards. It is most common, however, in adult age, and more frequently attacks the male than the female sex.

**Treatment.**—In classing ramollissement of the cord with inflammation, it might appear necessarily to infer that the treatment should be strictly antiphlogistic. It is questionable, however, whether this mode of treatment is advantageous; and it may be laid down as a general rule that bleeding ought not to be had recourse to after palsy has occurred. It is then plainly improper; for, the nervous influence being intercepted, the powers of the lower part of the body are so reduced that gangrene will rapidly supervene—a tendency which loss of blood greatly increases. Previous to that symptom it may be admissible; and it may be stated that so long as the affected muscles are convulsed, rigid, and irritable, the use of antiphlogistics and counter-irritants is indicated; but when the means calculated to subdue excitation have failed to arrest the further progress of the disease, and paralysis supervenes, stimulants are the only remedies which have the power of restoring to functional activity those nerve-cells and conducting fibres which are not irretrievably destroyed (MERYON). The chances of saving the patient by other antiphlogistic remedies mainly rest on acting on the alimentary canal so as to produce three or four motions in the twenty-four hours, and thus create such a derivation as in some degree to relieve the parts. The greater number of patients that recover are restored by these means. The particular purgative is not perhaps important; but as the neutral salts act not only on the intestines, but also on the bladder, that class of remedies is generally preferred. At the same time that the bowels are kept free, the patient should be allowed a liberal supply of wine, from six to eight ounces daily, and should have animal food at least once a day.

With respect to local counter-irritants, as blisters, moxas, or setons, little favorable can be said, unless they are employed previous to paralysis, as the tendency to gangrene renders their application of doubtful utility. When had recourse to, however, it will be found better to apply them above the seat of the disease than immediately over it, the greater vitality of the superior parts giving more assurance of the disposition of the wounds to heal. Of all stimulant remedies, *electricity* and *strychnine* are the most potent and the best; and *secale cornutum* has been recommended as a



remedy possessing the same power as *strychnine* (BARBIER, PAYEN, MERYON). When there is no great pressure beyond that which simple congestion produces, nor actual disorganization of the spinal cord, the remedial power of *secale cornutum* is said to be very great. It seems especially to resuscitate the muscular contractility of the rectum and bladder, and pelvic viscera generally (GUERSANT, TROUSSEAU, BROWN-SÉQUARD, MERYON). The *ergot of rye* is best given in the form of *ethereal tincture*, in doses of *from ten to twenty drops twice or three times a day*. It does not relieve the reflex convulsions, which are sometimes alleviated by *prussic acid*, *digitalis*, or *belladonna* (MERYON, *l. c.*, p. 40), *chlorodyne* or *chloro-morphine*.

After the local pain in the back has been subdued by the regular and repeated application of two or three leeches to the painful part, followed by a large warm poultice over the whole length of the spine, and a belladonna plaster of equal length to follow it, or an occasional blister on each side of the spine, together with mild, warm purgatives, if necessary, Dr. Meryon has found no remedy so effectual as *strychnia*, in the dose of *one-twentieth of a grain*, repeated *more or less frequently (twice or three times a day)* according to the evidence of its action. It may be combined advantageously with *ipecacuanha* in cases where the intestinal mucus seems deficient.

The absence of pain and of spasmodic muscular contraction necessitates great caution in determining the precise moment when the spinal cord is likely to be benefited by the energetic excitement of *strychnia*. The internal administration of this remedy ought, therefore, to be always preceded by its external use, together with other stimulants in the form of embrocations over the spine, when the stage of excitation has been subdued.

*Electricity*, after the activity of inflammation has been subdued, is a therapeutic agent of great value; and the continuous current of galvanic electricity seems to be just as efficacious as the induction or intermittent current. But whether galvanism or electromagnetism be employed, no high degree of tension is required for the restoration of muscular power; on the contrary, Dr. Meryon justly believes that the favorable course of many a case has been retarded by the employment of strong currents. Dr. Althaus, also, is in favor of weak currents.

In cases with a history of syphilis, and where there may be some reason to believe that hardening or induration of the cord or its membranes has taken place, the *iodide of potassium* may relieve the early phenomena, and by the aid of *setons*, for reasons already given (vol. i, p. 701), the progress of the disease may be held in abeyance so long as the discharge is maintained from the *seton*.

When disorganization of the spinal cord has become an accomplished fact, the disease is incurable; but yet the exigencies of the patient, as Dr. Meryon justly observes, are not the less pressing on the careful attention of the physician, and in nothing more so than in the protection which is called for against bed-sores, which will sometimes occur in spite of the greatest care (*op. cit.*, p. 40).

## PARALYSIS, OR PALSY.

LATIN Eq., *Paralysis*; FRENCH Eq., *Paralysie*; GERMAN Eq., *Paralyse*—Syn., *Lähmung*; ITALIAN Eq., *Paralisi*.

**Definition.**—*Palsy or paralysis are terms commonly restricted to affections where motion is lost, while the term anæsthesia implies a palsy of the nerves of sensation. Such various forms of palsy, or paralysis, are rather symptoms of a lesion than specific diseases.*

**Pathology.**—Palsy of a part is a very constant symptom of structural disease of the brain or of the spinal cord, but it occasionally occurs from a diseased state of a nerve-trunk itself. Palsy may affect a whole limb, or merely a part of one, and it is also limited to the muscles of certain regions. Palsy of a finger, a hand, an arm, or a leg, is an example of the first; palsy of the facial muscles of expression, from disease connected with the *portio dura* of the seventh pair, or facial nerve, is an example of the second.

The interesting clinical lectures of the late Dr. R. B. Todd describe the following conditions as giving rise to paralysis of motion: (1.) Lesion of a nerve in some part of its course destroys its power of transmitting that force which is expressed by a contraction of the muscle into which the nerve is distributed. (2.) A lesion of some part of those central parts of the nervous system whence the nerve takes its origin, or with which it may be connected directly or indirectly. And, as a correlative statement, it may be written that whatever interferes materially with the conducting power of nerve-fibre, or the generating power of nerve-vesicle, will constitute a paralyzing lesion. Poisoning of the nervous matter will operate in this way. *Chloroform, ether, opium*, the poison of *lead* and of *mercury*, applied directly to the nerve-fibre of a living animal, suspends its power of transmitting the nervous force so long as the influence of the poison lasts. Poisons formed or retained in the living body operate in the same way, such as the retained *urinary* and *biliary* principles, as in *Bright's disease*, the poisons of *rheumatism, gout*, and probably also *syphilis* in some of its more severe tertiary effects. Whatever, in short, impairs the natural structure of the nerve-matter, such as *inflammation, atrophy, condensation, softening* (spinal, as in the form of *tabes dorsalis*), *solution of continuity*, either by simply cutting the trunk of a nerve, or by the deliquescence of the nerve-fibres, as a result of disease, such as *white softening, a sanguineous (spinal apoplexy)* or *serous effusion, pressure on a nerve or a nervous centre*, are causes which will produce more or less complete paralysis. Of this there is abundant proof—*e. g.*, the inclusion of a nerve in a ligature, compression of a nerve by a tumor, a depressed piece of bone in fracture of the skull, or an apoplectic clot on the exterior of the brain.

Four different conditions of the muscles are to be observed in cases of paralysis, namely: (1.) A condition little different from that of health, but less firm, less excitable by the galvanic stimulus, when the paralyzing lesion is not of an irritative kind. (2.)

Complete relaxation of the muscles ; characterized by softness, imperfect nourishment, and rapid wasting—so rapid that in a few days the size of the limb experiences a marked diminution. Such muscles scarcely, if at all, respond to the galvanic stimulus. (3.) Contraction of the muscles, with rigidity and wasting (the flexors being always more rigid than the extensors)—a condition which is due to a chronic shortening of the muscles themselves, and generally associated with a form of muscular atrophy. (4.) Nutrition not impaired, constant firmness and rigidity, incomplete paralysis, increased susceptibility to galvanic stimulus.

The practical inferences to be drawn from these conditions are of great value in treatment. Thus, early rigidity indicates local bleeding or counter-irritation, while complete relaxation is against anti-phlogistic treatment.

The different forms of paralysis of common occurrence are due—(1.) To disease of the brain or spinal cord, in which form the muscles may be rigid or relaxed, the disease of the brain being the result of *apoplexy*, *minute hemorrhages*, *softening*, *renal disease*, *induration*—the result of *syphilitic poison*,—the *epileptic* or *choreic* state; (2.) To pressure upon or injury to a nerve; (3.) To hysteria; (4.) To the influence of poisons, such as *lead*, *arsenic*, *mercury*, and some kinds of food-grains in a diseased state.

Typical forms of paralysis comprehend *Paralysis of the Insane*, or *General Paralysis*; *Local Paralysis*—e. g., *Facial Palsy* and *Scriver's Palsy*; *Hemiplegia*; *Paraplegia*; *Wasting Palsy*, or *Progressive Forms of Paralysis*; *Locomotor Ataxy*; *Paralysis from Blood-Poisons*; *Infantile Paralysis*; *Palsy from Lead Poisoning* or other poisons in food or drink, or specific disease, such as *Diphtheritic Paralysis*.

#### FACIAL PARALYSIS.

**Definition.**—*Paralysis of the muscles of the face, usually confined to one side, but sometimes, though rarely, affecting both sides, and apt to be followed by contracture or tonic spasm of the muscles previously paralyzed* (“*Spasmodic Tic*” of Marshall Hall.)

**Pathology.**—In previous editions of this text-book erroneous doctrines were stated regarding facial paralysis, in accepting and perpetuating the views of Dr. Todd, that “*the fifth nerve is more or less involved in the paralyzing lesion*,” and in mixing up the phenomena of facial paralysis, depending on central or cerebral lesion, with facial paralysis due simply to lesion of the trunk or branches of the *portio dura*. I am indebted to my friend Dr. Sanders, of Edinburgh, for kindly directing my attention to this error, and for references to his important paper in the *Lancet* of Oct. 21st, 1865. From his papers on this subject, and the clinical lectures of the late Professor Trousseau on facial paralysis, the following account is given of this affection:

Facial paralysis is due to one of two causes, namely,—(1.) To a lesion simply of the trunk or branches of the *portio dura* nerve at some part of its course, and independent of cerebral disease. This form of facial paralysis is sometimes also known as *Bell's paralysis*,

or *peripheral facial hemiplegia*. (2.) Facial paralysis may be due to a cerebral lesion, in which case it is usually accompanied by hemiplegia of the limbs on the same side. This form of facial paralysis is known as *cerebral* or *centric facial hemiplegia*. (3.) A third form of facial paralysis is ascribed to reflex paralysis of the seventh, consequent on paralysis or lesion of the fifth pair. The first affection is that most frequently met with in practice; and although it is not a dangerous form of paralysis, it is one from which recovery is very slow, and in which prognosis, as to complete recovery of symmetry of the face, is uncertain. The second form is the more rare, and is always a very grave disease, and not usually recovered from. Both kinds of facial paralysis present many points of resemblance and of contrast, of great interest and importance in diagnosis; and the occurrence of paralysis from one of the causes mentioned by no means excludes the other, and therefore cases of both may exhibit extremely complex symptoms.

The phenomena of facial paralysis are confined to the "muscles of expression," including the *buccinator*, and do not involve the masticatory muscles (*masseter*, *temporal*, *pterygoid*) supplied by the fifth pair of nerves.

A description of the phenomena of *facial paralysis* must therefore have due regard to the course and distribution of the *seventh nerve*. The facial nerve, or *portio dura* of the *seventh pair*, is the motor nerve of the face. It emerges from the lateral column of the spinal cord, as that column passes under the *pons Varolii*, and enters the *internal auditory meatus*. At the bottom of this *meatus* it enters the *aqueduct of Fallopius*, and follows the windings of that canal to the lower surface of the skull—namely, to the *stylo-mastoid foramen*—by which it leaves the osseous canal. It is then continued forwards through the substance of the parotid gland, and separates in the gland behind the ramus of the lower maxilla, into two primary divisions—the *temporo-facial* and the *cervico-facial*—from which numerous branches spread out over the side of the head, the face, and upper part of the neck, forming what is known as the "*pes anserinus*." Within the temporal bone it is connected with the auditory nerve by filaments of union, and where it swells into its gangliform enlargement it is joined by the large superficial petrosal branch from the Vidian nerve, and also by the small superficial petrosal nerve. Close to the *stylo-mastoid foramen* it gives off several small branches—namely, the posterior auricular, a branch from the *digastric muscle* and *stylo-hyoid*, and a twig to the *stylo-glossus*. In front of the mastoid process it divides into an *auricular* and an *occipital* portion, and is connected with the *great auricular nerve* of the cervical plexus. It also gives off branches to the *digastric* and *stylo-hyoid* muscles, joining the *glosso-pharyngeal* near the base of the skull, and the plexus of the *sympathetic nerve* on the external carotid artery. The main trunk of the facial then separates into two primary divisions, the larger of the two being the *temporo-facial*, which is directed forwards through the parotid gland. Its ramifications and connections with other nerves form a network over the side of the face, extending as high as the temple and as low as the

mouth. Its branches are arranged in *temporal*, *malar*, and *infra-orbital* sets.\* The smaller of the two primary divisions—the *cervico-facial*—is directed obliquely through the parotid gland towards the angle of the lower jaw, and gives branches to the face, below those of the *temporo-facial* division, and to the upper part of the neck. Its branches are named the *buccal*, *supra-maxillary*, and *infra-maxillary*. In the gland this division of the facial is joined by filaments of the *great auricular nerve* of the cervical plexus, and offsets from it penetrate the substance of the gland.†

As the course, distribution, and connection of this nerve are most important to be remembered in acquiring a clear understanding of the phenomena of its paralysis, whether due to cerebral or peripheral causes, they are thus minutely given from the best text-book of anatomy (Quain's, the edition edited by Drs. Sharpey, Thomson, and Clelland).

One very important point to remember is that the *portio dura* of the seventh pair is the only motor nerve of the *buccinator muscle* for all its actions, whether of expression or of mastication; and that the fifth pair supplies it, not with motor, but with sensory fibres (MAYO, VOLKMANN, LONGET). *The buccinator muscle is inter-*

\* (a.) "The *temporal branches* ascend over the zygoma to the side of the head. Some end in the anterior muscle of the auricle and the integument of the temple, and communicate with the temporal branch of the upper maxillary nerve near the ear, as well as with (according to Meckel) the auriculo-temporal branch of the lower maxillary nerve. Other branches enter the *occipito-frontalis*, the *orbicularis palpebrarum*, and the *corrugator supercilii* muscles, and join offsets from the *supra-orbital* branch of the ophthalmic nerve."

(b.) The *malar branches* cross the malar bone to reach the outer side of the orbit, and supply the orbicular muscle. Some filaments are distributed to both the upper and lower eyelids: those in the upper eyelid join filaments from the lachrymal and supra-orbital nerves; and those in the lower lid are connected with filaments from the upper maxillary nerve. Filaments from this part of the facial nerve communicate with the malar branch of the upper maxillary nerve.

(c.) The *infra-orbital branches*, of larger size than the other branches, are almost horizontal in direction, and are distributed between the orbit and mouth. They supply the *buccinator* and *orbicularis oris* muscles, the *elevators of the upper lip and angle of the mouth*, and likewise the integument. Numerous communications take place with the *fifth nerve*. Beneath the *elevator of the upper lip* these nerves are united in a plexus with the branches of the upper maxillary nerve; on the side of the nerve they communicate with the nasal, and at the inner angle of the orbit with the *infra-trochlear nerve*. The lower branches of this set are connected with those of the *cervico-facial* division. Near its commencement the *temporo-facial* division of the facial is connected with the *auriculo-temporal nerve* of the fifth, by one or two branches of considerable size which turn round the external carotid artery; and it gives some filaments to the *tragus* of the outer ear (Quain's *Anatomy*, edited by Drs. Sharpey, Thomson, and Clelland, p. 618).

† (a.) The *buccal branches* are directed across the masseter muscle to the angle of the mouth; supplying the muscles (buccinators), they communicate with the *temporo-facial* division, and on the buccinator muscle join with the filaments of the *buccal branch* of the lower maxillary nerve.

(b.) The *supra-maxillary branch*, sometimes double, gives an offset over the side of the maxilla to the angle of the mouth, and is then directed inwards, beneath the depression of the angle of the mouth, to the muscles and integument between the lip and chin: it joins with the labial branch of the lower dental nerve.

(c.) The *infra-maxillary branches* perforate the deep cervical fascia, and, placed beneath the platysma muscle, form arches across the side of the neck as low as the hyoid bone. Some branches join the superficial cervical nerve beneath the platysma, others enter that muscle, and a few perforate it, to end in the integument (Quain's *Anatomy*, by Drs. Sharpey, Thomson, and Clelland, p. 615).



rupted in all its functions, whether of expression or of mastication, whenever the *portio dura* is paralyzed; it is unaffected, and all its actions are preserved, in motor paralysis of the FIFTH PAIR (Sanders, *Lancet*, October 28, 1865, p. 478.)

The threefold functions of the *portio dura* must also be recognized. Considered as a musculo-motor nerve, it contains within its common trunk the following sets of fibres serving different functions: (1.) *Voluntary motor fibres*, by which the voluntary movements of the features are performed, and by which especially labial and buccal speech and mastication are accomplished; (2.) *Emotional fibres*, by which the features express the passions more or less involuntarily; (3.) *Reflex motor fibres*, which are involuntary, for the act of winking and for the movements of the nostrils in the acts of respiration. These different sets of fibres are believed to derive their peculiar functions solely from the nature of their origin and place of central connection in the brain or *medulla oblongata*. In lesion, therefore, of the nerve trunk, in which all the fibres indiscriminately are equally liable to be affected, not only voluntary but emotional and also reflex motions will be suspended. But when the cause of the paralysis is cerebral, the origin or course of certain sets of fibres may alone be involved by the lesion, while others may entirely escape injury. The symptoms, therefore, of central paralysis will vary with the special seat of the central lesion. The voluntary and emotional actions (either or both), which have their origin in the cerebrum, will usually suffer; while the fibres for reflex action, which have their source in the *medulla oblongata*, may be expected to retain their power. The play of the features will be lost, and buccal and labial speech and mastication (so far as the buccinator muscle is concerned) impaired; but the natural position of semi-closure, and involuntary winking of the eyelids, will be preserved.

Looking, therefore, at the circuitous windings of the *portio dura*, its intricate distribution, and the various textures through which it passes, it can readily be understood how varied are its tendencies to be involved in disease, and even traumatic injury. Tumors, hemorrhages, or other lesions, may involve the nerve within the cranium. While it traverses the circuitous windings of the *aqueduct of Fallopius* it may be injured, and pressed upon by the results of necrosis or caries of the bone, or of suppuration or lesions of the fibrous sheath or periosteum; while lesions or tumors involving the parotid gland, may injure the nerve and paralyze the parts it supplies.

Three forms of paralysis of the facial nerves ought to be discriminated in considering the diagnosis of central facial hemiplegia, namely, *Voluntary Motor Paralysis*, *Emotional Paralysis*, and *Reflex Paralysis* (Sanders, *Lancet*, October 28, 1865, p. 479).

**Symptoms and Causes.**—The accession of facial paralysis (when peripheral) is usually sudden, and is generally discovered by the patient when he begins to eat. He feels something peculiar in the act of chewing, and has some difficulty in mastication. When the food gets between the paralyzed cheek and the teeth, the cheek is instinctively squeezed or pressed upon by the hand, in order to push

the food between the teeth again. The difficulty of mastication only concerns the buccinator muscle, and not the other muscles of mastication. There is no pain. The patient is soon and often abruptly told, by the first kind friend who happens to meet him, that his mouth is awry, and that it becomes considerably more so when he laughs. He then naturally wishes to see all this for himself; and on looking at his face in the looking-glass, he may verify the observation of his friend, and is then generally greatly frightened and alarmed by the discovery.

When the face remains at rest the paralyzed side looks slightly flatter, and more flaccid and pendulous, than the sound side. The eye of the paralyzed side is also more widely open than the eye on the sound side. When speaking, and still more when laughing is attempted, the angle of the mouth on the paralyzed side remains perfectly motionless, but on the sound side it is immediately drawn upwards and outwards. The eyelids, the cheek, and half the lip of the paralyzed side remaining thus motionless when efforts are made to contract the muscles, a singular and characteristic expression is given to the face. The eyelids remaining motionless on the paralyzed side, the patient is unable to shut the eye, but the globe of the eye itself moves perfectly in any direction at will, which shows that the motor muscles of the eye are not affected, and that the paralysis affects exclusively the *orbicularis palpebrarum* muscle, and does not involve the *levator palpebræ superioris*. Sight is unimpaired. The tongue is protruded with ease and regularity; lingual articulation is sufficient; but articulation of certain words is difficult, on account of the paralysis of the cheek. Sometimes the arch formed by the pillars of the fauces is larger on the paralyzed side than on the sound one, by the uvula inclining to the sound side. Cutaneous sensibility is unimpaired; and the patient may be in the best of health, the ailment being purely local—namely, paralysis of the facial muscles of expression on one side. There is an absence of all electric excitability of the paralyzed muscles supplied by the seventh pair.

One of the most common causes of *facial paralysis* is exposure to cold, especially to cold when sleeping in a draught or in the open air, or exposure to cold after being in a state of perspiration, or sitting in a railway or other carriage with the side of the face exposed to the wind. The influence of *rheumatism* is also conspicuous in many cases, the patient being seized as suddenly as in *lumbago*, acknowledging a similar cause; residence in a damp place, or other exposure to cold and wet, being sufficient to bring on the attack. The influence of mental emotion—a sudden fright or start—has been known to induce the paralysis. Of traumatic lesion the facial paralysis of new-born infants is not uncommon. It is generally due to the compression of the trunk of the facial nerves by the use of the forceps. If such compression has been severe or excessive, the injury may be permanent; otherwise, it is soon recovered from. Wounds of the *portio dura*, or fractures of the skull, involving lesion to the *aqueduct of Fallopius*, may also be followed by paral-

ysis, and in all these instances the paralysis is sudden. When the facial nerve, during some part of its course, is interrupted in its functions by the secondary influence of advancing organic disease, the nerve becomes compressed gradually, and ultimately altered. In such cases the paralysis comes on slowly. In severe chronic inflammation of the internal ear, with the destruction of the tympanum and ossicula, the petrous portion of the temporal bone becoming carious, facial paralysis is apt to supervene, and the lesions may be fatal.

The form of paralysis due to such causes is that known as *peripheral facial hemiplegia*; but the paralysis may acknowledge a *cerebral lesion* as its cause, such as cerebral hemorrhages, lesions of the *pons Varolii*, implicating the fourth ventricle. In such cases the facial palsy is usually partial, affecting more especially the muscles of the mouth and cheek, leaving the closing movements of the eyelids unaffected. As a rule, also, the paralysis due to a cerebral lesion gives rise to a hemiplegia involving parts beyond the face, such as one or more limbs of the affected side. But there are also instances in which the paralysis due to a cerebral lesion (small cerebral hemorrhages) has been exclusively limited to the face (GRAVES, DUPLAY). This is in keeping with other forms of hemiplegia (cerebral) in which the paralysis is localized; as when cerebral hemorrhages or apoplectic-like seizures paralyze the tongue only, or an arm, or distort the features more or less, but generally combined with a hesitation in the movements of the leg, and of which the patient is unconscious (TROUSSEAU). The previous existence of peripheral paralysis in such cases may render superadded paralysis from a cerebral lesion very difficult to diagnose. When the cerebral lesion is limited to the *pons Varolii*, to slight injury to the fourth ventricle, the paralysis of the face which results has many of the characters of the peripheral or Bell's paralysis, even to the absence of electric insensibility of the paralyzed muscles (VULPIAN, quoted by TROUSSEAU, who in the course of a very long practice never saw a case of the kind himself). Trousseau considers the most distinguishing characteristic of paralysis due to a cerebral lesion to be in the *absence* of complete paralysis of the *orbicularis palpebrarum*. "However complete hemiplegia of cerebral origin may be, I have never seen *complete paralysis of the orbicularis palpebrarum*—the eye can always be closed;" while in Bell's paralysis, the palsy of the *orbicularis palpebrarum* is never absent, and the eye cannot be completely closed (TROUSSEAU). This coincides with the experience of Dr. Sanders. There is, however, a slight modification to be acknowledged here, namely, that "although the patient with cerebral hemiplegia can close both eyes simultaneously, he cannot voluntarily close the eye on the same side as the paralysis while the other remains open" (BAZIRE). But it appears to me that this would only show *some extent* of paralysis if the patient were known to have been able to close either eye at will previous to his attack—a feat which many are unable to perform who are free from any paralysis. From the threefold functions of the *portio dura* already

noticed, and the diverse origins of its several sets of fibres,\* it will easily be understood how, in very limited and localized cerebral lesions, *all* the branches of the facial nerve may not be affected. The extent of apparent paralysis in cases of cerebral facial hemiplegia is not usually so great, and therefore not so alarming to the patient, as in cases of Bell's paralysis, where the trunk of the nerve only is affected. But the prognosis must be more unfavorable. In ordinary facial paralysis of *cerebral origin*, Dr. Sanders has always found that the muscles of expression, including the buccinator, were more or less affected, while the action of the masseters, temporals, and pterygoids was unimpaired. The usual facial distortion was exhibited to a greater or less degree according to the amount of the paralysis, but it rarely approached the completeness usually seen in peripheral paralysis from lesion of the nerve trunk. With the exception of the slightness of implication of the *orbicularis palpebrarum* muscle, the phenomena of cerebral hemiplegia of the face are entirely similar to those of facial paralysis produced by lesion of the seventh nerve itself, and, as in the latter, are confined to the muscles of expression, including the buccinator, and do not involve the masticatory muscles (masseter, temporal, pterygoid) supplied by the *motor fifth*.

Another point of diagnosis is to be recognized in the behavior of the paralyzed muscles under electric irritation. In facial paralysis of cerebral origin, the muscles respond *normally* to electric irritation; but if the paralysis is due to a lesion of the *portio dura*, their contractility is not at all, or scarcely at all, roused by an electric current (DUCHENNE, SANDERS, VULPIAN, TROUSSEAU). *The symptoms of cerebral facial paralysis* vary as the central lesion is more or less distant from the origin of the seventh pair. There is therefore much more variety in the expression of the face (according to the varying extent of the paralysis) than in cases of Bell's paralysis. The peculiar aspect of the face in *cerebral facial hemiplegia* is due to the want of symmetry between the two halves. The contraction of the muscles on each side is not co-ordinate. The sound cheek is wrinkled and shortened. The labial commissure on the sound side is drawn outwards and upwards, and is on a higher level than on the opposite side; the angle of the mouth on the paralyzed side is lower than its fellow; and if the paralysis is extreme, the commissure on the paralyzed side remains partially open, so that the saliva constantly escapes. The flaccid cheek is the result of paralysis of the buccinator.

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\* The seventh pair of nerves appear on each side, at the posterior margin of the pons, between the middle and inferior peduncles of the cerebellum, and nearly in a line with the place of attachment of the fifth nerve.

The *portio dura*, or facial nerve, placed a little nearer to the middle line than the *portio mollis*, may be traced to the *medulla oblongata*, between the *restiform* and *olivary fasciculi*, with both of which it is said to be connected. Some of its fibres are derived from the pons. Phillipeaux and Vulpian affirm that the fibres arise from the outer wall of the fourth ventricle, and that many of them decussate in its floor. Connected with the *portio dura*, and intermediate between it and the *portio mollis*, is a smaller white funiculus. The roots of this accessory or intermediate portion are connected deeply with the lateral column of the cord (Quain's *Anatomy*, by Drs. Sharpey, Thomson, and Clelland, p. 587).



The nostril on the paralyzed side is more closed than on the sound side; but "in the great majority of cases of cerebral hemiplegia of the face, the orbicularis palpebraris is not materially affected; the act of winking and of voluntary closure of the eyes continues on the paralyzed as on the sound side, with the small exception that the voluntary closure is usually weaker on the palsied side.

These phenomena furnish the best diagnostic marks between *centric* and *peripheral* paralysis of the face; the hanging cheek, with wide open, staring, unwinking eye, denotes lesion of the *portio dura*; the flaccid face, with the natural position and motion of the eyelids, is the sign of cerebral lesion, and indicates a more serious disease" (RECAMIER, TODD, SANDERS).

There is difficulty in the articulation of labial consonants and vowels. The tongue protrudes in the normal direction, and if its point seems to diverge from the median line to the paralyzed side, it only seems to do so in consequence of the commissure of the lip being drawn in the opposite direction—namely, away from the paralyzed side. But sometimes the tongue, too, is paralyzed and really deviates, and then it is drawn to the sound side, when the branches of the *portio dura*, going to the *stylo-glossus* and the *genio glossus*, are implicated. Opinions differ greatly as to the implication of the tongue, the uvula, and the soft palate in cases of cerebral facial hemiplegia. Trousseau, Todd, Hasse, Longet, Romberg, Bidder, and Sanders, all refer to such cases. Dr. Sanders shows that the position of the uvula varies frequently, both in the natural and hemiplegic palate. The only reliable sign that the palate is involved in the lesion of the *portio dura* is the existence of a vertical relaxation, or lowering of the (paralyzed) corresponding half of the *velum palati*, with diminished height and curvature of the posterior palatine arch on the paralyzed side (SANDERS). Such cases indicate lesion before the geniculate ganglion in the Fallopian duct, and implicating the petrosal nerves. In both forms of facial paralysis the action of spitting is difficult, so also is whistling, as the patient cannot close the mouth nor inflate the cheek. Mastication is badly done, but only so far as the buccinator is concerned, because the food accumulates between the paralyzed cheek and the teeth. The hand is often seen to be held almost instinctively against the cheek to give it support, and to press the food to between the teeth for proper mastication, which is efficiently performed by the muscles of mastication (*temporals, masseters, pterygoids*).

In some cases of facial paralysis it has been noticed that hearing on the affected side becomes much more acute; and the taste is sometimes perverted, both indicating lesion high up within the bony canal traversed by the *portio dura* (TROUSSEAU, ROMBERG, BROWN-SEQUARD, LANDOUZY, BAZIRE).

A peculiar and characteristic result of facial paralysis is the contracture and tonic spasms, or convulsions, of the muscles of the face which have been paralyzed—the "*spasmodic tic*" of Marshall Hall. The simple contraction of the muscles is very common after Bell's paralysis, when it has lasted a long time and been extreme. It comes on after the affection appears to be cured, and after the sym-



metry of the face has been apparently restored. At first, and most usually, the contraction is partial, and is mixed up with some degree of paralysis, the contraction of the muscles not being affected at will. This contraction is common to all muscles which have been paralyzed and out of use for some time. The spasms are sometimes painful, and the contracted muscular masses are painful on pressure. Sometimes one muscle and sometimes another is thus affected. Sometimes it is the orbicular muscle of the eyelid, when the eye, instead of being more open, as it was when paralyzed, now looks smaller, and is more closed. Such contraction is a most frequent termination of paralysis due to *rheumatism*. A rapid return of tonicity in a paralyzed muscle, the electro-muscular contractility of which had been completely abolished, is always a sign of approaching contraction (DUCHENNE).

**Diagnosis** of facial paralysis, differentially, is based on a knowledge of the circumstances under which the paralysis set in, the progress of its development, and the attending phenomena, bearing in mind the anatomical and physiological history of the *portio dura* already given. Sometimes the paralysis affects both sides; and one important test is given by Marshall Hall, by which it may be known whether the cause of double facial paralysis is seated in the brain or in the course of the nerves. If the lesion is central (cerebral) the conducting power of the nerve trunks is retained for an indefinite period, so that by galvanizing the trunk and the principal branches of the facial nerves, all the paralyzed muscles supplied by the nerves are thrown into contraction, as if the muscles themselves were being galvanized; whilst, when the paralyzing lesion is *in the course of the nerves*, the nerves very easily lose their conducting power. If reflex movements be seen in the paralyzed muscles, the cause of the paralysis is assuredly a lesion of the nerve centres.

**Prognosis.**—Bell's form of peripheral facial paralysis generally gets well in time; and all the more rapidly that it sets in suddenly, and the patient young. From four to ten months is the ordinary duration of the affection; but there are instances in which the paralysis yields in twenty-four, fifteen, or even twelve hours, but such cases are exceptional (TROUSSEAU). The prognosis in *central facial hemiplegia* is less hopeful; so also in *double facial hemiplegia*; because the cause indicates—from the symmetrical nature of the exciting lesion (generally periostitic)—some constitutional source as the origin of the mischief, *e. g.*, scrofulosis or syphilis.

**Treatment.**—Paralysis due to lesions which are destructive of the nerve are beyond medical aid. In cases suitable for treatment the agents used with most benefit are,—Bloodletting by leeches behind the ear of the affected side, or over the mastoid region. They ought to be applied repeatedly, especially if any pain exists on pressure in that region. Blisters, with dressings to keep them open, medicated by veratria or strychnia ( $\frac{1}{2}$  to  $\frac{1}{4}$  of a grain of *sulphate of strychnia* or of *veratria*), mixed with five or six times their weight of pounded sugar. Electrization in the form of Faradization by a Stöheres two-celled volta-electric machine; or in the form of an *interrupted galvanic current*, from a *continuous current*

battery (as from a Muirhead's battery of fifteen cells), the number of cells being very gradually increased.

In using Faradization each facial muscle should be separately galvanized, instead of attempting to pass the current through the facial nerve. The muscles are thus more powerfully influenced, and they are more apt to regain *voluntary* contractility, at different periods, some muscles before others. At first the apparatus should be one capable of producing currents with very rapid intermissions; but when any muscles begin to *contract* the intermissions should be few, and the sittings at long intervals; otherwise contraction of the muscles is apt to increase and become incurable.

The interrupted galvanic current, from a continuous current battery, induces contractions when the circuit is closed and when it is opened, and more powerfully at the moment of closure. It is said to restore to palsied muscles the power of contracting under the influence of Faradization. When improvement follows the use of the interrupted galvanic current from a continuous battery, the number of the cells should be gradually increased as the muscles get less susceptible to their influence. One of the electrodes should be gently moved along the surface of the muscle, just as the brush is used in Faradization.

When contracture of muscles supervenes, they ought to be stretched or pulled out mechanically; and to remedy contraction of the buccinator, Duchenne recommends the use of a small billiard ball, worn for a long time inside the cheek, to be replaced after a time by a larger one. The late Dr. Bazire gave favorable testimony to the value of this mechanical aid.

The daily action of the bowels ought to be carefully regulated by such medicines especially as keep the colon in functional activity. *Bichloride of mercury* (*corrosive sublimate*) in small doses, continued till the gums are slightly tender, is sometimes of service. In cases where rheumatism prevails or periostitic affections, small doses of *iodide of potassium*, persevered in for a lengthened period, are of service; with the frequent use of *phosphate of soda* as a daily morning aperient.

#### HEMIPLEGIA.

LATIN EQ., *Hemiplegia*; FRENCH EQ., *Hémiplégie*; GERMAN EQ., *Hemiplegie*—Syn., *Halbseitige Lähmung*; ITALIAN EQ., *Emiplegia*.

**Definition.**—*A form of paralysis affecting one lateral half of the body.*

**Pathology.**—It is that form of palsy to which the name of "*paralytic stroke*" is commonly applied. Either half of the body may be affected; and the parts which are actually involved are generally the upper and lower extremities, the muscles of mastication, and the muscles of the tongue on one side. The paralysis may be either complete or incomplete as regards motor power. Consciousness may or may not be perfectly retained; and whether it is so or not, the patient, when seized, falls to the ground, because the power of

maintaining his equilibrium is destroyed by the failure of the antagonizing muscles of one-half of the body. The affected arm and leg lie as if lifeless on the side, all power of motion in them being destroyed. Stimulation, however, of the extremities of the sentient nerves, by slight titillation with the fingers, sometimes gives rise to active movements. The combined effect of such stimulation and the resulting movements is to cause considerable pain. These excited motions, to which the name of "*reflex actions*" has been given, occur almost exclusively in the lower extremities. Other involuntary movements of the paralyzed limbs occur simultaneously with the action of yawning, or result from emotions of surprise, joy, pleasure, grief, laughter, crying.

When the *fifth* nerve is implicated in the hemiplegia, the proper masticatory movements are unequal on the two sides, in consequence of paralysis of the temporal, masseter, and pterygoid of the affected side. There is a want of force in the masseter muscles of the paralyzed side; and there is therefore apt to be lateral displacement or obliquity of the inferior maxilla, either when at rest or during mastication. The mesial line between the lower incisors is thus also apt not to correspond to that in the upper jaw.

Sometimes the *third* nerve may be paralyzed, in which case the upper eyelid drops, and there is inability to raise it, combined with outward squint and dilated pupil.

The protrusion of the tongue is also characteristic in hemiplegia. It is pushed out towards the side affected, and on being retracted it is drawn towards the healthy side. Imperfect articulation exists in *hemiplegia*, and results from the palsy of the *ninth* and *fifth* nerve; and where the power of speech is wholly lost, or utterance is limited to monosyllables, the sign is not favorable, but denotes, with other symptoms, extensive lesion of the brain, superficial as well as deep. When deglutition is impaired, serious and extensive lesion of the brain, connected with the *vagus* or *glosso-pharyngeal* nerve, is denoted.

The lesions which give rise to hemiplegia are of the following kinds: (1.) *Hemiplegia* typical of diseased brain depends on a softening clot, abscess, tumors, or exudation, involving or compressing some considerable portion of the centre of volition, such as the *corpus striatum* or *optic thalamus*, or in the immediate vicinity of those parts. Unless pressure be produced, or the fibres otherwise interfered with, paralysis does not result. The centre of volition "reaches from the *corpora striata* in the brain down the entire length of the anterior horns or gray matter of the spinal cord, and includes the *locus niger* in the *crus cerebri*, and much of the vesicular matter of the *mesocephalon* and of the *medulla oblongata*." Disease of any part of this range of structure is capable of producing paralysis; and the palsy is on the side of the body opposite the lesion. (2.) The intracranial portion of this range exercises the greatest and most extended influence in the production of voluntary movements, and the most extended and complete paralysis takes place from disease of the *intracranial* portion. (3.) In cases of cerebral disease it must be observed and remembered that the intracranial portion of the centre

of volition for the left side of the body is situated on the right side, and that for the right side is situated on the left side of the cranium, while the *intraspinal* portions maintain relatively their respective sides. These two portions are connected by the oblique fibres coming from the anterior pyramidal column of the *medulla oblongata*, which (crossing from right to left) decussate with similar fibres proceeding from left to right. (4.) Exudations which are the result of inflammatory or other diseased state of the membranes of the brain, which, as they increase and cause pressure on the surface, transmit the effects of pressure downwards to the *corpus striatum* and *optic thalamus*, and thus cause paralysis. (5.) Morbid states which affect or destroy fibres of deeper-seated parts, such as the *crura cerebri*, or of the *cerebellum* in its *crura* (because a connection exists between the hemispheres of the cerebellum and the fibres of the pyramids in the *pons Varolii*), cause paralysis. (6.) The slow accession of paralysis following symptoms of irritation indicates a gradual morbid change, such as from exudations slowly taking place. (7.) An important feature in paralysis is due to the condition of the muscles, as to whether they are rigid or relaxed. (8.) Rigidity, whether supervening or occurring simultaneously with the paralysis, indicates irritative disease within the cranium. (9.) In cases where the rigid condition of the muscles does not come on till after a long period of paralysis, and after the muscles are perhaps wasted from atrophy, such a condition indicates loss of substance in the brain, and that the cicatrix is undergoing contraction. (10.) *Hemiplegia* typical of spinal disease, where the palsy is on the same side of the body as the disease, is caused by a lesion involving a lateral half of the spinal cord *below* the decussation of the pyramids. (11.) In *hemiplegia* typical of epilepsy the lesion is transient, the palsy in general remaining only a few hours, or at most a few days, after the epileptic attack. It is termed *Epileptic Hemiplegia*. (12.) The *hemiplegia* associated with chorea occurs during acute attacks of that disease, and is termed *Choreic Hemiplegia*. (13.) The *hemiplegia* associated with hysteria is also of transient endurance. (14.) There is a form of *hemiplegia* where the morbid phenomena seem to spread from the periphery to the central parts.

In all these forms of hemiplegia the paralysis is a paralysis of motion more or less complete. In general, however, sensation is also more or less impaired. In estimating the condition of the sentient functions, the same method is to be adopted which Weber devised in comparing the sensibility of the surface of the skin in different parts of the body. It consists in ascertaining how near the sharp points of a pair of compasses may be approximated, and yet be distinctly felt as two points by the patient.

The special lesions of the brain causing hemiplegia are—(1.) Obstruction of a principal cerebral artery by a plug of fibrin detached from an excrescence on one of the aortic or other valves of the heart,—the result of a former endocarditis (KIRKES and VIRCHOW). (2.) A coagulum formed in an artery, resulting from some altered nutrition of its wall, and connected in general with a rheumatic or other morbid state of the blood. (3.) A softened state of the brain,

such as the condition known as white softening, which follows the retardation and diminution of cerebral circulation by diseased arteries, or by the complete stoppage of an artery by a plug. (4.) Apoplexy, induration, or tumors—*e. g.*, tubercle or cancer in the parts indicated above.

**Treatment.**—The object to be aimed at in the early treatment of hemiplegia is to keep down the frequency and force of the heart's action. For this purpose strict maintenance of the horizontal position is necessary; and when Consciousness exists, let the Mind be kept tranquil by every means. Remove any local impediment to the easy flow of blood, and let the head be slightly raised, sufficient to prevent gravitation favoring the escape of blood from the ruptured vessels, but not so as to create any impediment to the flow, and so embarrass the action of the heart. Let the bowels be cleared out, so that no irritation from them may operate injuriously on the brain. In so doing, enemata ought to be employed; and failing these, castor oil or calomel, with compound jalap powder, may effect an efficient evacuation.

With regard to bloodletting, there are three objects to be attained,—(1.) To diminish an undue amount of blood to the head; (2.) To check hemorrhage, or to prevent it; and (3.) To quiet the action of the heart (TODD).

The circumstances under which its use is inadmissible are thus defined by Dr. Todd: If the patient be cold and collapsed; if the heart's action be feeble and intermittent; if there be an anæmic state; if the patient be of advanced age; if there is evidence of extensive disease of the arterial system or of the heart; or, lastly, if it can be ascertained that already a large amount of hemorrhage has taken place into the brain; these, singly or conjointly, are reasons why bleeding *ought not to be resorted to*. If none of these objections exist, it is to be considered whether any of the indications noticed require to be fulfilled, and whether they can be fulfilled by a local or general bloodletting. Modern investigations show that the brain is not generally in a hyperæmic state; so that it is chiefly to check or to prevent hemorrhage that bleeding is to be resorted to in such cases. The sudden or rapid abstraction of a moderate quantity of blood, either from the arm or temple, or by skilful cupping, may check hemorrhage, but the quantity taken should be small; and so, likewise, the quantity drawn ought to be moderate if it is desired merely to lessen the frequency and force of the heart's action. "Generally," writes this distinguished physician, "I have come to the conclusion that, in cases of white softening, you are less likely to err by omitting than by adopting the practice." The rigidity of the muscles, which comes on very early, and which indicates an inflammatory process going on round the clot, is to be combated by urinary and alvine evacuants, and by counter-irritation. It is not desirable to interfere in the *later* forms of muscular rigidity. With regard to the use of expedients for promoting the restoration of the paralyzed limbs to their normal conditions, Dr. Todd writes that he knows of nothing which is of more



decided benefit than a regulated system of exercise—active when the patient is capable of it, passive when he is not.

Any congestion of the spinal cord apt to supervene on cerebral hemiplegia must be diminished. The patient should not lie on his back, but, if possible, flat on his belly, the arms and legs being incased in flannel, and placed at a lower level than the spine. A hot douche may also be applied to the spine, the water falling from a height of at least *four* or *five* feet, through a tube *three-quarters* of an inch or *one inch* in diameter; and its temperature should be between 98° and 100° Fahr. The applications should be continued two or three minutes, and continued daily for some time. *Dry cupping* over various parts of the spine may also be of service, and so also may blisters, moxas, cauteries (BROWN-SÉQUARD). The most useful internal remedies are those which tend to lessen congestion in the cord, namely, *belladonna* and *ergot of rye*. The dose of *ergot* in powder is at first *three grains twice a day*, and gradually the dose may be increased till it reaches *six grains twice a day*. *Belladonna* may be applied to the spine in the form of a plaster *four inches wide and six or seven inches long*; and if no amelioration of the symptoms follows in a few weeks, the *extract of belladonna* may be given in doses of *a quarter or a third of a grain twice daily*; and if after six or eight weeks of this treatment no improvement is observed, Dr. Brown-Séquard recommends that *iodide of potassium* in doses of *five or six grains twice a day* may be given in addition to the *belladonna*. If meningitis is supposed to exist along with chronic myelitis, then the *iodide of potassium* should be given from the very commencement, along with the *belladonna*, or with *secale cornutum*, as recommended by Dr. Meryon. Spermatorrhœa may be relieved by pressure applied over the region of the prostate (THOMAS and MERYON).

To prevent atrophy of paralyzed limbs, the application of galvanic currents and the use of the flesh-brush are recommended; and when œdema of the limbs exists, a warm bath to the part every night is of service. The bowels must be kept open, and if anodynes are required, opium should be avoided, and hyoscyamus, conium, or Indian hemp, should be used instead. Dr. Brown-Séquard recommends hyoscyamus. Iodide of potassium ought to be taken before food in the morning, and an hour before dinner, so as to avoid its presumed decomposition by the gastric juice.

The nutrition of the spinal cord ought to be improved by the daily use of the cold douche; and sea-bathing may be of service. If symptoms of irritation do not exist, *belladonna* is not to be given; it is only in cases of congestion that it may be useful in diminishing paralysis. And the same rule applies to *ergot of rye*. *Strychnine* increases the amount of blood in the spinal cord. It may be employed in paralysis only when there is no sign of irritation, and ought to be avoided when there are signs of congestion or irritation (BROWN-SÉQUARD).

## PARAPLEGIA.

LATIN EQ., *Paraplegia*; FRENCH EQ., *Paraplégie*; GERMAN EQ., *Paraplegie*; ITALIAN EQ., *Paraplegia*.

**Definition.**—*A form of paralysis affecting the lower half of the body, in which both legs, and perhaps, also, some of the muscles of the bladder and rectum, are paralyzed.*

**Pathology.**—Of this kind of paralysis there are at least two forms, which differ as to their mode of origin—namely, (A.) *Reflex Paraplegia*; (B.) *Paraplegia due to myelitis in some one of its numerous forms.*

(A.) *Reflex Paraplegia.*—[The term *reflex paraplegia* or *reflex paralysis* was first proposed and used by Dr. Brown-Séquard (*Lectures on the Diagnosis and Treatment of the Principal Forms of Paralysis of the Lower Extremities*, 1861), and is objected to by Dr. Jaccoud in his work, *Les Paraplégies et l'Ataxie du Mouvement*, 1864, because it involves a physiological contradiction in terms, the constant and pathognomonic characteristic of reflex phenomena being movement, and it is precisely for this reason that physiology uses synonymously the two expressions, reflex action and excito-motory action. Even if Dr. Brown-Séquard's theory of the pathogeny of this form of paralysis be correct, it is not the paraplegia which is reflex, but the contraction of the bloodvessels of the medulla, and that the name should correspond correctly with the theory, it ought properly to be called *paraplegia from reflex vascular contraction*, or more properly, *paraplegia from reflex ischæmia*. Dr. Jaccoud proposes to name this variety of palsy, *paralysis from peripheral irritation*. Dr. Handfield Jones employs the term *inhibitory paralysis*, for this class of nervous disorders (*Clinical Observations on Functional Nervous Diseases*, 1864), believing that it is not the energetic operation of an afferent nerve which produces the effect, but its being injuriously affected by some impression made upon it.]

Physicians and surgeons at different times have met with and recorded cases of paralysis which the amount of disease present in the nervous centre or its covering after death would not account for; which blood-poisoning would not account for; but which were found to be uniformly associated with injuries or diseases of parts or organs remote, and not directly contiguous to the spinal marrow. Such cases were assumed to be cases of "Reflex Paralysis;" and they have now been long recognized and described by various observers. Mr. Stanley, in 1833, published a paper in *The Medico-Chirurgical Transactions* "On Irritation of the Spinal Cord and its Nerves in connection with Disease of the Kidneys." He there records cases of paraplegia in which no morbid appearances were detected in the cerebro-spinal axis, but where inflammation of the bladder or kidneys, or gonorrhœa, had existed for some time. Similar cases of "Urinary Paraplegia" have been recorded by Romberg, Graves, Rayer, and several other writers. Mr. Spencer Wells recorded in *The Medical Times and Gazette* cases of this kind, in 1857, having made them the basis of an excellent clinical lecture at the Grosvenor School of Medicine. Dr. Brown-Séquard, in a series

of four lectures, published in 1861, relates the grounds of diagnosis and the principles of treatment of these various forms of paraplegia. Lastly, Dr. Meryon describes seven forms of paralysis as due to reflex action, namely—(1.) *Emotional paralysis*. A case of this kind is described by Dr. Meryon, in his work already referred to, p. 172. Dr. Wiblin, of Southampton, in *The Lancet* of August 11, 1860, records a case of so-called emotional paralysis. The subject of that case died in October, 1864, and I had an opportunity of removing the brain and cord in connection, which I sent to Mr. Lockhart Clarke for his inspection. From the post-mortem appearances, I regarded the case as one of *chronic meningitis*, associated with disease of the bloodvessels. In this view Mr. Lockhart Clarke concurred, and mentioned that he found cysts also in different parts of the brain—two or three small ones in the white substance round the *corpus dentatum cerebelli*. One of these contained a fluid that was perfectly milky, and appeared under the microscope in the form of granular globules of oil, about twice the size of pus-globules, with a multitude of oily molecules. Several cysts were found in the *cerebrum*; and the *corpus quadratum* of the right side was hollowed out into a large cyst. Such lesions remove this case from the class of so-called “*Emotional paralysis*.” (2.) Pregnancy is apt to induce such *reflex paraplegia*, or sometimes *hemiplegia* or *amaurosis* (CHURCHILL). (3.) *Neurolytic paralysis*, in which, from no adequate cause, the functions of the cord seem suspended for a time, associated generally with exposure to cold and wet. (4.) Paralysis from the *irritation of worms* in the intestinal canal (DAVAINE, MERYON). (5.) Paralysis from the *irritation of teething* in children. (6.) *Urinary Paralysis*. (7.) Paralysis from *uterine disease*, as from *dysmenorrhœa*, *metritis*.

[8. Sudden paralysis of remote parts of the body from *mechanical injuries*, particularly gunshot wounds; for example: a wound involving the muscles of the right thigh followed by reflected paralysis of the right arm and left leg; a wound of the right thigh causing paralysis of the right arm; a wound of the right testicle, followed by paralysis of the right anterior tibial muscle and peroneus longus; a wound of the external part of the left thigh producing anæsthesia and analgesia of a corresponding part of the right thigh; a wound of the right thigh probably involving the crural nerve, in which there was motor paralysis of the right arm. However great the lesion of motion or sensation at first, in all instances it grows better early in the case, and continues to improve until the part has nearly recovered all its normal powers; but nearly in all some relic of the paralysis remains, even after eighteen months or more from the date of wounding. In some, the part continues weak; in others there is still some slight loss of sensibility; and in others there persists considerable loss of power and sensory appreciation. In a case of reflex paralysis from a wound, we have therefore some right to expect that the patient will recover rapidly up to a certain point, but that in most cases a small amount of loss of power and sensation may be left (S. W. MITCHELL, MOREHOUSE, KEEN).

The first recorded cases of this variety of reflex palsy were, it is believed, by Drs. S. Weir Mitchell, George R. Morehouse, and W. W. Keen, of Philadelphia, while acting as medical officers in the U. S. A. Hospital

for Injuries and Diseases of the Nervous System, in that city, during the late war (*Circular No. 6, Reflex Paralysis, Surgeon-General's Office, March 10, 1864*).

Hitherto it appears to have escaped the notice of observers, probably from its infrequency, for "among some two hundred or more carefully studied instances of [gunshot] wounds of the nerves, only seven cases of reflex paralysis of remote organs were met with, in which the influence was prolonged or severe. In the majority of cases the effect is either very slight or very transient, and for one or both of these reasons unlikely to attract notice from surgeons on the battle-field, or in division and corps hospitals" (*l. c.*, p. 1). "The effect of grave wounds is to cause a condition of the [nerve] centres which gives rise to a general and profound feebleness, and in rare cases the central effect is so intense as in some way to cause paralysis, which may last for hours, days, or months" (*l. c.*, p. 4).]

The views entertained by these several writers regarding the existence, especially, of a "Reflex Paraplegia" have been called in question by many able pathologists, and chiefly by Drs. Gull, Nasse, and Valentiner; but the facts on which the belief in "Reflex paraplegia" rests, and which show that "a paralysis of the lower limbs may be caused by some alteration in the *periphery*, or the trunk of various sensitive nerves," may be shortly stated as follows: (1.) A very rapid cure of the paraplegia follows the removal or cure of the primary disease which involved the peripheric sensitive nerves. Such rapid cures do not result in cases of paraplegia when the spinal cord or its membranes are primarily diseased. (2.) There are certain characteristics of *reflex paralysis* which tend to show how distinct it is from the cases of paralysis depending on organic alteration of the nervous centres. These characteristics are,—(a.) An outside excitation connected with some morbidly sensitive surface or nerve, and which exists for some time before the paralysis comes on—*e. g.*, stricture of the urethra, gonorrhœa, disease of the kidney, prolapse of the womb, and the like; (b.) Variations in the degree of this excitation are followed by variations in the paralysis; (c.) The cure of the paralysis follows the subsidence of the primary disease.

Cases of "reflex paralysis" are also well known to occur in all the upper parts of the body—*e. g.*, paralysis of the optic nerve is sometimes due to injury of the frontal nerve; paralysis of the auditory nerve is sometimes due to neuralgia of the face; local palsy, as of the eye, the neck, the trunk, the bladder, or the rectum, is sometimes due to teething, worms, or other sources of excitement of the sensitive nerves, terminating in the mucous membranes of the skin; and the cure of amaurosis after the expulsion of worms is by no means uncommon.

Cases of paraplegia following diseases of the womb, and cured after the cure of this disease, are recorded by Romberg, Hunt, Wolf, Brown-Séquard, and others. Cases of paraplegia due to a disease of the urethra are on record by Graves, Wells, and others. Cases of paralysis consequent on inflammation of the bladder—gonorrhœal cystitis—have been related by Rayer and Brown-Séquard. Cases of reflex paralysis are also due to diseases of the

prostate or kidney, to enteritis, dysentery, diphtheria, and affections of the lungs and pleura. The paralysis which attends teething has been known to increase and decrease as the molars are cut. Irritation of the nerves of the skin, generally following cold and wet, has been known to induce reflex paraplegia (*neurolytic paralysis* of Handfield Jones); and so also has disease of the knee-joint. In all these instances the paralysis follows the primary disease which is the cause of the local "peripheric excitation." Increase or decrease of this excitation and of the attendant paralysis, according as the cause persists or is suppressed, is among the recognized phenomena of such cases.

There are at least two ways by which the reflex action from the outside irritation may induce paraplegia, namely—First, *the reflex contraction of bloodvessels*. Brown-Séquard has the great merit of having actually demonstrated this. We know that the bloodvessels can contract with energy on the application of a stimulus or excitant, sometimes to the extent of a real and prolonged spasm; and we know that the nervous system is amply supplied with bloodvessels. In *three* different places—namely, (1.) *In the spinal cord*; (2.) *In the motor nerves*; (3.) *In the muscles*—such a contraction of bloodvessels is found to cause "paraplegia." In the vessels of the pia mater of the spinal cord, Dr. Brown-Séquard has seen how the vessels contract when a ligature was applied on the *hilus* of the kidney, or when a similar operation was performed on the bloodvessels and nerves of the *suprarenal* capsules.

Generally, in those cases, the contraction is much more evident on the side of the cord corresponding to the side of the irritated nerve. These demonstrations also coincide with other physiological evidence. More than half a century ago, Comhaire extirpated the kidney from living dogs, with the effect of instantly producing paralytic weakness of the hind leg of the side operated on. Brown-Séquard found the same result on extirpating either a kidney or a suprarenal capsule. It is, therefore, quite legitimate to conclude that irritations of these parts from disease may produce a paraplegia, by causing contractions of the bloodvessels of the cord similar to those which took place in these experiments.

There are other reasons which show that such paralysis is due to reflex action and probable contraction of bloodvessels—namely, the fact familiar to surgeons, that chills, tremors, and even convulsions, are often connected with catheterism. Another kind of proof is that of a negative kind, which shows that "the spinal cord may have its functions impaired, and even lost, and that suddenly, without any anatomical lesion." This is proven by the post-mortem examination of cases dying after symptoms of reflex paraplegia. Such cases are related by Stanley, Rayer, Cruveilhier, Chomel, Drs. Stokes and Graves, of Dublin, and Dr. Gull.

The present position of our knowledge of the structure and functions of the spinal cord hardly entitles us to assert, without careful microscopic examination, whether alteration of the cord does or does not exist in cases of *reflex paralysis*. Of all these cases, only in that of Dr. Gull was any microscopic examination



made; and it is not impossible, but it is very probable, that a functional lesion, established even in a reflex way, may go on to organic mischief in the end.

[Dr. Brown-Séquard's theory of the production of paralysis due to peripheral causes is, that a state of irritation, beginning eccentrically, is propagated along the sensory nerves, of which the result is, primarily, contraction of bloodvessels in, and secondarily, exclusion of the due amount of blood from, one or more of the following parts—the spinal cord, the nerves going to or coming from the cord, and muscles. An impartial critical investigation of this hypothesis, pronounced by an opponent as "all but as conclusive as it is masterly and original," would, however, seem to sustain the objections made against it by Gull, Eisenmann, Valentiner, Nasse, S. W. Mitchell, Jaccoud, and others. The eminent physiologist asserts that when he irritated the nerves of the kidney and suprarenal capsules, he saw the bloodvessels in the spinal pia mater contract, and that generally the contraction was most marked on the side the nerves had been most irritated. The first inquiry which naturally is made is relative to the constancy of the result of the experiment. Dr. W. W. Gull, who repeated the experiment, with the assistance of Dr. Pavy and Mr. Durham, got only negative results (*Guy's Hospital Reports*, 1861); moreover he asserts that the anatomical disposition of the bloodvessels of the medulla and its meninges is such as to render their contraction very difficult to be seen by the naked eye. Raoul Leroy d'Etiolles irritated the kidneys directly in five dogs, and in no instance did paraplegia follow (*Des Paralysies des membres inferieurs*, 1857). Comhaire's experiments, in which paraplegia followed extirpation of the kidneys, are not satisfactory, and neither Gull, Leroy, or W. A. Hammond (quoted by Mitchell) found paralysis of the lower extremities to follow the removal of these organs in animals. Schiff never could induce reflex movements in the lower extremities by irritation of the kidney, when the irritation was limited to the gland itself (*Lehrbuch der Phys.*, 1858). The hypothesis requires that the contraction should not be transitory, but persistent within certain limits, to produce a degree of spinal ischæmia capable of causing insufficient nutrition, and consequent loss of function. But this is contrary to well-established physiological laws, for everywhere, exhaustion follows unnatural excitement, and, we find passive dilatation of the vessels succeeding to active contraction, the more rapidly, and greater in degree, as the latter has been extreme. Donders and Van der Becke Callenfels found, on irritating the cervical sympathetic in the rabbit, the arteries of the cerebral pia mater contract, but this was speedily followed by dilatation, and Dr. Brown-Séquard himself attributes the convulsions in epilepsy to temporary anæmia resulting from contraction of the bloodvessels of the brain (*Researches on Epilepsy*, 1860). Moreover, the experiments of Sir Astley Cooper, Kussmaul, and Jenner show that after excessive hemorrhage convulsions are apt to happen. Dr. Handfield Jones objects that it is difficult to suppose that a spasm of reflex origin should be limited to such a very small extent of vessels as would be involved in some instances, as palsy of one of the sixth nerves, ptosis of one eye. Dr. Jaccoud is of the opinion that the medulla is so well provided with bloodvessels it would not be possible by a localized contraction to cut off the vascular supply from any considerable portion. The pathological hypothesis of Dr. Brown-Séquard in peripherally induced paralysis—spinal ischæmia—offers so many obvious defects, and is so unsupported by facts and analogies, that it becomes very difficult to admit it. The

theory which supposes paralysis from peripheral causes to be due to long-continued or sudden and violent irritation of a centre through its afferent nerves, thereby exhausting it so completely as to occasion a palsy of the parts depending upon it for nervous influence—paralysis by irritation and consequent exhaustion—seems to have been, partially at least, recognized by Drs. Gull (1860), Eisenmann (1860), and Handfield Jones (1861), but was distinctly set forth and advocated by Drs. S. W. Mitchell, Morehouse, and Keen (Circular No. 6, April, 1864), and has been elaborately and ably maintained by Dr. Jaccoud (Dec. 1864). "It appears to us, possible," observe the American physicians, "that a very severe injury of a part may be competent so to exhaust the irritability of the nerve centres as to give rise to loss of function, which might prove more or less permanent. A strong electric current is certainly able to cause such a result in a nerve-trunk, while a general electric shock, as a stroke of lightning, may destroy the irritability of every excitable tissue in the body. Reflecting, then, upon the close correlation of the electrical and neural force, it does not seem improbable that a violent excitement of a nerve-trunk, however brought about, should be able to exhaust completely the power of its connected nerve-centre. The central change thus occasioned would, no doubt, involve the consequent or immediate occurrence of nutritive changes, which would gradually yield as time went on" (p. 16). Dr. Handfield Jones believes that the so-called inhibitory influence is adequate to account for the phenomena of many of the reflex palsies. "The enfeebled state of the nerve itself, or of the centre to which it proceeds, or the severity or malignity of the impression, may give rise to the familiar effect" (*loc. cit.*, p. 10). He believes with Lister that "one and the same afferent nerve may, according as it is operating mildly or energetically, either exalt or depress the functions of the nervous centre on which it acts." Against this explanation of functional paralysis from peripheral irritation, it may be urged, that in health, inhibitory phenomena are only known to occur in the pneumogastric nerve, and within the range of the sympathetic; and we have no right to include pathological phenomena involving the spinal centres and nerves, or the voluntary muscles; and that there is reason to suspect that the so-called inhibitory phenomena are due to exhaustion of nerves or ganglia, by over irritation (S. W. MITCHELL). Dr. M. G. Echeverria, whilst acknowledging exhaustion as among the causes of reflex paralysis, and without denying the contraction of the bloodvessels of the cord, which, however, he considers a secondary element, believes it induced through the agency of the sympathetic system (*Reflex Paralysis*, 1866).

Dr. C. B. Radcliffe, examining Dr. Brown-Séquard's argument, writes (Reynolds' *System of Medicine*, vol. ii, p. 659, *et seq.*): "It is, no doubt, true enough that a state of irritation in vaso-motor nerves may lead to contraction in bloodvessels, and thereby exclude a due amount of blood from the part to which these vessels belong; but it is not less certain that the same state of irritation carried beyond a given degree, either in time or in intensity, may, by paralyzing the vaso-motor nerves, lead to relaxation of vessels, and, thereby, to the admission into them of an undue amount of blood. Moreover, it may also be assumed, as a thing by no means improbable, that the contraction of the coat of the relaxed and paralyzed vessels in rigor mortis may prevent any marked traces of such vascular engorgement being met with after death. . . . It is easy enough to find marked differences between paraplegia from myelitis and reflex paraplegia; but the case is far otherwise when a comparison is instituted between paraplegia from spinal congestion and reflex para-

plegia. In reflex paraplegia the paralysis is incomplete, and all muscles are not affected equally; in paraplegia from spinal congestion it is so also. In reflex paralysis the paralysis is not associated [usually?] with tingling, numbness, or anæsthesia: in paraplegia from spinal congestion it is the same, with the single exception, that there may be at one time or other a trifling degree of tingling at the extreme tips of the fingers or toes. In reflex paraplegia there are fluctuations in the degree of the paralysis; so also in paraplegia from spinal congestion. In reflex paraplegia there is no marked change in the nutrition of the muscles; so also in paralysis from spinal congestion. And, lastly, in reflex paraplegia, as in paraplegia from spinal congestion, a cure is neither an impossible nor even an improbable, event."

There is no doubt that too often paralysis has been called nervous, functional, or sympathetic, when its only title to the name has been the shortcomings of the necroscopic examination, or an ignorance of the action of the blood in certain conditions upon the medulla; and a rigid critical analysis of the reported cases of paralysis *sine materiâ* will greatly reduce their number. Mitchell, Jaccoud, and Echeverria have shown that many cases usually explained as functional paralysis, or presumed to be brought about by peripheral irritations, may with more reason be assigned to other causes,—some material lesion of the nerve-centres; and carefully conducted microscopical investigations will, very likely, prove that central and peripheral nerve-lesions are the rule and not the exception in the pathogeny of so-called reflex paralysis. Still, with all our methods of investigation, we find, as has been remarked by Dr. S. W. Mitchell, "after every rational exclusion has been effected, numerous cases of palsy to which we can assign no possible cause, except an external irritation, in some way acting through nerves upon nerve-centres to occasion loss of function." (On this subject see Jaccoud, *Paraplegies et l'Ataxie du Mouvement*; S. Weir Mitchell, *Paralysis from Peripheral Irritation*; Echeverria, *Reflex Paralysis*; C. Handfield Jones, *Clinical Observations on Functional Nervous Disorders*.)

Paralysis due to *myelitis* having been already noticed in connection with that disease, it may be useful to contrast here, in a tabular form, the leading phenomena of the two diseases, as given by Dr. Brown-Séquard, in his admirable lectures *On Paralysis of the Lower Extremities*; and which, to some extent, are subscribed to by Dr. Edward Meryon.

#### PARAPLEGIA DUE TO

##### (A.) URINARY REFLEX IRRITATION.

1. *Preceded* by an affection of bladder, kidneys, or prostate.
2. Usually lower limbs alone paralyzed.
3. No gradual extension of the paralysis upwards.
4. The paralysis is usually incomplete—an extreme debility or weakness of the limbs rather than paralysis.
5. Some muscles more paralyzed than others.
6. Reflex power neither much increased nor completely lost.

##### (B.) MYELITIS.

1. Usually no disease of the urinary organs, except as a consequence of the paralysis.
2. Usually other parts paralyzed besides the lower limbs.
3. Most frequently a gradual extension of the paralysis upwards.
4. Very frequently the paralysis is complete.
5. The degree of paralysis is the same in the various muscles of the lower limbs.
6. Reflex power often lost, or sometimes much increased.

## (A.) URINARY REFLEX IRRITATION.

7. Bladder and rectum rarely paralyzed, or at least only slightly so; *sphincter ani* weak.

8. Spasms in paralyzed muscles extremely rare.

9. Very rarely pains in the spine, either spontaneously or on the application of pressure, percussion, or a hot moist sponge, or ice.

10. No feeling of pain or constriction round the abdomen or the chest.

11. No formication, pricking, nor disagreeable sensations of cold or heat.\*

12. Anæsthesia rare; the tactile sensibility being but slightly if at all impaired, but the muscular sense may be almost lost.

13. Usually obstinate gastric derangement.

14. Variations in the degree of the paralysis corresponding with changes in the primary disease.

15. Urine is usually acid, unless the urinary organs are diseased.

16. Cure of the paralysis frequently and rapidly obtained, or taking place spontaneously, after a notable amelioration or cure of the urinary affection.

17. *Usually* muscles do not become atrophied, and temperature is little lowered.

## (B.) MYELITIS.

7. Bladder and rectum usually paralyzed completely, or nearly so.

8. Always spasms, or at least twitchings.

9. Always some degree of pain, existing spontaneously or caused by external excitations.

10. Usually a feeling as if a cord were tied tightly round the body at the upper limit of the paralysis.

11. Always formications, or pricking, or both, and very often sensations of heat or cold.

12. Anæsthesia very frequent, and always at least numbness.

13. Gastric digestion good, unless the myelitis has extended high up in the cord.

14. Ameliorations very rare, and not following changes in the condition of the urinary organs.

15. Urine almost always alkaline.

16. Frequently a slow and gradual progress towards a fatal issue; very rarely a complete cure.

17. Atrophy of the muscles of the paralyzed parts.

It is necessary, therefore, to look for some primary disease to account for *reflex paraplegia*, believing at the same time that such primary disease may ultimately establish a myelitis, while a diagnosis of *reflex paraplegia* is mainly based on a contrast of the phenomena which attend the various forms of paraplegia due to other causes.

In addition to the characters in the table, there are some symptoms peculiar and almost pathognomonic of forms of paralysis due to other local lesions—*e. g.*, meningitis of the spinal cord induces rigid spasms of the muscles of the back; intense pain on motion of lower limbs or of the spine; spontaneous acute pains that radiate from the spine to the lower extremities (similitude to rheumatism); frequency of cramps; and pressure on the cord by a tumor or diseased bone. They may and often do produce a myelitis. Pressure causes a feeling of tightness and pseudo-neuralgic pains. A tumor in the gray matter of the cord is attended with anæsthesia from the very

\* [Graves, describing reflex paraplegia, says: "With respect to sensation, it appears to be affected as well as motion. In the acute form it is generally the first symptom noticed by the patient. There is first a feeling of numbness, followed by formication and tingling pains, then loss of power and diminished sensation (*Clinical Lectures*). In Stanley's cases sensation was affected. In the fifty-three cases collected by R. Leroy d'Etiolles (*loc. cit.*), sensibility was generally diminished and perverted, especially in urinary paraplegia. In the cases reported by Drs. Mitchell, Morehouse, and Keen, "four of the seven cases had stinging, smarting, and burning pain in the part paralyzed. Pain was an early symptom."—EDITOR.]

first, which may reach a higher degree than the power of motion; and reflex power below the lesion becomes then extremely exalted. In hemorrhage into the cord there is pain, then sudden paralysis, and often convulsions.

**Treatment.**—In cases of *reflex paralysis* means must be taken—1st. To diminish the external irritation which causes the paralysis; 2d. To improve the nutrition of the spinal cord; 3d. To prevent the ill effects of rest on the paralyzed nerves and muscles.

In cases of disease of the urethra or prostate, an injection of a solution of one grain of the extract of belladonna, in twenty drops of laudanum, is to be made into the urethra, and the injection should be retained for half an hour, or even an hour, after which some emollient decoction should be used, such as linseed tea, to wash out the passage. Every two or three days this operation should be repeated. If the bladder be diseased, Dr. Brown-Séguard advises a solution of one grain of the extract of belladonna, in twenty drops of laudanum, to be used as an injection, after a complete emission of urine. Injections of carbolic acid are also of use in counteracting decomposition of urine and epithelium in the bladder. If the prostate is enlarged, a suppository ought to be put at times into the rectum. One of the best suppositories for this purpose is composed of the following ingredients, namely: *White sugar, white soap, and gum-arabic in powder, of each three grains; opium, in powder, a grain and a half; or belladonna extract, one grain; or both combined. These being mixed together, the mass is to be formed into a conical shape, and being dipped in melted wax, is ready to be introduced, when required, into the rectum* (SIMPSON).

When the irritation causing reflex paraplegia starts from the vagina or uterus, a pill of half a grain of the extract of belladonna, with a grain of opium, surrounded by a piece of cotton-wool, is to be introduced into the vagina, and made to surround the neck of the womb. By means of a thread it may be withdrawn so soon as the pain ceases or diminishes (TROUSSEAU, BROWN-SÉQUARD).

*Belladonna* ought not to be used constantly. *Opium*, combined with *strychnia*, is of greater use in reflex paralysis; and of all remedies, *strychnia* is best suited for promoting the second indication, which points to increasing the nutrition of the spinal cord. The dose combined with opium must be a small one—namely, *one-fortieth to one-thirtieth of a grain daily*; and when used alone, its dose may be *one-twentieth of a grain*; and when employed together with *belladonna*, its dose must be still larger.

In cases where no congestion nor inflammation of the spinal cord exists, *strychnia* ought to be persistently employed; but its use ought to be suspended at once whenever it produces spasms, or even numbness of the feet in getting out of bed in the morning (BROWN-SÉQUARD). *Sulphur baths* are also productive of great benefit.

The third indication—namely, to prevent the ill effects of rest on the paralyzed nerves and muscles—is best met by the employment of galvanism and shampooing of the paralyzed limbs. Two or three applications, of ten minutes each, in a week, are sufficient; but of all things, it is necessary that the primary disease (causing



by its persistent existence the reflex paralysis) should be cured or mitigated.

There are yet [five] forms of so-called paralysis to be noticed, characterized by a tendency to progress from bad to worse. These are—(1.) Progressive Muscular Atrophy, or Wasting Palsy; (2.) Progressive Locomotor Ataxy; (3.) Glosso-laryngeal Paralysis; (4.) Progressive Paralysis of the Insane. [(5.) Acute Creeping Paralysis.]

### PROGRESSIVE MUSCULAR ATROPHY.

LATIN Eq., *Atrophia ingravescens*; FRENCH Eq., *Atrophie progressive*; GERMAN Eq., *Progressive Arophie* [*Progressive Muskel-Atrophie*; *Progressive Muskel-lähmung*]; ITALIAN Eq., *Atrofia progressiva*.

**Definition.**—*A peculiar wasting of muscles, with atrophy of their substance, and lesion in the anterior roots of the nerves of the spinal cord, and paralysis.*

**Pathology and Symptoms.**—Idiopathic paraplegia is doubtful; yet it is certain that cases do occur, concerning which it is not easy to say, during the life of the patient, nor even after a post-mortem examination, what is the cause of the paralysis. For example, the cases of so-called “wasting palsy,” the *paralysie musculaire atrophique* of Cruveilhier, are not yet made out to be a really specific disease independent of the state of the spinal cord. When spinal cords are examined in the way they have hitherto been examined, most of them are pronounced healthy. Of the numerous cases of “wasting palsy” collected together by Dr. Roberts, in thirteen only was the nervous system examined, out of one hundred and five in all, and of these thirteen, four of them had disease of the cord. Obscure structural changes in the gray substance of the cord, or even only in the gray substance of the ganglia on the posterior roots of the nerves, may affect the nutrition of parts to which they are subservient, without interfering with the function of motion or sensation in the first instance.

A very interesting case of local atrophy of muscles, and local paralysis of the specially affected groups, is related in Beale's *Archives of Medicine* for October, 1861, which would no doubt have been set down as a case of “wasting palsy” independent of disease of the cord, if that organ had not been sent to Mr. Lockhart Clark, and carefully examined by him. He found, and demonstrated, that lesions existed in it which occurred in small isolated spots, sufficient to account for the limitation of the disease to particular muscles. If, therefore, we are ever to arrive at accurate and available results, both as regards physiology and pathology, our mode of examining the spinal cord must be more searching and exact than heretofore. There is no royal road to the removal of the spinal cord. Time, patience, and great caution are necessary to avoid injuring the soft parts, so that, when hardened perfectly, entire sections may be got for examination under the microscope. If this plan were more systematically adopted, it would undoubtedly open up a new field

of pathological and physiological study, furnishing results which no experiment in vivisection could possibly reach, if the history of the case was complete. Numerous cases of wasting of the muscles of one or more limbs, independent of any well-defined cause, have been from time to time observed, and are recorded in various publications; and we are indebted to Dr. William Roberts, of Manchester, for the first systematic treatise on the subject, in 1858. Such cases have hitherto been looked upon as *extraordinary* or *anomalous cases*, and were described as instances of "creeping palsy," "partial" or "local palsy," or "anomalous hemiplegia." In France it has been described by the name of "*atrophie musculaire progressive*," or "*paralysie graduelle du mouvement par atrophie musculaire*" (DUCHENNE, ARAN, CRUVEILHIER), corresponding to the third form of progressive paralysis described by Meryon—namely, from "granular degeneration of the muscles, and where no disease or lesion is found in the nervous centres."

In 1851 Dr. E. Meryon gave an account of a gentleman's family in which three boys were the subjects of general muscular degeneration. His observations were original, and are published in the *thirty-fifth* volume of the *Medico-Chirurgical Transactions*, p. 73. Several cases have been described since Dr. Roberts called attention to the subject; and the more closely and carefully the spinal cord has been examined after death by competent observers, the more surely has it been found diseased. In all the cases observed the muscles affected are those under the control of the Will; hence the course of the disease is easily followed, either by the changes produced in the external form of the parts, the absorption of masses of muscles, the displacement of bones, the abnormal position of joints from loss of their muscular supports, or by the failure of certain movements which contribute to outward expression or inward function, such as facial physiognomy, deglutition, vocalization, or respiration—all of which depend on the operation of striped muscles under the influence of the Will. When disease affects the muscles of the limbs the *disappearance* of the *muscles* causes very notable changes of conformation. The rounded contour gives place to a lean and withered aspect, the bony levers stand out in unaccustomed distinctness, so that the limb has the appearance of a skeleton clothed in skin (ROBERTS). As a general rule, when one limb is attacked, its fellow on the opposite side shares its fate; when the disease is unilateral, the right side is more likely to be its seat than the left; but the disease seems to be extremely capricious and uncertain in its line of attack, scarcely two examples being exactly alike in the combination of muscles implicated, or the relative degree in which they suffer. But when the malady affects the shoulder it scarcely ever fails to include the upper arm; so also the forearm and hand are generally associated in the disease. In the upper limbs the morbid action seems to radiate from two centres: one in the hand, from which the forearm is invaded; and the other in the shoulder, from which are reached the muscles of the upper arm, and those which brace the shoulder-blade to the ribs. When the hand and arm are destroyed, the evil does not then pass up

the arm, but starts away to the shoulder, or to the opposite hand. In the same way, when the shoulder is first attacked, the disease does not descend along the upper and forearm to the hand, but, passing over the elbow, it begins afresh in the ball of the thumb, and from that focus spreads up the limb; so that the parts latest reached are those about the elbow, especially the masses that take their rise from the humeral condyles.

The invasion of wasting palsy is usually slow and insidious. It creeps on unawares; and the victim of its attack only becomes cognizant of the disease when he notices some marked failure in certain muscular powers. The tailor notices that he cannot hold his needle; the shoemaker wonders that he cannot thrust his awl; the mason's hammer has grown too heavy for his strength; the gentleman feels an awkwardness in handling his pen, in pulling out his pocket-handkerchief, or in putting on his hat (ROBERTS). On comparing the weakened member with its fellow, it is seen to be wasted, and the failure of power increases; the lifting power is reduced to nothing; the grasp is gone; and at last palsy becomes complete. In the majority of cases the disease commences in the upper extremities; and if the disease commences in the legs, it is probable that the atrophy will spread to the trunk. In more than *one-third* of the cases noticed by Dr. Roberts the hand was the member originally seized; and the exact spot nearly always the ball of the thumb; and the right hand more often than the left. Next to the hands, one or other shoulder is the favorite starting-point.

*Loss of power* is a chief phenomenon, and it corresponds to the grade of the atrophy of the muscle; and it is only in extreme cases that any part is reduced to absolute immobility. *Muscular vibrations*, consisting of little convulsive twitchings or quiverings of individual muscular bundles, are also early phenomena. They do not impart any movement to the entire muscle, but parts of the muscle seem to spring beneath the skin in quick momentary tremors, undulating over the surface of the muscle. Tactile sensation retains its delicacy in the skin over the affected muscles.

[Cutaneous anæsthesia has, however, been noticed in some cases (DUCHENNE, BENEDIKT); and Remak and Benedikt have remarked an exaggerated facility of reflex movement, chiefly in the muscles subject to the quiverings described above. Electric contractility exists, but is less in the affected muscles, and the amount of diminution is in direct proportion to the degree of their atrophy (DUCHENNE).]

Pain is by far the most common of the nervous symptoms. It is present in about half the cases recorded. It is generally transient, and is usually marked at the commencement of the disease.

[In some instances sharp and continuous pains in the joints and along the muscles, from the outset, and lasting for several months, have been noticed (REMAK, BENEDIKT, JACCOUD). And pain in the dorsal spine and in the extremities is mentioned by Moritz Meyer, Roberts, and Friedberg. Remak insisted particularly on pain in the bones in wasting palsy.]

The advance of the malady has, in one case at least, been attended by an almost childish degree of helplessness, and a most pitiable state of mental irritability and hypochondriac depression (W. T. GAIRDNER, ADAMSON, BELL, GULL, DAY). *Unusual sensitiveness* to low temperature is occasionally a prominent and very annoying symptom.

[The temperature of the affected parts is always appreciably lowered, and in a case observed by Dr. Jaccoud, was variable, an important pathogenic phenomenon, if other observations show its frequency. In this case there were daily (morning) unilateral chilly fits, the patient complaining of great cold in the right arm and leg. The thermometer showed a fall of temperature in the limbs of both sides, but it was more marked in the right or most affected members, being less by  $3^{\circ}$  to  $5^{\circ}$  C. than the common temperature of those parts; while on the right side there was a difference of only  $1^{\circ}$  C. A paroxysmal ischæmia of the vaso-motory centres would appear to have regularly occurred, producing contraction of the peripheral vessels, and consequent fall of body-heat in the affected parts.]

As a rule, the general health does not seem impaired. Intelligence is clear, Judgment firm, and the Emotions under control, and all the organic functions appear to be performed with regularity.

**Prognosis.**—The *progressive forms of paralysis* are the most intractable in the domain of physic, and the gravity of the prognosis depends upon the disease confining itself to the extremities and the muscles thereto relating, or extending to the trunk and face. The signs of extension to the face are a diminished mobility of the lips, a slur in the articulation of words, frequent sighing, and fibrillary muscular tremors on different parts of the chest, abdomen, or face. If the disease has been hereditary, there is every fear that it will be fatal; and so also if the lower limbs are first attacked. The disease runs its course with great slowness. Some cases complete their history in six or eight months, others linger on through many years. Recovery, permanent arrest of the palsy, or death, are the various terminations of the disease. The mean duration of the cases ending in recovery has been *one year and two months*; of those ending in permanent arrest of the palsy, the mean duration of the cases has been *two years and three months*; of those terminating fatally, the mean duration was *five years and two months*. The longest duration of a case ending in recovery has been *two and a half years*, and the shortest period *eight months*. The longest case ending in arrest continued active for *seven years*, and the shortest for *four months*. Fatal cases have not been known to terminate under *twelve months*, while one lingered for *twenty-three years*, another for *eight years*, and some beyond *four years* (ROBERTS).

In those cases which terminate by permanent arrest the wasting of the muscles ceases, and the limb continues for an indefinite period in its maimed condition, neither amending nor deteriorating. The muscles atrophied are not regenerated, but remain *in statu quo*; and those which are not completely atrophied continue to exercise their feeble powers under the influence of the

1. *Chlorophyll a* and *Chlorophyll b* were determined by the method of Arar and Collins (1971) using a Shimadzu 1010 spectrophotometer.

1. The first group of people who are interested in the results of the study are the researchers themselves. They want to know if the study was successful in achieving its goals and if the data collected is reliable and valid. They also want to know if the study has contributed to the field of research and if it has provided any new insights or findings.

*[The page contains extremely faint, illegible horizontal lines of text, likely bleed-through from the reverse side.]*

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difficult to say whether it begins in the nerves associated with the muscles, and secondarily affects the spinal marrow connected therewith, in the manner referred to by Türck and Graves, and to which the term "creeping palsy" is applied; or whether (as Lockhart Clarke's observations tend to show) the lesion of the spinal cord is the primary lesion. In all cases the question to determine is, whether or not the atrophy of the muscle is subsequent to the paralysis. If so, there are good reasons for believing that the wasting of the muscular tissue is an immediate or direct effect of the lesions in the cord; and the paralysis is generally too rapid to be accounted for by mere inactivity of the paralyzed muscles. Dr. Meryon believes that only in the last form the disease commences in the muscles. Dr. Roberts, on the other hand, contends that the muscles are the parts essentially diseased, in the first instance, in the cases of "wasting palsy" which he has described. A blight (?) seems to wither the muscles, of the nature of a degeneration. He believes the disease to be of constitutional origin; and the evidence of this rests upon the facts that it is transmissible from parent to offspring, and that in its march it exhibits a bilateral symmetry.\*

[The question whether progressive muscular atrophy is an essential and primary disease of the muscular substance, or whether it is consequent upon structural change in the spinal cord, has been debated from the time of Cruveilhier to the present, and is still an open question. The author in his definition (p. 492), gives "lesion in the anterior roots of the nerves of the spinal cord," as the anatomical character; and in the text, the result of the post-mortem examinations of Dr. Roberts, and Mr. Lockhart Clarke (and those of Cruveilhier, Drs. Guy, Herard, Virchow, Luys, and others might have been added), are cited in support of this view. The condition of the spinal cord and of the spinal nerves in thirty-four cases has been tabulated by Bergmann (*St. Petersberger Medecin Zeitschrift*, Bd. vii, 1864). "The results of the investigation have not been by any means uniform," says Dr. Roberts, in his Article on Wasting Palsy, in Reynolds' *System of Medicine*, vol. ii, p. 174. "In 16 cases the cord and the nerves were pronounced healthy, and in 6 of these the parts were examined microscopically. In 6 cases the cord itself was found healthy, but there was marked atrophy of the anterior roots of a certain number of spinal nerves. In 1 case both the spinal cord and nerves were healthy, but there existed disease of the medulla oblongata. In 6 cases the cord was found diseased when examined microscopically, though it appeared sound, or nearly so, to the naked eye. Lastly, in 7 cases the cord appeared to the unaided senses palpably softened and disorganized." The cases examined by Mr. Lockhart Clarke are brought forward to show that where the lesions of the cord are not coarse enough to be seen by the unaided eye, the microscope may detect molecular changes. The late Dr. Bazire (Trousseau's *Clinical Lectures*, vol. i, p. 305), remarks that of the three cases examined by Mr. Clarke, one alone seems to have been a clear and undoubted case of progressive muscular atrophy, and that however valuable and interesting the results arrived at may be, they yet cannot be said, when his cases are critically examined, to have settled the ques-

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\* [Neither Dr. Meryon's nor Dr. Roberts's views are exactly stated in the text. See Editor's addition.]

tion at issue—whether this affection is due to disease of the gray matter of the spinal cord. The negative evidence in support of the views of Duchenne, Oppenheimer, Hasse, Meryon, Eisenmann, Friedreich, Friedburg, Cohn, that the cord is not initially at fault, is hardly more conclusive. Dr. Meryon, indeed, looks upon the affection as an affection of the muscular system analogous to that of the osseous system known as rickets, and mentions an instance, in a boy 8 years old, in which the bones seemed to participate in imperfect nourishment of the muscles; but there is nothing to hinder the two diseases from being coexistent.

In 1854 Schneevooft, of the Hague, published a fatal case of progressive muscular atrophy, with the autopsy, in which he found fatty-change of the cervical and dorsal sympathetic. In 1864, Dr. Jaccoud examined two cases, in which there was the same alteration in the external cervical plexus, the superior cervical ganglion, and the filaments which anastomose with the cord. In all those cases atrophy of the roots of the anterior spinal nerves existed, but the cord was *apparently* healthy, for there does not seem to have been any microscopic examination. From these cases Dr. Jaccoud concludes that both the cause and anatomical character of this disorder are in that portion of the nervous system which is supposed to preside over nutrition—the great sympathetic (*Leçons de Clinique Médicale*, Paris, 1867). This pathogenetic view is ably argued by Dr. Jaccoud, and, it must be admitted, that the case-histories of the affection, and analogy, go to support it, and it is to be regretted that more extensive observations in this direction have not been made. In two cases, however, reported by Landry and Baillon, some years ago, where the sympathetic was examined, no apparent disease was found. Bärwinkel, Remak, and Benedikt, have for many years insisted upon this pathogeny of progressive muscular atrophy, but their hypothesis was unsupported by anatomical facts. Trousseau would seem to also favor this view, though on purely theoretical grounds. He says: “A careful study of this disease does not allow me to group it with diseases of the spinal cord; yet I dare not say that it is primarily a disease of the muscles. It is infinitely probable that the nerves of organic life, which accompany the terminal and muscular branches of the arterial tree, are so modified in their functions as deeply to disturb the nutritive phenomena over which they preside” (*l. c.*, p. 302). Dr. William Roberts, in his excellent article already referred to, writes: “The opinion seems to be steadily gaining ground, that the nutrition of the muscles is placed under the control of a special set of organic nerves, having upward connections with the sympathetic ganglia and the cerebro-spinal axis, which are by no means identical with the central connections of the motor nerve-fibres of the same muscles. Assuming the existence of such nutritive centres, all the clinical phenomena of wasting palsy, and the various findings of the post-mortem examinations admit of easy explanation, on the supposition that these centres, or some of their ganglionic connections, are the primary seat of the disease. And the numerous associations and complications can scarcely be accounted for by any other hypothesis” (*l. c.*, p. 179).]

The disease has been observed to follow cases of fever and sun-stroke, severe falls, and blows on the back of the head or spine; and it is well to observe in all cases whether or not the brain has received any accidental shock; and it is probable that several distinct diseases have been described under the name of “wasting palsy” (W. T. GAIRDNER).

**Treatment of wasting palsy**, as laid down by Dr. Roberts, appears

to be chiefly hygienic—namely, *methodical exercise* and *douche baths*, or *cold mineral baths*; so also *thermal* or *sulphur baths*, and *galvanism*. *Frictions*, with stimulating liniments (such as camphor liniment), are also favorably spoken of. *Localized electricity*, in the form known as “Faradization,” has been highly spoken of (DUCHENNE, GROSS, MEYER, ROBERTS, ALTHAUS). “Faradization” consists in the employment of the electricity of the induced or secondary current in the helix round the magnet, originally discovered by Faraday. It ought to be practised at least three times a week, *for from five to ten minutes each time*, and continued at least a month before it is given up, if negative results are only obtained. Every muscle ought to be Faradized in a special manner, according as it has suffered more or less in its electric contractility and nutrition. The power of the current ought never to be strong. When the sensibility of the muscle returns, the intensity of the current may be diminished. Its application should never be protracted beyond ten or fifteen minutes at the most, one minute, on an average, being allowed to each muscle, or distributed over several at a time.

Volta-electric machines are made for medical purposes by Messrs. Stöhrer, of Dresden. Magneto-electric machines are to be got from most of our philosophical instrument makers. The apparatus for applying a continuous current, as modified by Dr. Althaus, is to be obtained of Messrs. Legendre & Morin, of Paris [and, a more recent one, of Weiss, of London].

If, on the other hand, it turns out, as the searching examinations of Mr. Lockhart Clarke would tend to show, that these forms of progressive paralysis or “wasting palsy” are always associated with disease of the spinal cord, the efforts of treatment should, in the first instance, be directed to that part of the cord corresponding to the connections of the nerves with the site of lesions—the line of treatment to be pursued being determined by the general symptoms.

[If this disorder is, as there is good reason to believe, one of nutritional deterioration, the treatment should be constitutional, and a general restorative one, on the principles already insisted on, be employed. Local means can only be helpful. The late Dr. Bazire says: “I have seen a general plan of treatment, the administration of preparations of iron and of cod-liver oil, together with localized Faradization, in some instances arrest the disease for awhile, by improving the general nutrition of the body” (*l. c.*, p. 301). Jakob has published a case of cure by this treatment (1866). Dr. Meryon speaks favorably of arsenic, and Dr. Hetzlar, of Aix-la-Chapelle, says that he has cured several cases with sulphur baths. Two cases are reported by Taylor in which the use of iodide of potassium was followed by much improvement (*Med. Times and Gaz.*, 1863); and Niepce (1853) and Rodet (1859) have each reported successful issues after the use of this drug, but in these cases there was coexisting constitutional syphilis.

Dr. Roberts says: “The most effective remedy is, undoubtedly, galvanism.” With regard to localized Faradization, Duchenne and Gos, of Moscow, speak hopefully of its influence, and Dr. Roberts is convinced that it rarely fails of some good effect when perseveringly employed, and he mentions a case where, after the daily application of the secondary

current, complete arrest of the disease had followed, and continued to last six years afterwards. The application of the constant current,—the positive electrode being placed in the region of the cervical sympathetic, and the negative electrode upon the cervical and upper dorsal regions of the spine—have, in the hands of Remak and Benedikt, been followed by long amelioration, if not by a positive cure (see Translation by the editor of *Lectures on the Treatment of Nervous Disorders by the Application of the Constant Galvanic Current*, by Dr. Remak, New York Medical Journal, vol. iii, 1866; and *Elektrotherapie*, von Dr. Moritz Benedikt, 1868). The rational use of galvanism in this affection, if the views of its pathogeny taken in this article are correct, would seem to be the conjoined or alternate employment of both the constant and faradaic currents, to secure the general and local effect of each, along with the constitutional treatment already indicated.

The cramps and nerve-pains may be controlled, or bettered, by warm alkaline baths, rest, Indian hemp, and hypodermic injections of morphia.]

#### PROGRESSIVE LOCOMOTOR ATAXY—SYN., PROGRESSIVE MOTORIAL ASYNERGIA.\*

FRENCH Eq., *Ataxie locomotrice progressive*; GERMAN Eq., *Rückenmarks Lähmung*, *Tabes Dorsalis*, *Progressive spinal paralysis*, *Degenerative Atrophie der spinalen hinterstränge*.

**Definition.**—A peculiar form of apparent paralysis, characterized by unsteady and disorderly muscular movements, but with muscular power entire, and more or less progressive loss of the faculty of co-ordinating power (voluntary and instinctive). There is sometimes temporary diplopia, with unequal contraction of the pupils. The course of the disease is slowly progressive; and the anatomical lesion is generally a degeneration of the posterior columns and horns of the spinal cord and posterior roots of the spinal nerves, and sometimes with peripheral structure-change in the cranial nerves; and chiefly the second, third, and sixth pairs, in cases where the sight is affected.

**Pathology.**—The disease is one of middle life; most common in the prime of life, between thirty-five and fifty years of age; it is very rare in youth or old age. It is also more frequent, in a very large proportion, in males than females.

The essential anatomical lesions are found in the posterior columns of the spinal cord, the posterior roots of the spinal nerves, the peripheral extremities of the cranial nerves, and, exceptionally, in those of the extremities. The membranes of the cord are generally unaffected or merely congested; sometimes thickened posteriorly by exudations, adherent to each other and to the posterior surface of the cord. The degeneration and atrophy of the posterior columns of the cord cause the cord to seem flat in its antero-posterior diameter. The posterior median fissure is effaced or marked only by a whitish line. The degeneration consists of an atrophy with disintegration of the nerve-fibres (gelatiniform degeneration of Cru-

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\* From *a*, privative, and *συνεργία*, joint-work, co-operation (BAZIRE); and Dr. Meredith Clymer suggests *motorial* in place of *locomotor*.

veilhier and Virchow), with proliferation of the connective tissue, giving to the columns a gray transparent aspect. *Corpora amylacea* are found embedded in the connective tissue. The bloodvessels which traverse the columns are loaded on their external coats, and surrounded to a variable depth with oil globules and granules of various sizes. The *dorso-lumbar* region of the cord is the most constant site of lesion, which may involve one or two inches, to nearly the whole extent; and may extend in depth to the surface of the lateral columns, and occasionally even along the edges of the anterior columns. Localized areas of some widely-spread degeneration have even affected the extremities of the posterior cornua and deeper parts of the gray substance (Lockhart Clarke, *Lancet*, June 10, 1865). The posterior nerve-roots, both within the cord and after leaving its substance, are similarly involved in the degeneration, and the general progress of the disease in the cord seems to indicate a central commencement advancing to the periphery; but in the cerebrum the phenomena indicate an opposite course—namely, affection of the peripheral distribution of the first, second, third, fourth, fifth, sixth, and seventh nerve, travelling to the centres. From the retina and optic nerve it has advanced as far as the *corpora geniculata*, and even to the *corpora quadrigemina*. The eighth pair seems the only cranial nerve that has not yet been implicated. The condition of the sympathetic is unknown.

These anatomical lesions (so exactly limited to the posterior columns of the cord, and the roots issuing therefrom) by no means account for all the symptoms which are clinically recognized as distinctive of this disease. The nature of the peculiarly characteristic gait is sometimes referred for explanation to the loss of the “*muscular sense*” of Bell, or the loss of “sense of muscular activity” of Gerdy. The incoordination is essentially a reflex disorder; and the lesions involve the coordinating centres (posterior column of spinal cord) of certain muscular acts, such as walking and standing. Some of the phenomena of the disease suggest involvement of the sympathetic—*e.g.*, the vesical phenomena, also the phenomena of irregular contractions of the pupils, and their becoming dilated during paroxysms of pain, or when the legs or arms are pricked or pinched, with local perspiration increased in some parts only of the skin, *e.g.*, forehead, palms of hands, and fingers\* (BAZIRE).

The disease, then, is a peculiar one of the nervous system, commencing insidiously with evidence of disorder of some of the *cranial* nerves, perversion of sensibility in different parts of the body, ultimately giving place to incoordination of motion—always associated with degeneration of the posterior columns of the spinal cord and posterior nerve-roots, but without loss of muscular power or impairment of the intellect. The connecting links between the initial disorders of the cranial nerves (advancing centrally) and

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\* Dilatation of the pupil is known to result from disease of the *third* cranial nerve or its roots; section or lesion of the sympathetic in the cervical portion; intra-vertebral section of the two upper dorsal nerves, followed by irritation of the peripheral ends of the cut roots.



the lesions of the spinal cord (progressing peripherally) have not yet been made out.

In addition to these characteristic post-mortem lesions, there is evidence during life, both at the commencement and throughout the course of the disease, of localized congestions, expressed by rachalgia and spinal tenderness.

**Symptoms.**—The outset of the disease is insidious and slow. Pains, generally ascribed to rheumatism or neuralgia, first attract attention, particularly in damp weather. These pains may be fixed and aching, or darting, piercing, and transitory. Affections of the *second, third, fifth, and sixth* pairs of cranial nerves, sometimes with headache, giving rise to imperfect or double vision, strabismus, or contractions of the pupil, or dilatations, or deafness, are also insidious phenomena which may attract early notice. These several phenomena generally appear at different times and singly, although sometimes several together; and, after persisting for some time, may pass away. Weakness in some of the limbs may supervene, but no actual paralysis. Another distressing harbinger of the disease is incontinence of urine, associated with spermatorrhœa during the night, with a great proclivity to sexual congress, which is no mere impotent desire, but results in effective sexual intercourse. But after a period (of perhaps two years) the emissions on sexual intercourse become hasty, and a gradual loss of sexual desire and the power of erection supervenes. The bladder becomes irritable. Constipation is occasional when the pains are severe, and it always aggravates them. Transient tickling sensations prevail in different parts of the body, such as the lips, nose, cheeks, forehead, with occasional numbness of the feet and arms, and the peculiar feelings of so-called “pins and needles, as if they were asleep.” After another long interval (say of two more years), undue fatigue after walking is experienced: the legs give way under the weight of the body, and there is a sensation as if the patient walked on a spring-board and could not keep his balance. These feelings may disappear and return; and eventually cutaneous hyperæsthesia or neuralgia of the skin (one side of the scrotum, testicle, buttock, or external part of thigh), always unilateral, supervenes.

Motorial incoordination may not come on till after a period of several years (four or five) of suffering from such nervous symptoms. The pains then occur very irregularly, and last from a few hours to many days. At times they dart from limb to limb, or from one part of a limb to another; or, fixing on a small circumscribed spot, they give a boring, gnawing, or tearing sensation. The pains leave behind them a stiffness and soreness of the part. There may be atrophy of the optic disc, with contracted pupil; cutaneous anæsthesia of the soles of the feet, legs, forearms, and lips; and tactile sensibility seems slowly conducted to the sensory centre. Reflex movements are not excited by tickling the soles of the feet. A painful feeling of constriction, as if by a tight band, is sometimes experienced round the body; and the sensation of “bearing down,” in the perineum and rectum, with constipation, prevails. The bladder imperfectly empties itself, and cannot retain the urine after

experiencing the desire to void it. The desire to pass it is frequent and imperative, especially during the night. Some degree of paralysis of the bladder is indicated by diminished force in the stream of urine—the jet is not well arched, and drops dribble away after the act is believed to be over. The urine sometimes passes involuntarily. Sexual desire is eventually abolished. The gait is characteristic. The steps are quick, short, and jerking. The leg and foot are well lifted from the ground, but they are thrown spasmodically and forcibly forward, the whole limb being extended. In bringing the foot down, the heel strikes the ground first. As the disease progresses, the limbs are thrown involuntarily to the right or left without purpose, and without the power of restraining in any way their irregular movements. In walking, the eyes are kept fixed on the legs; and a stick is used, over the handle of which a handkerchief may be placed to increase the surface of contact required by the numbness of the hand. The muscular force remains good, so that efforts to bend or extend a limb against the will can be resisted with strength. Involuntary jerkings of the limbs in bed prevent sleep. It is especially at starting that the patient has the greatest difficulty in maintaining equilibrium.

When muscular incoordination of the upper extremities supervenes, the fingers become numb, and objects are handled with increasing clumsiness. The clothes cannot be buttoned, nor small things removed from the pockets by the fingers. If the patient is set on his legs with his eyes closed, and his feet close together, although he has muscular power to stand, yet he cannot preserve his body from falling, or guide himself in taking a few steps in the dark or with his eyes shut. He has no idea of the position of his lower extremities except from sight.

The prolonged course of the disease is characterized by frequent natural pauses in its progress, sometimes lasting for a long time, before its onward progress is resumed—a point to be borne in mind in the treatment of the disease.

Electro-muscular sensibility is generally lessened; and during the early stages of the disease there is neither paralysis of muscles, nor wasting of their substance, nor defective *nutrition* of the limbs. The patient may be strong enough to bear and to carry considerable weight; so that there is not muscular weakness, far less paralysis, in the ordinary sense of the term. The seeming paralysis is entirely due to the peculiar deficiency in the power of co-ordinating voluntary movements. The duration of the disease ranges from a few months to thirty years, with a mean duration of *seven* years in 119 cases whose progress has been observed (Dr. CLYMER).

**Diagnosis.**—In the early stage of the disease the differential diagnosis is between intracranial lesion and the peripheric lesions of the cranial nerves which characterize the commencement of progressive locomotor ataxy. Here the ophthalmoscope becomes of use. In the amaurosis of intracranial disease there is always evidence of recent or past *neuritis* of the optic disc; but such is not the case in this disease (H. JACKSON). Care must also be taken not

to confound the disease with progressive muscular atrophy, already noticed, p. 492.

**Prognosis** is decidedly unfavorable. The lesions may remain stationary for long periods, but recovery never takes place, and eventually the disease progresses onwards to death. Of forty-three fatal cases, the immediate cause of death in *six* were lesions of the brain or spinal marrow, with softening, hemorrhage, and progressive muscular atrophy. Three died of acute inflammation of the urinary organs, and four from extensive sloughs over the sacrum. Thirty died during the course of intercurrent diseases, not directly connected with the motorial asynergia—namely, thirteen of pulmonary consumption, three from typhoid fever, four from pneumonia, others from pericarditis and dysentery.

**Causes.**—The only positive determining causes seem to be prolonged exposure to damp, cold, depression of the nervous system from insufficient diet; mental exhaustion, trouble, and anxiety; venereal excesses, especially onanism. If there be an hereditary history of various nervous diseases, *motorial asynergia* may be connected with those diseases, and ascribed to a common origin.

**Treatment.**—The intercurrent localized congestions in the region of the spinal cord point to topical bloodletting by cupping or leeches, cautiously employed, and perhaps frequently repeated; with persistent counter-irritation over the spine (by *blisters*, *moxæ*, *actual cautery*, or *ice*), particularly over those parts where there is tenderness or pain. *Phosphorus*, in the form of *phosphates of metals* and *salts*, of *diluted phosphoric acid*, as a drink in the daily allowance of water, of *phosphate of soda* as an aperient, may be given with benefit. *Nitro-muriatic acid* as a tonic, and *cod-liver oil* as a dietetic agent, may also be of use. Flannel should be worn next the skin, and chills from cold and damp carefully avoided by the use of a chamois leather, close-fitting jacket over the flannel, and reaching from the height of the clavicles as low as the folds of the *nates* behind. The diet ought to be of the most nutritious materials which the patient can digest. *Cannabis Indica* and *bella-donna* give the greatest relief to the pains.

So long as there is active local hyperæmia the use of electricity is hurtful. Faradization may be of use in restoring to some extent sensibility to the skin, where local cutaneous anæsthesia has prevailed—after which patients seem to walk better for a time; but when used at all, electricity ought only to be employed during the pauses in the course of the disease, and then with great caution, in the form of a constant current rather than the induced current, or Faradization.

[Recently the *phosphuret of zinc* has been given by Dr. Gueneau de Mussy, and other Paris physicians, with advantage, some of the symptoms having considerably lessened under its use. The writer has lately had some evidence of the ameliorating influence of *ergot* in this affection.]

GLOSSO-LARYNGEAL PARALYSIS—*SYN.*, GLOSSO-PHARYNGEAL PARALYSIS.

**Definition.**—*Diminution and subsequent loss of the motor power of the tongue, soft palate, and lips, associated with structural changes in the roots of the motor nerves which supply the affected muscles. The disease progresses always rapidly to a fatal termination.*

**Pathology.**—Concurrent paralysis of the tongue, palate, and vocal cords, together with the associated muscular movements of deglutition, is more or less capable of explanation by the close anatomical connection between the muscles supplied by the *vagus*, the *spinal accessory*, and the *lingual* nerves. The *lower* rootlets of the spinal accessory nerve (forming the external branch) arise, in common with the anterior roots of the spinal nerves in the cervical and brachial region, from the anterior gray substance of the spinal cord; while the *upper* rootlets (forming the internal branch) have a totally different and a double origin—one from a special nucleus continuous with that of the pneumogastric behind the central canal, and the other from the proper nucleus of the hypoglossal, in front of the canal. Some of the fibres of the hypoglossal seem to take their origin from the spinal accessory nucleus (LOCKHART CLARKE, *Beale's Archiv.*, No. 3).

The constant anatomical lesion in the paralysis under consideration is *atrophy of the motor roots of these nerves, sometimes extending to the anterior roots of several of the upper spinal nerves, attended with more or less paralysis of limbs and incipient muscular atrophy.*

Glosso-pharyngeal paralysis and progressive muscular atrophy are found to be attended with the same nerve-lesions—namely, atrophy of motor roots of nerves, cranial as well as spinal; and in all patients suffering at first from glosso-pharyngeal paralysis there is a tendency for the paralysis to become general (TROUSSEAU). Gradual disappearance of the nerve-elements, and proliferation of connective-tissue from the neurilemma, are the characteristic anatomical lesions at the roots of the affected nerves.

**Symptoms.**—The earliest and most noticeable are those which are due to palsy of the muscles of the tongue, the soft palate, and the lips, those of the larynx and pharynx becoming implicated at a later period. The origin, progress, and termination of the disease are so characteristic that, according to Trousseau, there is no other identical affection in the whole range of nosology. Embarrassment of speech first attracts attention. The tongue seems less supple, and the utterance becomes more and more thick. The food is apt to lodge between the teeth and the cheek, the cause of this being different from that which obtains in Bell's paralysis. In such facial paralysis it is due to paralysis of the buccinator muscle; here it is due to the circumstance that the tongue being more or less paralytic, awkward, and incapable at the tip, the patient is obliged to use his fingers to remove the food from between the teeth and the cheek, and so replace it on the tongue. Pronunciation of certain words is made through the nose. The vowels *o* and *u* cannot

be properly sounded, on account of the deficient contractility of the *orbicularis oris* muscle. Saliva is apt to dribble from the lips and corners of the mouth. The paralysis continuing to progress, the tongue at last lies motionless in the hollow of the mouth, behind the lower teeth. Its apex and base are equally motionless, and not a word can be articulated. The shape of the tongue is also altered. It has sunk down in the centre, presenting a hollow in the middle line, with its edges raised. The soft palate also droops, and the tip of the uvula rests upon the tongue, and is generally callous or insensible to irritants. The first stage of deglutition thus becomes impossible. The morsels are swallowed by holding the head backwards, and facilitating their gliding down by fluids. Sometimes only a small quantity of the food gets into the œsophagus, the remainder being propelled upwards through the mouth and nostrils, and sometimes small portions of food will find their way into the larynx, causing great distress. The appetite remaining good, but swallowing being thus impossible, constant hunger aggravates the distress (Trousseau).

Excessive weakness of the respiratory movements is soon super-added to these already serious symptoms. The walls of the chest and the diaphragm scarcely move. If the patient be asked to blow out a candle, he cannot do it. The flame will be scarcely agitated by his utmost efforts to blow upon it. Coughing is equally inefficient; so that if catarrh should supervene, there is great difficulty in expelling the increased secretion of mucus. Asphyxia is thus apt to prove fatal. The heart's action becomes abnormally rapid, but fever does not exist. The body-temperature tends to sink below the normal, and betokens, with the other phenomena, the imperfect oxygenation of the blood. General debility now makes rapid progress, and the patient rather inclines to remain in bed, sitting up with his head supported on pillows, inclined to one side sufficiently to allow of the saliva flowing away which he is now unable to swallow. Sleep is disturbed by paroxysms of suffocation, and death is apt to ensue suddenly by cessation of the heart's action, unaccompanied by pain or noise (Trousseau).

**Diagnosis.**—From the general paralysis of the insane it is distinguished by the intellect of the patient remaining perfectly clear; and the gravity of the complaint impresses itself on the mind. The convulsive movements of the lips are also absent. In cases of hemiplegia, the palsy is unilateral. In double facial paralysis—a rare affection—the movements of the tongue are free, and deglutition remains efficient, all the muscles of the face are paralyzed, so that expression is as blank and fixed as marble, the patient laughs or cries as if from behind a mask (Duchenne); but in glosso-laryngeal paralysis the lower part of the face alone remains motionless.

**Prognosis** is unfavorable. The disease has always proved fatal. Its progress is generally rapid and continuous. From three to six months it may not seem to make so rapid a progress as it does later; but as soon as deglutition becomes imperfect, death is rapid.

**Treatment.**—Faradization is of doubtful efficacy at the commence-



ment, but gives relief at the later periods, by temporarily restoring function to the affected muscles, to the lessening of the trouble of deglutition, and by exciting the respiratory acts.

[PROGRESSIVE GENERAL PARESIS.—GENERAL PARESIS.—  
GENERAL PARALYSIS OF THE INSANE.]

LATIN, *Dementia Paralytica*. FRENCH, *Paralysie Générale des Aliénés*; *Delire des Grandeurs*. GERMAN, *Allgemeine Progressive Paralyse der Irren*.

(DR. CLYMER.)

The first notice of this disorder as a special disease is by Bayle (*Traité des Maladies du Cerveau et de ses Membranes*, Paris, 1826). Calmeil's classical work (*De la Paralysie considérée chez les Aliénés*, 1826) was published about the same time, and gives an admirable account of the symptoms, and, within certain limits, of the true pathogeny of the disorder. Dr. Sankey has shown, however, that Dr. Thomas Willis, who wrote in 1672, clearly described it (*De Animâ Brutorum*, cap. ix, p. 281). Requin (*Eléments de Pathologie Médicale*) has written an excellent article upon it, and also Baillarger. The reader will find the subject also well treated of by Dr. Sankey, in his *Lectures on Mental Diseases*, 1866, and in a compendium of our present knowledge on the subject by Dr. Westphal, of Berlin, translated and published in the *Journal of Mental Science*, July, 1868. The term *general paresis* (*παρεσις*, *relaxation, want of strength*), has been proposed and adopted by some writers (ERNST SOLOMON, C. H. JONES, SANKEY), as preferable to that of *general paralysis*, actual motor paralysis not properly existing at any time in the disorder, but only muscular debility.

This form of *progressive paralysis* is constantly met with in this country, and is believed to be on the increase. The access may be sudden, immediately preceded by intense energy of mind and body, violence of manner, and increased body-temperature (see p. 419 *ante*), soon to be followed by commencing imbecility, and incipient paralysis of speech and limb (BLANFORD). More frequently the invasion is gradual, the mental feebleness and muscular weakness being so slight as scarcely to attract attention. In whatever way it begins, however, when sufficiently well-marked to be observed, there is a feeling of weariness of the lower extremities, the patient becomes tired after little exertion, and has a peculiar gait, which is very characteristic; it is walking to and fro, without a definite object—a kind of “pacing the deck”—with a certain expression of restlessness in it. As the disorder advances there is more attention paid to the walk, and the patient moves with caution and studied attention, looking neither to the right or left, the head poised, and the centre of gravity carefully kept. There is no elasticity in the step; the foot comes down flat upon the ground; the legs are thrown somewhat apart; and the steps are short and shuffling. At the same time, and often for some time before, hesitancy in the articulation of a word, or of only a single letter, particularly labials, will be noticed, from want of perfect co-ordination of the muscles of the lip, which are a little tremulous, and a feeling of stiffness in them may be complained of. Later the speech is drawling or mumbling. In attempting to speak, the corners of the mouth are often spasmodically drawn down; the head is shortly and sharply nodded, as if to extricate a letter, the upper lip twitches or quivers, and the chin purses.

The facility of articulation varies very much at the beginning of an at-

tack, being more clear and free at one time than at another, and being often, to a certain degree, under control, and words thickly spoken in a common tone, may be clearly enunciated if shouted out. The difficulty is progressive, and the speech soon becomes thick like that of a drunken man, until articulation is impossible. If the patient is asked to put out the tongue, the hand is often as it were, involuntarily raised towards the head, the mouth only is opened, or if the tongue is protruded, it is done in a jerking way, and it is very tremulous. When erect, the weight is poised on both legs equally—there is no “standing at ease”; the position of the arms and hands is constrained; sitting is square and graceless, the head slightly pendent, the thighs parallel, and the knees bent at just a right angle, each hand resting on a knee, or on the elbow of the chair (SANKEY). There is a mask-like, doughy expression of the face.

In some cases the motor troubles last for some time without any mental disturbance (BAILLARGER, REQUIN). Generally, however, the mental phenomena are perverted and the patient suffers under some delusion, often of a peculiar kind—as the idea of some good fortune, as great wealth, high birth—with exuberant animal spirits, contentment, and good humor, except when he is thwarted or contradicted, which brings on an outbreak of ungovernable passion. There is great fickleness and irritability of temper, and no consistency in the views held and expressed from day to day. The delusion may be of a suspicious character, with continual fear of assassination, or of some injury. Settled deep melancholy has been occasionally noticed. More frequently, when the invasion is gradual, the mind slowly weakens; it cannot be steadily applied; there are feeble will and purpose; the memory is treacherous; words are omitted in speaking and in writing, or sentences repeated; and there are fits of absence. The pupils may be at first contracted, with subsequent irregularity, the iris not forming a true circle, and the edge, occasionally, folded in. The irides may show different degrees of excitability in contracting and dilating, or the irregularity may be initial. More or less anæsthesia probably exists, and impairment of tactile sensibility; the handwriting is altered; needlework is clumsily done; there is more or less fumbling; a thread is seized with difficulty, and then dropped; indeed all acts of delicate manipulation requiring a keen sense of touch are imperfectly executed. Patients fall about, and knock themselves, without apparently feeling pain, and later pass their hands clumsily over their faces, or pull their features (SANKEY).

In the first and second stages the appetite and digestion, as a rule, are good, and the patient has the appearance of being well-nourished.

The body-temperature of general paralysis has been studied by Dr. T. S. Clouston (*Journal of Mental Science*, April, 1868). Of 14 cases in every stage of the disease, ten observations being taken in each, the average mean of the morning temperature was 97.37°, and of the evening temperature 98°—a difference of .7°. The temperature is high in the first stage of the disorder, lower in the second, and again, usually, very high in the third stage. The evening temperature is most increased as compared with that of the morning in the third stage, and least in the second stage:

	Morning.	Evening.
1st stage, . . . . .	96.5 . . . . .	97.52
1st stage, . . . . .	98.0 . . . . .	98.14
3d stage, . . . . .	99.6 . . . . .	99.9
3d stage, . . . . .	98.5 . . . . .	100.7
3d stage (moribund), . . . .	102.5 . . . . .	103.5

After the disorder has continued for some time, epileptiform and apoplectiform attacks are apt to happen.

Its march is from bad to worse, but not always steadily progressive, as there are often remissions in the symptoms, leading to deceptive hopes of recovery, though the irritability of temper and weakness of memory rarely improve; but after any mental effort the symptoms recur with increased severity, and there is a gradual decline of bodily and mental power, ending in complete imbecility and paralysis. The excito-motory system becomes gradually affected, the first evidence being imperfect deglutition, the mouth being filled and the food kept there, or rolled from side to side. It may get impacted in the pharynx, or fall into the larynx, and choke the patient. The conjunctiva is covered with secretion, and its vessels are turgid. Reflex action is weakened, shown by the want of muscular contraction on tickling the soles of the feet. Electro-irritability of the muscles is lessened or absent (DUCHENNE, BRIERRE DE BOISMONT). The power of utterance is lost, or is very imperfect; the face is stupid or vacant; there are constant muscular twitchings, with restlessness, even when the sufferer is lying on his back. A propensity to gather up the bedclothes and roll them over, is very common in the third stage. All instinct of decency is lost. The special senses are impaired or gone.

Death may happen from exhaustion, asphyxia,—when the respiratory muscles are invaded,—meningeal apoplexy, during an epileptiform seizure, or from some intercurrent disease or accident.

Calmeil asserts the average duration of the disease to be thirteen months, and Esquirol says few who are afflicted with it survive three years; but it undoubtedly may last much longer.

**Causes.**—Patients already suffering from insanity do not seem liable to this affection; the mental trouble is coincident with, or subsequent to, the motory-paretic disorder. It is a disease of the prime of life, when the mind and body are in the fulness of their vigor, the most common period being about thirty-five years of age. It is rarely met with under twenty-five, seldom after sixty, and never after seventy years. Males are much more liable to it than females; according to Blanford, in the proportion of 50 to 15. It is said never to be met with among women in the higher walks of life. The degree of liability is 1. Males of the lower classes; 2. Males of the upper classes; 3. Females of the lower classes; 4. Females of the upper classes. Dr. Sankey found hereditary predisposition in 14½ per cent. The causes would seem to be, prolonged over brain-work, probably combined with sexual excesses. Dr. Sankey says, of those affected with it, we find chiefly such who have led a fast life—reckless imprudent individuals, swayed through life by their lusts and passions.

**Prognosis.**—Death is the common termination. Baillarger has collected nine cases of recovery, but some, at least, are of doubtful nature. When the issue is happy, Dr. Sankey says that the patients have apparently been cured by critical formation of boils, or of abscesses. Dr. W. Dornett Stone has reported in full an authentic case of this affection in the second stage, which got well, under abstinence from mental work, good diet, constant exercise, varied amusement, diversion of the mind, and a restorative drug treatment—iron, cod-liver oil, phosphorus, nux vomica, &c. (*The Lancet*, February, 1867.)

**Diagnosis.**—Progressive general paresis has some symptoms in common with 1. Progressive muscular atrophy; 2. Lead and mercurial poisoning; 3. Some forms of palsy from cerebral and spinal disease; and 4. Chronic alcoholism. From the three first, the history of the case, and the absence of characteristic phenomena, particularly the special form of mental

trouble, should prevent an error of diagnosis. In chronic alcoholism the diagnosis is sometimes more difficult in respect especially to the motor troubles, particularly if the two affections, as it may be, are coexistent. In chronic alcoholism hallucinations are constantly present, but are rare or wanting in progressive paresis. The delusions in the one are a dread of some persecution or bodily harm, with a tendency to suicide, and a sense of shame and degradation, and timidity; in the other elation, a joyous state of mind, extravagant notions (*monomanie des grandeurs*)—constant mental depression in chronic alcoholism, and a constant mental exaltation, at least in the first two stages, in progressive general paresis. The tremor of the upper extremities in drunkards is wanting in the paretic who suffers from incoördination-troubles in his arms and hands. The gastric symptoms are widely different, the appetite in paresis being good, and digestion excellent. The pupils of the paretic are irregular; in the sufferer from chronic alcoholism they are nearly always dilated. Thermometric examination will also serve as a diacritic sign, the body-heat being always high in paresis (see *ante*, p. 50). The mask-like and stolid expression of face of the confirmed drunkard resembles very much that of the subject of general palsy.

**Patho-anatomy.**—Griesinger says: The appearances found in paralytic mania and dementia are more markedly characteristic than in any form of mental disease. Yet though coarse structural alterations of the brain substance—as discoloration, hardening, &c.—are frequent, still they are not constant, but vary in kind in different cases, and no essential morbid gross lesion seems to have been as yet made out.

According to the microscopical observations of Dr. Franz Meschede,\* this disease is a parenchymatous inflammation of the cortical structure of the hemispheres, beginning in the inner layer, and spreading from thence—a slow inflammatory(?) degeneration and destruction of the brain-cells. In acute cases, running a rapid course, there are hyperæmia and parenchymatous swelling of the inner layer—a congestive turgescence and succulence. The inner layer has a bright red look, sometimes extending to the surface, and this is chiefly seen in the anterior lobes, on the convexity, and temporal lobes. The capillary network is greatly developed, with points of extravasation, the vessels being tortuous and elongated. In more chronic cases this layer will appear dark, dull, and yellowish; its consistence is generally firmer than in the normal state, owing to the shrinking of the tissue on the destruction of the cells, and from condensation of the connective tissue and wasting of the vessels; sometimes it is softer. The cells are in different stages of degeneration; at first turgid and swollen, they afterwards appear as aggregations of fat-globules, with the outline and nucleus of cells; or, with half the cell outline, the rest being composed of fat and pigment granules; or their outlines may be entirely lost, and they seem mere heaps of granules. These researches confirm Calmeil's theory of the pathogeny of the disease, as shown by the name he gave to it—*périencéphalite chronique diffuse*†—and is probably the right one, if for the term inflammation one could be substituted which would indicate one of those processes of degeneration which modify and degrade texture.

Mr. J. Lockhart Clarke has found in vertical sections of the cerebral

\* [Virchow's *Archives*, 1865. *Journal of Mental Science*, October, 1866. Blanford's *Lectures on Insanity*, London Med. Gaz. and Times, vol. ii, 1866.]

† [Calmeil, *De la Paralyse considérée chez les Aliénés*, 1826. *Traité des Maladies Inflammatoires du Cerveau*, 1859.]

convolutions of those who have died from general paresis a series of streaks or lines radiating through the white and gray substances towards the surface; and in sections hardened in chromic acid it is common to find, in the white substance especially, what seems to be a number of vertical fissures and oval slits, but which under the microscope are found to contain bloodvessels surrounded by secondary sheaths, first described by Robin (*Jour. de Physiologie*, t. ii), which are thicker, darker, and more conspicuous than in the healthy brain. In the brain of paretics he found the hematosin granules abundantly scattered, or collected in groups. Mr. Clarke also insists on a considerable increase of the number of the contained pigment granules, which, in some instances, completely fill the cell. Sometimes the cell loses its sharp contour, and looks like an irregular heap of particles ready to fall asunder. (*The Lancet*, vol. ii, 1866.)

Dr. Sankey, who has investigated the coarse and microscopical anatomy of this disorder with care, writes: "My own opinion is that as yet we are not in a position to demonstrate any distinct and unequivocal alteration in the nerve-elements or nervous structure of the brain in this disease. I think that Rokitansky's views of excessive growth of connective tissue may have a solid foundation, though the power of demonstrating them is not yet satisfactory. . . . My own examinations of the capillaries lead me to the conclusion that the capillaries of the cortical substance are more or less diseased in every case of general paresis. . . . The presence of hyaline around the capillaries, and the frequent appearance of something like fibres traversing the cortical substance, appear to be due to an excess of connective tissue. . . . It is highly probable that the hypothesis of Rokitansky and Wedl is correct relative to the formation of connective tissue from a material thrown out by the capillaries, viz., that in the first stage this material is hyaline and afterwards contracts; that in contracting it throws the capillary into kinks; that as it goes on contracting it becomes less hyaline, more fibrous, and at length like a sheath; but if converted into fibres at all, it has no share in the formation of the fibrous condition of the brain-substance described by Rokitansky" (*Lectures on Mental Diseases*, p. 174-5-6-7).

**Nature.**—Dr. Skae (*Ed. Med. Jour.*, vol. v) says, "This disease may be described either as a form of insanity complicated with general paralysis, or as a general paralysis complicated with insanity. Calmeil, Esquirol, Georget, Pinel, even Griesinger and the German school, look upon the paretic symptoms as epiphenomena,—a mode of termination of mental disorder; whilst the French authorities of the day regard general paresis as a distinct morbid species." Dr. Sankey thus sums up the argument in favor of the last view: "I think the evidence all tends to prove that general paresis differs essentially from other forms of mental disease; whether we view the symptoms during life or the appearances after death, the cases have well-marked characters peculiar to themselves. The symptoms connected with the mental functions are distinct, as also those of the bodily functions; the mode of attack is distinct; the persons liable to the attack, the course and duration of the phenomena, all differ. The morbid alterations after death have strongly marked characters also. . . . It is more in accordance with our present knowledge, to call general paresis a distinct species rather than a variety of mental disease" (*l. c.*, p. 183).]



## [ACUTE CENTRIPETAL PARALYSIS—ACUTE CREEPING PARALYSIS.]

FRENCH, *Paralysie Ascendante Aigue; Paralysie Extenso-Progressive.*GERMAN, *Acute Aufsteigende Paralyse.*

(DR. CLYMER).

Another form of general progressive paralysis, unattended, however, with any mental disturbance, has been described by Ollivier (d'Angers), Cruveilhier, Landry, Kussmaul, Liégard, Pidoux, Duchenne (de Boulogne), Levi, and others. Landry calls it *acute creeping paralysis*, from its rapid progress; and Dr. Pellegrino Levi† has proposed the name of *Centripetal* or *Acute Extenso-progressive, Paralysis*. The celebrated Cuvier perished by it.

**Symptoms.**—There is a premonitory period, lasting from a few days to several weeks; the prodromata are tingling in the fingers and toes, weariness after slight exertion, feebleness of the lower extremities, and, sometimes, heaviness in the head, and great drowsiness in the evening, but no cephalalgia. The paralysis of the lower extremities makes suddenly rapid progress, and there is complete paraplegia. In exceptional cases, and Cuvier's was one of them, the muscles of the pharynx and œsophagus are initially attacked, and dysphagia is the earliest symptom. A few cases have been observed, in which the upper extremities seem to have been first affected. In the ordinary form of the disorder, the arms become implicated soon after the lower extremities; then the diaphragm, and other respiratory muscles, and the muscles of the pharynx; dyspnoea increases, the epigastrium sinking during inspiration; there is dysphagia, the deglutition of solids, and even in some cases of liquids, causing great distress. According to Landry the motor system is usually invaded in the following order: 1. The muscles of the toes and feet, then the posterior muscles of the thigh and pelvis, and afterwards the anterior and internal muscles of the thigh. 2. The muscles of the fingers, the hand, and those of the arm which arise from the scapula, and then the flexor muscles of the forearm. 3. The muscles of the trunk. 4. The respiratory muscles, and finally those of the pharynx, œsophagus, and tongue. These symptoms may vary from day to day. No pain in the vertebral column is complained of, and none is elicited by pressure. The muscles seem to retain their sensibility, and in some cases this is increased. At no period of the disease are there muscular spasms, or tremor. Reflex movement is diminished or abolished; electro-muscular contractility in some cases would seem to be unimpaired, though Duchenne declares that it is always abolished, and thus establishes a differential diagnosis between this affection and general paralysis of the insane. General sensibility is sometimes only slightly diminished, but never to the extent of motility. The special senses are not at all, or but slightly affected; binocular diplopia was noted in one case; the speech is at times a little thick, and there may be embarrassment in the movements of the tongue and lips, but there is no accompanying tremor. The intellect is good, and memory unimpaired. There are anxiety and sleeplessness. Constant and obstinate constipation is present, but micturition is easy. Decubitus dorsal, with total muscular relaxation of the lower extremities. The pulse never rises above 100, and is often less; the body-temperature is said to be natural, but no reliable thermometric observations have yet been made; profuse general perspirations are common.

The ordinary course of the disease is rapidly towards a fatal end,

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\* [Archives Générales de Médecine, Feb., 1865.]

death occurring by asphyxia from paralysis of the respiratory muscles, in from four to ten days. Landry has reported one case of recovery (1852). Exceptional cases have been reported, presenting many of the symptoms of the disorder, where there has been a temporary arrest of the symptoms, with recurrence after several months, and subsequent fatal termination.

**Causes.**—In 16 cases 4 were females, the oldest of whom was 31 years of age. Most of the reported cases were between 20 and 30 years; still it has happened in young children, and in persons of over 60 years (CAUSIN, *Gaz. des Hôp.*, 1866). In several cases it has quickly followed sudden exposure to cold whilst the body was overheated.

Of its *pathogeny* and *morbid anatomy* we know nothing, the naked eye and microscope failing to detect any lesion of the spinal marrow or brain. In two cases examined by Dr. Ed. Monneret there was considerable hyperæmia of the spinal cord (*Traité de Pathologie Interne*, t. i, p. 193).

Dr. Jaccoud has lately (*Leçons de Clinique Médicale*, 1867) called attention to another rare form of progressive palsy, which he has called Progressive Nervous Atrophy (*Atrophie Nerveuse Progressive*). Its access is sudden, with numbness and dull aching in the lower and upper extremities. In a little while the fingers become clumsy, and afterwards the hands and forearms are heavy, and motion is difficult or impossible, and wrist-drop may happen. The lower extremities are soon implicated, and the patient stumbles, or falls about, until he is unable to walk. The muscles of the upper and lower extremities atrophy rapidly; reflex movement is lost; and electro-muscular contractility abolished in the affected muscles. Anæsthesia is marked over the regions to which the damaged nerves are distributed. In such cases the brain, cranial nerves, and spinal cord are found healthy, but some of the spinal nerves at their roots and in their course have undergone granulo-fatty degeneration.

Jaccoud (1864), and Duménil, of Rouen (1866), have each reported cases where the changes have begun in the peripheral extremities of the nerves, followed by paralysis (*Contributions pour servir à l'histoire des paralysies périphériques, et de neurite ascendante*, *Gaz. Heb.* 1866).]

## APHASIA.

**Definition.**—*Inability to communicate thoughts, ideas, or names of things, either by words or by writing, the intellect being more or less perfect otherwise, and the organs of phonation free of paralysis. The affection may be associated with unilateral paralysis, or spasms, and be subsequent to apoplexy. There may be disease of the third left frontal convolution, or in the left anterior lobe of the brain, near to this convolution, and various other lesions of no constant kind.*

**Pathology.**—Every shade of deficiency or inability characteristic of this affection is to be met with, from temporary loss of memory of certain words, the names of common objects, and their misuse in conversation, up to persistent and complete inability to give expression by words, writing, signs, or gestures, to thoughts or ideas. The non-expression of the names of things, or the substitution of wrong ones, is among the most common forms of this inability. “Hand me the — to stir the —.” “Poker” and “fire,” are the words which are incapable of being spoken. “Give me my — ;

you know," pointing to his head. "Your hat?" "Yes, my hat." Many other examples are given by Trousseau. The substitution of one word for another is another form of this affection—*e. g.*, "boots" for "head." Forgetting your own name, or the name of the person you go to visit, is another form. In another series of cases there is a sudden loss of the general faculty of speech, without paralysis or incapacity of thought. In a third set of cases there is unilateral palsy or unilateral spasms; and sometimes it occurs after an attack of apoplexy, or after an attack of severe typhus or typhoid fever.

In some cases, however, the thought or idea which leads to verbal expression, as Dr. Gairdner shows, is not absent, so that the faculty remains of communicating by the usual written signs.

The several theories of localization of the brain lesion believed to be present in cases of aphasia are not capable yet of positive demonstration. The disorder may exist in any of the typical forms, and yet no appreciable change be detected in any part of the cerebral substance or its bloodvessels. The following are the general results of *clinical* and *post-mortem* investigation: (1.) In a large majority of cases of aphasia the peculiar inability of speech is associated with unilateral palsy, indicating disease of the left hemisphere of the cerebrum. (2.) In numerous cases, but not in all, there is disease of the third left frontal convolution of the cerebrum, or in the left anterior lobe, very near to this convolution. (3.) The whole of this convolution and the left anterior lobe of the brain may be wholly or partially destroyed, and yet no loss of speech has been observed. (4.) Aphasia may exist with apparent integrity of the left anterior lobe of the cerebrum, and indeed of the whole brain.

As a separate and distinct disease due to a specific lesion, *aphasia* cannot yet find a place in nosology. The phenomena are complex in the extreme; and the connecting link between the mental defect and loss of speech, with material change in the brain, is both clinically and anatomically still incomplete. Mental co-operation is defective in all the cases.

**Prognosis.**—Aphasia following typhoid fever has been the most favorable in its result; and even with obvious brain lesion, speech may return after having been absent for years. Where the lesion has been believed to be the result of syphilis, cases of aphasia have also recovered. The prognosis in all cases must be a guarded one.

**Treatment.**—No definite or specific line of treatment can be given. Every case must be prescribed for on its own merits, and after a careful study of the probable origin and nature of the circumstances which have led to aphasia.

#### PARALYSIS DUE TO BLOOD-POISONING.

As a clear diagnosis of the various forms of paralysis referred to in the previous pages is specially of importance with regard to the line of treatment to be adopted in any particular case, it is only necessary to notice here that paralysis may be due to blood-poisoning. Several of these forms of paralysis have been already noticed—*e. g.*, paralysis from the use of the vetch known as the *lathyrus sati-*

*vus* (vol. i, p. 796); also from lead-poisoning (vol. i, p. 781); and from alcohol (vol. i, p. 770). To these must be added paralysis from diphtheria (vol. i, p. 521); also paralysis from syphilis (vol. i, p. 699).

With reference to paralysis from poisons, the principle of treatment must be that by elimination; and on this subject the reader is referred to an able article by the late Dr. Easton, Professor of *Materia Medica* in the University of Glasgow, in the sixth volume of the *Glasgow Medical Journal*, p. 155, *et passim*.

### ANÆSTHESIA.

LATIN EQ., *Anæsthesia*; FRENCH EQ., *Anesthésie*; GERMAN EQ., *Anæsthesie*; ITALIAN EQ., *Anestesia*.

**Definition.**—*The term anæsthesia implies a diminution or loss of function in a sensory nerve, from its nervous force being reduced or destroyed—paralysis of sensation.*

[The late Dr. Beau (*Archives Gén. de Méd.*, 1848), showed that two kinds of sensibility existed in the skin and the mucous membranes around the natural orifices; the one, tactile sensibility, by which we distinguish, by means of the touch, the form, temperature, consistence, &c., of a body, and the other, the sense of pain. To the first he limits the term *anæsthesia*, while the latter he calls *analgesia*. The skin may lose the sense of pain without tactile sensibility being affected; rubbing the finger over the skin is felt, whilst there is total insensibility to pricking and pinching; but where there is loss of tactile sensibility of the skin, the sense of pain is always wanting. The only exception to this rule is the remarkable case (No. 2, *Circular No. 6*), reported by Dr. Mitchell and his colleagues, where there was absolute loss of sense of pain in the right leg, belly, chest, and arm, without disorder of tactile sensibility in the parts. But this was the result of reflex paralysis, following gunshot injury, and therefore centric. Although it is to the investigations of modern observers, and more particularly those of the present time, that we owe a knowledge of the frequency and nature of anæsthesia, still sensory-paralysis, independent of motor-paralysis, was known to the ancients. Aretæus thus speaks of it: “Paralysis autem motus tantum fere est actionisque defectio. Quod si nonnunquam solus tactus deficit (raro autem id evenit) potius anæsthesia, id est sensus abolitio, . . nominatur” (*De Causis et Signis diut. Morborum*, lib. i, cap. vii.).]

**Pathology.**—Sensibility admits of varying degrees, being different at different parts of the cutaneous surface, as proved by the experiments of Weber. His mode of experimenting may be adopted, as already noticed, as a practical means of diagnosis in paralysis of sensation.

Anæsthesia most commonly occurs as an immediate antecedent of motor palsy, or coincident with it, and also exists in various parts of the body independent of paralysis of motion. It varies in extent from a mere numbness of the parts to a complete loss of sensation. The cutaneous nerves are those most frequently affected, and from this cause the disease most usually attacks the integuments of a portion of the trunk, or of an arm, or a leg, or some

portion of the extremities, and also the whole face or parts of the face.

[The function of sensation depends upon the concurrence of three sets of organs, anatomically continuous, yet to a certain extent physiologically distinct. (1.) The nerve-extremities, which receive an impression; (2.) The afferent nerve-fibres which carry the impression to the centre; (3.) The centres, which receive the impression and transform it into a sensation. Thus the three factors of sensation are, the sensitive nervous extremities, the sensory conducting filaments, and the sentient portions of the nervous centres. If the integrity of any one of these is affected, there must result diminution or loss of sensation. If perception in the sensorium is destroyed, the nerves may transmit the impression to the sentient centres, but there is no sensation. This is one cause of anæsthesia, and occurs in certain lesions of the brain, as cerebral hemorrhage, softening, and tumors; or the function of the organ may be temporarily in abeyance from the effect of certain agents, as chloroform, or narcotics. Another way in which anæsthesia is produced is by interruption of the continuity of the nerve-trunk, by section, or compression, or atrophy, or tissue-degeneration. A third mode is, whilst both the nerve-centres and the nerve-trunks are sound, the sentient nerve-extremities have undergone some morbid change, and the impression is not received, as in insensibility of the skin from the application of intense cold, blindness from disease of the retina, loss of the sense of smell during coryza, certain cutaneous eruptions, &c. It sometimes happens as the result of reflex action in visceral disorders, and after mechanical injuries (BROWN-SÉQUARD, S. W. MITCHELL). This pathogeny of anæsthesia being admitted, can we make a practical application of it, and determine clinically the seat of the morbid condition, and refer it to a given lesion of the brain or medulla, or of the nerves themselves? Is the anæsthesia central or peripheral?

(1.) *Anæsthesia resulting from cerebral disease* is usually of large extent, and may involve the whole body; generally unilateral, and on the side opposite to the brain lesion; voluntary motion is lessened or abolished; the reflex movements are commonly intact; intelligence is disordered; and the nutrition of the paralyzed parts impaired. (2.) *Anæsthesia consecutive to spinal disorder* is mostly less general than that caused by disease of the brain; it is as a rule bilateral and symmetrical, but when unilateral it is on the same side as the affected column of the medulla (though Dr. Brown-Séquard admits crossed spinal paralysis); voluntary motion is lessened or destroyed; reflex functions may be preserved or absent, according as the medulla is or is not sound below the site of the lesion; nutrition of the affected parts is often greatly impaired. (3.) *Anæsthesia depending on some lesion of the sensory nerves* is often very limited, its site corresponding exactly to the distribution of the nerve; voluntary motion intact if a nerve exclusively sensory is involved, or if it be a compound nerve, the paralysis is limited to a single group of muscles; reflex movements constantly wanting where the anæsthesia is complete; and the state of the nutrition of the affected part variable, but often good.]

The most important form in which insensibility comes under the notice of the practical physician, is that known as **FACIAL ANÆSTHESIA**. It consists in a deficient or entire loss of sensibility in the parts supplied by the fifth pair of nerves.



**Symptoms and Diagnosis of the Seat of Lesion.**—(1.) The more the anæsthesia is confined to single filaments, the more peripheral is the seat of lesion. (2.) If the loss of sensation affects a portion of the facial surface, together with the corresponding cavity of the face, the disease may be presumed to involve the sensory fibres of the fifth pair before they separate, or one of the main divisions after its passage through its cranial foramen. (3.) When the entire sensitive tract of the fifth nerve has lost its sensation, associated with derangements of the nutritive functions in the affected parts, the Gasserian ganglion, or the nerves in its immediate vicinity, may be the seat of the disease. (4.) If the fifth nerve is complicated with disturbed functions of adjoining cerebral nerves, it may be presumed that the lesion is situated at the base of the brain (ROMBERG).

The symptoms may develop themselves gradually, or may come on suddenly, and are not unfrequently preceded by neuralgia; and owing to the differences in the seat of lesion, what may seem to be a permanent symptom in many is absent or less marked in others. It is frequently complicated with facial palsy of the *portio dura* of the seventh nerve. In a well-marked case the symptoms may be generally stated to be loss of tactile sensibility of the parts supplied by the nerve—namely, the integuments of the cheek and side of the head, the eyelids, conjunctiva, tongue, Schneiderian membrane—accompanied by a loss of taste on the side of the tongue which is affected, frequently by loss of smell and hearing, and by inflammation of the eye, terminating in ulceration of the cornea, and by no means uncommonly in total disorganization of the globe. Paralysis and wasting of the deep muscles of mastication are also sometimes associated. The real nature of the affection may escape notice, if the attention is directed solely to any one prominent symptom, such as ophthalmia. The disease makes its onset in two or three ways. It is often sudden, preceded by a slight perversion of sensibility, or by a tingling sensation, frequent attacks of headache, and pains in the occipital region and side of the face. Dimness of vision is so prominent a symptom in some cases, that loss of sensibility may not at first attract attention. Of twenty-four cases, vision was affected in fifteen, hearing in nine, and smell in six (Dr. J. B. Cowan, in *Glasgow Med. Journal*, No. 2, July, 1853).

There are two important features in facial anæsthesia worthy of special notice—namely, 1st. That the parts do not waste as in muscular palsy; 2d. That though the parts are insensible to touch, they still remain sensible to changes of temperature, and to pain arising from inflammation in the nerve itself.

**Causes.**—The anæsthesia may result from disease—(1.) Of the cerebrum where the fifth nerve takes its origin; (2.) Of the nerve within the cranium; (3.) Of the nerve after it has emerged from the cranium; and according to the seat the symptoms vary.

The disease may be a consequence of some injury, such as the extraction of a tooth, as frequently happens to the submental branch, which conveys sensation to half the lower lip.

Dissections show that condensation, atrophy, softening, and the pressure of tumors, are the morbid conditions out of which the

anæsthesia springs. So varied are the sources whence the anæsthesia results, that its treatment can only be palliative, and directed towards mitigation of the symptoms. Spontaneous cures are said not to be unfrequent. The local applications, such as blisters, leeching, and cupping, are said to have been sometimes useful, combined with the administrations of purgatives [and electricity].

[Some other forms of anæsthesia may be briefly mentioned. Those of the special senses are loss of sight from *amaurosis*, nervous deafness, *paracusis*, loss of smell, *anosmia*, and loss of taste, *ageusia*. *Cutaneous anæsthesia*, as has been shown, may be centric or peripheral, and vary from slight numbness to complete abolition of sensation, with no sense of pain, or appreciation of heat (*thermo-anæsthesia*), and be bilateral, or unilateral, or limited to a single spot of small extent; sometimes its degree varies in different parts of the body. It is frequently associated with anæsthesia of the mucous membrane, generally the conjunctival, and with muscular anæsthesia. Besides organic lesions of the brain, medulla, and nerve-trunks, it occurs in functional disorders of the centres, as hysteria, chorea, and occasionally in insanity (AXENFELD); in visceral disease (the reflex form); after excessive fatigue (SANDRAS); after the local application of cold and certain irritating substances, as lye (ROMBERG); from the effects of certain toxic articles, as lead, arsenic, bromide of potassium, sulphuret of carbon, nitrous oxide gas, sulphuric ether, chloroform; in the course of and after septic diseases; and in asphyxia (FAURE). *Muscular anæsthesia*, has been defined to be a diminution or loss of the muscular sense. There is abolition of muscular consciousness; the patient, if his eyes are withdrawn from the limb, is unable to tell its position, whether it is flexed or extended, abducted or adducted, and may try to extend a limb, which is already stretched out; there is no appreciation of the amount of force required to meet or overcome any opposing force; the weight of the limbs is not felt; and the degree of density of various bodies cannot be estimated. The perception of muscular contraction is totally or measurably gone. Muscular anæsthesia, when fully developed, is even more unequivocally shown by the absence of sensibility when the muscles are pinched, squeezed, or shampooed, or an electric current passed through them, or a sharp instrument pushed into them. It may be unaccompanied by loss of the function of motility, shown by the ability to execute all necessary movements when the eye is on the limb, and by the amount of muscular vigor that can at need be exhibited, and by the feeling of lightness, and the absence of all sense of fatigue, in the extremities, so opposite to the heaviness and weariness of muscular paralysis; or there may be motor palsy, as in hysterical paralysis; or it may be accompanied by insufficiency in the power of co-ordinating voluntary movements. It frequently coexists with cutaneous anæsthesia. Landry speaks of numbness and formication as precursory phenomena, but they probably really belong to the primary disorder of the nervous centres. It is more fixed than cutaneous anæsthesia, and rarely presents, even in hysteria, those fitful mutations of site so common in the latter. The prognosis depends upon the pressure of centric lesions. (BEAU, BROWN-SÉQUARD, SZOKALSKY, LANDRY, BELLION, VULPIAN, YELLOWLY, BRACH, DUCHENNE (de Boulogne), BOURDON, AXENFELD).]

## NEURALGIA.

LATIN EQ., *Neuralgia*; FRENCH EQ., *Néuralgie*; GERMAN EQ., *Neuralgia*; ITALIAN EQ., *Neuralgia*.

**Definition.**—*Excruciating pain, which returns with renewed violence in a part after periods of temporary remission, and which is believed to be due to some unknown morbid state of the nerves of sensation, a symptom of a local lesion, or more commonly of a general affection.*

**Pathology.**—All neuralgias are symptomatic either of an organic lesion, of which the neuralgic pain is a reflex or sympathetic expression, or the pain is due to a more or less grave organic lesion, involving, compressing, or otherwise irritating various branches of nerves. In some cases neuralgia is symptomatic of various cachexias—*e. g.*, of chlorosis, of lead-poisoning, of anæmia, of malaria, of rheumatism, of syphilis; or it is a reflex induction from an acute inflammation, as from a carious tooth, a necrosed bone, a tumor, or a phlegmon. The form of the neuralgia from these several sources may vary, but *pain* of the characteristic kind defined in the definition is alike common to all. In most cases marked peripheral lesions are the starting-point of neuralgia, as in cases of decayed teeth, of necrosed bones, of tumors developed in the vicinity or in the substance of nerve-trunks, or of inflammation, including nerves within their area (TROUSSEAU).

The pain of neuralgia is distinct in character, and involves the minimum of organic change in the affected part, while the whole circumstances of the affection point to lowered vitality as the antecedent cause of the pain (ANSTIE). “The general bodily health is always at a low point when the attacks occur, and the nerves of the part are habitually in circumstances which must tend to lower their functional activity.” Dr. Anstie gives circumstantial evidence of this in his own case, consisting of the passive flow of tears, the hair of the right eyebrow becoming decidedly gray at a point exactly opposite the *supra-orbital nerve* (the one affected). These he justly regards as so many indications of *defective nervous energy*. Pain under such circumstances is generally the direct consequence of a further depression of an already feeble vitality in the nerves (ANSTIE, *op. cit.*, p. 84).

The principal varieties of this affection are—(a.) *Facial neuralgia* or *tic douloureux*; (b.) *Brow ache* or *hemicrania*; (c.) *Sciatica*; (d.) *Intercostal neuralgia*.

**Symptoms.**—The symptoms are similar in kind, whichever nerve is affected, modified only by the position, connections, and distribution of the nerve.

The branches of the *trifacial* nerve may be attacked separately or conjointly; most commonly, however, only one branch is affected, less frequently two, and the case must be severe in which the three branches, or the whole side of the face, is affected. Nevertheless, it sometimes so happens, extending even over the summit of the head, and over the temporal region, by the deep branch of the fifth pair, which emerges to the surface anterior to the external meatus.

It is even also associated with a similar affection of the occipital branches at the same time.

The attack is sometimes sudden, but more generally it is preceded by a dull aching pain at the points where the nerve issues from the cranium, or becomes superficial. After this threatening symptom has lasted a few hours or a few days, the patient is seized with a violent darting or shooting pain in the course of the nerve, returning at intervals—phenomena which are characteristic of the disease. The paroxysm is short, lasting only a few seconds or a few minutes; but the pain is perhaps the most severe that the human frame is capable of suffering. Some patients have compared it to an electric shock of great intensity, others to the conflagration of gunpowder, and others to the intensity and violence of a fulminating powder. The late Dr. Pemberton was known to have stamped the bottom of his carriage out during the paroxysm; and Valleix mentions a physician who, suffering from neuralgia, was induced, by excessive agony, to make deep incisions into his face, and then to apply the actual cautery to the wound; but his pain not being mitigated by these methods, he several times attempted suicide. Even in mild cases the patient often, on the instant of attack, becomes fixed like a statue, fearing to move a muscle or a limb, lest he should aggravate the pain or reproduce the seizure. This is a condition common to many nervous affections attended by excruciating pain, such as *angina pectoris*.

In cases of ordinary intensity the effect is so completely limited to the nerve that the skin is not discolored, while the organs immediately in connection with it are little affected—the eye, perhaps, being only watery, the nose hot, and the teeth aching. In severer cases, however, and where the disease affects the nerve generally, or the whole face and scalp, the condition of the patient is most lamentable. The mouth is spasmodically drawn, as in palsy, so that the saliva flows over the chin and neck, or the teeth chatter by the clonic spasms which sometimes attend the disease. The saliva is increased in quantity and altered in quality; for in cases in which the patient is afraid to brush his teeth, lest the paroxysm should return, the whole of the teeth of the lower jaw become so incrustated with tartar as to form one solid mass, indicating at the same time a depraved state of the digestive organs. The eye and eyelid are likewise frequently convulsed, the conjunctiva injected, and the nose discharges a muciform matter. To touch even the hair of the head produces pain, and sometimes the affected nerve may be traced by a red line marking its course. The recurrence of the paroxysm is uncertain: in slight cases it may return only once in a few weeks, or in a few days; but in some severe cases it will return every quarter of an hour, every five minutes, or every minute, and even every few seconds. In a few cases the paroxysms occur periodically, and at stated intervals. In general, however, the times of recurrence are uncertain; sometimes the patient is attacked with great violence many times a day for many days or weeks together, so that the disease is almost continuous; at other times it intermits for a week, a month, six months, or a year. The

disease is situated nearly as often on the right as on the left side of the face; and sometimes on both sides.

When the branches of the fifth cranial nerve are affected, the most painful points are at the exit of the ophthalmic of the superior and of the inferior maxillary branches. Next to those the frontal, and next the parietal, and lastly the occipital, although its origin is independent of that of the trigeminal; and whether the trigeminal was affected by itself, or the occipital nerve as well, Trousseau has always observed that "pressure made on the spinous processes of the first two cervical vertebræ always caused pain, and in a certain proportion of cases immediately brought on shooting pain in the diseased nerves. When the nerves of the brachial plexus are affected, pressure made over the spinous processes of the last cervical vertebræ gave pain;" so also in cases of intercostal, lumbar, or sciatic neuralgia, similar pressure over corresponding regions would also produce pain. Trousseau, therefore, makes the general statement, that "in neuralgia the spinous processes of the vertebræ are tender on pressure at a spot nearly corresponding to the point of exit of the nerve from the intervertebral foramen, and that the pain pretty frequently extends a little farther up along the vertebral column." Thus neuralgia reveals itself by acute pain when pressure is made over the spinous processes which correspond to the origin or point of exit of the implicated nerves.

*Cutaneous hyperæsthesia* at the points of exit of the nerve-trunks is another peculiar characteristic of neuralgia. It is most marked in cases of intercostal, lumbar, and crural neuralgia. In such cases a slight scratch of the skin, or gently rubbing the skin with a blunt end of a pencil, will cause pain of a burning or pricking kind; and in many cases the track of the implicated nerve may be followed by the tip of the finger as far as its cutaneous distribution. More rarely the reverse of this condition supervenes—namely, *anæsthesia*. It is most apt to attend neuralgia apparently of a rheumatic origin, or which is due to a slight lesion of the cord. In such cases, when the *hyperæsthesia* has lasted a long time, it is followed by *anæsthesia*. In *herpes zoster* this is apt to occur; also in *sciatic neuralgia*.

Certain *superficial tender spots* are also characteristic of neuralgia. It is generally at the point of exit of nerves from an osseous foramen that pain is most particularly felt. Thus, in neuralgia of the fifth pair, it is over the *supra-orbital notch*, where the ophthalmic branch becomes superficial; over the *infra-orbital foramen*, which gives passage to the superior maxillary branch; and over the *mental foramen*, through which emerges the inferior maxillary division of the fifth pair. But when *supra-orbital neuralgia* is intense, the point over the nasal branch is also extremely tender on pressure over the point of exit of this small nerve; and pressure over the frontal eminence also causes acute pain; and another tender point is over the *zygomatic process* in front of the ear (TROUSSEAU).

The particular form of cachexia has also a remarkable influence on the seat of neuralgia. In chlorosis, neuralgia is apt to affect several regions, but notably the *trigeminal nerves* and nerves of the solar



*plexus.* In cases of anæmia from *uterine hemorrhages* or *leucorrhœa*, the neuralgia is mainly *gastric* and *intestinal*. In malaria, the ophthalmic nerve is mostly implicated. In rheumatic cachexia it is generally the occipital and sciatic nerves; and neuralgias of rheumatic origin are generally multiple in their manifestation, and frequently alternate with articular pains (Trousseau).

The exceptional cases, where pain is relieved by pressure, show that the conditions causing the pain have their site to the central side of the place pressed upon. Such cases may be relieved by division or removal of a portion of the nerve-trunk at the part pressed upon.

The duration of the disease is uncertain. In some cases it terminates after a few paroxysms; in others it lasts from one to six months; and in some cases it becomes chronic, and embitters existence during the whole period of a long life. It seldom disappears suddenly, but oscillates with decreasing intensity, the intervals gradually becoming lengthened, till at last the disease subsides.

**Causes.**—The remote causes of neuralgia have been already indicated. Extremes of heat or cold, or sudden changes from the one to the other, often stand in the relation of cause to this disease. It is often a result of impaired general health. Thus women, after profuse menorrhagia, or after childbirth, or persons recovering from fever or other severe disease, are apt to suffer from neuralgic affections. Persons who have attempted to poison themselves with arsenic suffer agonizing pains along the course of the nerves of the limbs during convalescence. Blows, or wounds, or the pressure of aneurismal or other tumors, sometimes seated in the nerve itself, are also causes of this affection.

Neuralgia is more common in men than in women; and in women it occurs rather more frequently before thirty than afterwards, especially in those whose menstruation is irregular either as to time or quantity. Among the poor and laborious classes neuralgic complaints are numerous; and, roughly speaking, they mainly present three varieties: (1.) *Neuralgia of the head and face*; (2.) *Neuralgia intercostalis*; (3.) *Sciatica*. The great majority of the patients are anæmic, and in a fair sprinkling of cases the anæmia is obviously connected with malaria (Anstie, *op. cit.*, p. 86).

**Diagnosis.**—The disease to which neuralgia bears most resemblance is rheumatism, but it is distinguished from it by the transitory nature of the attack, and by the absence of all swelling. There are certain points of the face which, upon being pressed in the interval of the paroxysm, give pain, and so far furnish phenomena which may aid in a differential diagnosis. Other diagnostic features are of importance to be noticed, such as, if pain exists—(1.) Where the nerve emerges from the bone, as at the supra-orbitary, infra-orbitary, and mental foramina, in trifacial neuralgia; (2.) Where the nerve passing through the muscles reaches the skin; (3.) Where the nerve terminates in the skin; (4.) Where the nerve becomes very superficial, as the peroneal nerves; and, lastly, (5.) Where the pain affects the spinous processes corresponding to the exit of the implicated nerves.

**Treatment.**—A practitioner has generally some mode of treatment

which, during his practice, he considers specific in this disease. *Sarsaparilla*, the *sulphate of iron*, the *carbonate of iron*, *arsenic*, *mercury*, or the *sulphate of quinia*, have all been recommended with confidence. Bleeding, either local or general, has had its advocates, while its opponents affirm this operation to be always useless and sometimes injurious. There can be no question that the disease has often subsided under the use of all these various remedies; but the tendency in neuralgia to a spontaneous intermission is so great that it is doubtful whether in any case medicine can be said to have cured it. Opiates are unquestionably serviceable in mitigating the sufferings of the patient, and perhaps in influencing the disease, but not to the extent generally supposed. *Belladonna*, both internally and as a plaster, may sometimes relieve the pain. *Stramonium* and *opium* have a similar temporary effect. *Belladonna*, and perhaps *stramonium*, are better than *opium* as an habitual remedy, and they appear to have done good occasionally. *Chlorodyne* is also to be recommended, the composition of which has been determined by the Pharmaceutical Society, and a formula for which is given in vol. i, p. 454.

The remedies of most value are the diffusible stimulants, such as *sal volatile*, hot tea, quinine in a full dose, alcohol in small doses, blistering, ammoniacal fluid to the skin immediately over the painful nerves, and the endermic application of a fifth of a grain of morphia. These remedies act by increasing the supply of blood to the painful nerve, and, *pro tanto*, heightening its vital energy (ANSTIE, *op. cit.*, p. 84). The most speedy way of obtaining a temporary relief is certainly the application of a local stimulant, and more especially of some volatile agent, such as mustard, or, still better, chloroform diluted with seven parts of some simple liniment (ANSTIE). A rapidly acting blistering fluid is still more effective. But the more profoundly the general health has been affected, and especially the greater the degree of anæmia, the more necessary is it to join with the use of stimulants (both local, such as above mentioned, and general, such as the *carbonate* and *muriate* of ammonia, taken in five and ten grain doses respectively) a treatment directed to improving the condition of the blood by "food tonics," such as cod-liver oil, arsenic, or steel, or a combination of some of them, joined with the use of local stimulation, by means of frictions with dilute chloroform, and the manipulations of the scientific "*shampooer*" (ANSTIE, *op. cit.*, p. 87). Dr. Radcliffe reports favorably of the *hypophosphite of soda*, and believes that the *phosphorus* of this salt acts directly as a food to the nervous tissue. The subcutaneous injection of small doses ( $\frac{1}{4}$ th to  $\frac{1}{2}$ th of a grain) of morphia in solution will give temporary relief, and is especially useful to those patients who are obliged to go through an ordinary day of labor (ANSTIE, *op. cit.*, p. 87).

When these or other general remedies have proved insufficient, recourse has been had to local remedies. The most efficient of these applications is the alkaloid *aconitina*, rubbed upon the pained part in the form of an ointment, in the proportion of one or two grains to one drachm of lard. Morphia similarly used, and blisters,

have also often exercised a beneficial effect upon the disease. Holding the head over steam, and the warm bath, are equally or even more beneficial in cases of facial neuralgia. The belladonna plaster is also a favorite application. When the neuralgia is superficial, compresses steeped in the solution of atropia have a good effect. Trousseau recommends the following formula:

*R. Atropiæ sulphatis, gr. v; Aquæ distillatæ, ℥iij; solve.* Renew the compresses several times in the twenty-four hours. Continue them for at least an hour each time, and cover them with oilskin, to prevent evaporation.

When general and local applications are unsuccessful, the cause may be sought for in a diseased tooth or stump, and in a *very few* instances an exostosis of the stump has been discovered, removed, and the disease cured. Sometimes, however, even when the patient submits to have every tooth in his head drawn, no relief or benefit has resulted. The division of a nerve is sometimes had recourse to; but even this operation is very uncertain.

[The *specific* treatment of neuralgia is no doubt constantly disappointing. The disorder is associated with an irritative state of the nervous system, indicating defective systemic vital power, and, consequently, deficient nerve-power, and requires a tonic and restorative treatment, similar to that already recommended in some of the other neuroses. For some years the writer has pursued this treatment of neuralgia, and with such a measure of success as to warrant the belief that it will prove more generally happy than one based on the supposed curative power of any single drug. The general nutrition must be bettered by the various means before named. Appropriate and constant outdoor exercise must be insisted on; the lungs developed; the diet nourishing, and containing, as Dr. Radcliffe very properly insists on, a due amount of oleaginous and oily matters; tea and coffee are to be avoided, as well as all saccharine articles, and milk and cocoa permitted; and in some cases alcoholic stimulants may be temporarily necessary. The restoration and maintenance of a healthy state of the skin is very important, and for this purpose baths and warm clothing are to be used; flannel should always be worn next to the skin, and even a buckskin under-suit may be necessary. Cold and dampness are to be avoided, the experiments of Ahrens and Nasse showing how cold is favorable to the development of pain by preventing the production of animal electricity, and how damp brings about the same result by favoring the conduction of electricity away from the body. All depressing, spoliative, and irritating remedies must be avoided; and it should be recollected that opiates are only temporarily relieving, and are open to the serious objection of deranging digestion, impairing the appetite, and thus nullifying the treatment recommended. Iron,—the syrup of the phosphates of iron, quinia, and strychnia, the tartrate of iron and potash, the ferri carbonas saccharata,—phosphorus, in the form of the compound syrup of the phosphates (chemical food), and arsenic, may all be required in the course of the treatment, and are often valuable adjuvants as eutrophics, or as special nerve nourishers, but given alone as specifics they are very unreliable. In many cases,—particularly of facial neuralgia,—electricity or galvanism gives great relief, and, possibly, contributes towards a cure. Dr. Radcliffe believes that good will arise from

insulating the patient and charging him with positive electricity, taking or not taking sparks from him,—thus improving the natural electrical condition of the system. He is also disposed to think that in some forms of neuralgia, a succession of shocks from an induction coil, during the paroxysm, has a beneficial influence, provided the operation be carried on long enough to bring on vascular reaction,—an artificial hot stage,—by paralyzing to a certain extent the vaso-motor nerves. (*Lectures on Epilepsy, Pain, and Paralysis*, Am. ed., p. 266). It not unfrequently happens after the repeated application of the constant current, that the nerve-pain, particularly in prosopalgia, gradually diminishes, and finally subsides altogether, and there are long intervals of relief from suffering. The partisans of this method of treatment claim that it is constantly curative. The writer's experience is not yet sufficient to enable him either to affirm or deny positively this statement; but its effects are often so immediately mitigating, and so far as his individual observation goes, measurably durable, as to warrant its trial (see Remak's *Lectures on the Treatment of Nervous Disorders by the Application of the Constant Current*).

Besides the means already given for the treatment of the paroxysm, subcutaneous injections of narceina or atropia will be found to give the best instant results. A recent high authority in neuro-therapeutics—Dr. C. B. Radcliffe—has written: "I have no hesitation in saying that the proper use of alcoholic stimulants is at once the natural corrective of the neuralgic habit and the most trustworthy of all anodynes. I have repeatedly known a paroxysm of neuralgia prevented and cut short by a glass of hot grog. . . . And I have too often seen the beneficial influence of rum and milk in the morning, in correcting the neuralgic habit, to have any room left for doubt on this score" (*loc. cit.*, p. 201). This is most dangerous teaching. The "hot grog" treatment should not be inconsiderately resorted to in chronic cases, and should be warily used only when the physician is satisfied that its employment is indispensable. The "proper use" of alcoholic drinks, so insisted on, is very difficult. Alcoholic stimulants are not "the natural corrective of the neuralgic habit, and the most trustworthy of all anodynes," though their immediate effect in neuralgia is often relieving, and thus a habit of resorting to them is formed, leading too frequently to the saddest consequences. The writer has seen more than one instance of confirmed intemperance in women of good social position, which could be laid at the door of this practice.]

#### [EPILEPTIFORM NEURALGIA.]

(DR. CLYMER.)

Dr. Trousseau has described, under the name of *epileptiform neuralgia*, a variety of prosopalgia, and which, from the suddenness of the invasion, the excruciating intensity of the pain, its short duration, from its running the same course as epileptic aura or vertigo, and the impossibility of relieving the paroxysms, he thinks ought not to be confounded with ordinary facial neuralgia, and merits a distinct place amongst the neuroses. He thus describes it: An individual who, but a moment ago, was perfectly free from suffering, is suddenly seized while talking, with horrible pain along the branches of the fifth pair. He puts his hand to his face, pressing or squeezing it forcibly, sometimes rubbing it so much and so often that the hairs on that side fall off. He goes on rocking himself, holding his head between his hands, and uttering half-suppressed groans. This scene lasts for ten, fifteen seconds, one minute at the most,

and all is over without convulsions, and he resumes his interrupted conversation until another attack comes on. The paroxysms sometimes recur every two or three minutes, when they may cease for weeks or even months, to relapse again. In the majority of cases the relief is not complete, for during the intervals a slight degree of pain or uneasiness is complained of at the point of emergence of the affected nerve. The act of masticating often causes unbearable darts of agony, and nutrition is obliged to be kept up by liquids.

A case reported by Dr. Ramskill (*British Med. Journal*, vol. i, 1867) set in with excruciating pain at the top of the head, over the area of distribution of the ophthalmic branch of the fifth nerve, this point was excessively tender, although no swelling could be detected, and the least touch upon it brought on a most fearful paroxysm of pain. There was great tenderness on pressure over both tibiæ, and also along the ulnar portion of the forearms; but a careful examination showed no thickening of the periosteum or any node. There was no family history of epilepsy, or personal history of syphilis. The intellect was unaffected; all the senses were perfect; no diminution of power in any of the limbs; nor sickness, nausea, nor giddiness.

Whatever may be the analogy between true epilepsy and epileptiform neuralgia, it must be admitted that the two disorders are only analogous and not identical. In neuralgia there is no impairment of the intellect, nor convulsions. Still, Dr. Trousseau was led to believe, from some cases which came under his notice, that epileptiform neuralgia is one of the manifestations of true epilepsy. Besides the intense severity of the pain, the lightning-like suddenness of the onset, the muscular spasms, and the presence of exquisitely tender spots, the least pressure on which cause intolerable agony, and which may be developed by a mere breath of wind, Dr. Anstie believes that another characteristic of this form of trigeminal neuralgia is its association with strong melancholy and a suicidal tendency, and a family taint of insanity (Reynolds' *System of Medicine*, article Neuralgia, vol. ii, p. 726). The disease is of disheartening obstinacy, resisting all remedies. In a practice of thirty-six years Dr. Trousseau has never known a single case to be radically cured, even after exsection of the nerve (*Clinique Médicale*, vol. ii, 2ème ed., 1865).

**Treatment.**—In *epileptiform neuralgia*, Dr. Trousseau says candidly that he has never cured, or seen cured, a case. He believes opium to procure more decided relief than any other remedy; and he relates several cases where he has given it in enormous doses. In one case a drachm of the sulphate of morphia was taken every day, and subsequently five to twenty drachms of crude opium, with the effect of quieting the pains. In Dr. Ramskill's case, after the ineffectual use of both the iodide and bromide of potassium, immediate and great relief was given by half-grain doses of morphia, four times a day, gradually increased to one-grain doses. Belladonna is almost completely powerless. Division of the painful branches of the trifacial gives immediate but not lasting relief. "I formerly believed," says Trousseau, "in the complete efficacy of this measure, but as I grew older I unfortunately lost all my illusions on that score." Dr. Nelaton states, however, that by resecting about one-fifth of an inch of the affected nerve, he has in two cases got complete cures. Almost instant relief has followed Faradization in some cases in the hands of Duchenne (de Boulogne), but it has generally failed. The constant current offers better results. Dr. Trousseau does not seem to have used narcotics hypodermically.]



## CHAPTER VIII.

## ON THE PATHOLOGY AND DIAGNOSIS OF DISEASES OF THE HEART AND LUNGS—OF THE THORAX GENERALLY—ITS CONTENTS AND PARIETES.

THE diseases of the heart and lungs are especially recognized and appreciated by *symptoms*, local and constitutional, as well as by so-called *physical signs*; and it is necessary to notice, in some preliminary sections, certain topics, a knowledge of which is essential to the accurate diagnosis of these diseases.

## SECTION I.—RELATION OF THE THORACIC VISCERA TO THE WALLS OF THE CHEST.

(A.) *Regions of the Thorax.*

In order to enable the student and physician accurately to describe and record their observations, and more conveniently to communicate to others precise information respecting the seat and signs of internal diseases, it has been customary to map out the exterior surface of the chest and abdomen by lines into different compartments termed *regions*. These lines have not always been drawn in the same manner; and therefore, on the contrary, there has ever existed great discrepancy among writers and teachers in this and other countries respecting the number, the extent, and the names of the regions defined by such lines. The regions are quite arbitrary, and the lines described upon the surface are understood to correspond with imaginary planes passing towards the centre of the body. The points or *landmarks* on the surface through which these lines are drawn ought to be at once fixed points and obvious to the senses; so that the boundaries and contents of every region may be accurately defined, with the view of localizing physical signs as precisely as possible.

As it is necessary that a complete inspection of the body shall take in simultaneously the abdomen and the chest, the topography of these regions is generally given together; and the annexed regional plans of the trunk are those devised by Mr. Paxton, of Oxford; and given by the late Sir John Forbes, in his excellent paper "On the Exploration of the Chest and Abdomen," in the first volume of the *Cyclopædia of Practical Medicine*.\* Prominent points of

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\* To facilitate accuracy in this method of recording physical signs, blank outlines of the trunk of the body were first used by Piorry, and more recently they have been largely used both in clinical teaching and in recording cases for publication, by Professors Bennett, in Edinburgh, Gairdner, of Glasgow, and Beale, of London; the latter of whom has especially described the great advantages from their use, in the sixth number of the valuable *Archives* which he publishes (vol. ii, p. 97). The outlines merely of the trunk (without the dotted lines and the numbers), given in the text to illustrate the topography of the chest and abdomen, have been engraved on

the skeleton are here made the basis of the system by which the regions are mapped out by vertical and horizontal lines. The descriptions of the boundaries and contents of the regions have been compiled on the authorities of Sibson, Walshe, Parkes, Lyons, Fuller, Quain, and Sharpey.

The vertical lines having relation to the chest are *eight* in number, and run as follows: (1.) Along the middle of the sternum, from its upper to its lower end; (2.) From the acromial end of the clavicle to the external tubercle of the pubes (right and left); (3.) Along the spinous processes of the cervical and dorsal vertebræ; (4.) Along the posterior or spinal border of the scapulæ, from the clavicular transverse line to the mammary transverse line. (See Figs. 5, 6, and 7.)

The horizontal or transverse lines are four in number, and are as follows: (1.) Around the lower part of the neck, sloping downwards to the upper end of the sternum anteriorly, and to the last cervical vertebra posteriorly; (2.) Around the upper part of the chest in the line of the clavicles; (3.) Around the middle of the chest by the lower edge of the *third* rib, above the line of the male nipple, and touching the inferior border of the scapulæ behind; (4.) Around the lower part of the chest, on a level with the xiphoid cartilage. (See figures as before.)

By these lines the lower part of the neck and the chest is divided into three horizontal and eight vertical bands; and by the intersections of these lines various compartments or regions may be indicated.

The most useful arrangement of the compartments and nomenclature of the regions is as follows:

*Anterior*, *lateral*, and *posterior* regions being recognized, the *anterior* are named the *supra-clavicular*, *clavicular*, *infra-clavicular*, *mammary*, *infra-mammary*, *supra-sternal*, *superior sternal*, and *inferior sternal*.

The *lateral* regions are,—the *axillary* and the *infra-axillary*.

The *posterior* regions are,—the *supra-spinous* region; the *infra-spinous* region (sometimes called the scapular); the *inter-scapular*; the *infra-scapular* (sometimes called the upper dorsal). Of these regions the three sternal are single, all the rest are double.

The **Subclavicular Region** is a small triangular space *above* the clavicle on either side, with its base internally at the trachea, its apex towards the outer end of the clavicle, and bounded below by the upper edge of that bone. A line drawn from the outer part of the clavicle to the upper rings of the trachea will limit its upper border.

In this region is found the triangular apex of the lung (Figs. 8, 9), sometimes reaching on the right side a little higher than on the

wood; so that the student can readily indicate the areas of physical signs by pencil, ink, or colored chalk lines.

“Outline Figures for Indicating the Areas of Physical Signs in the Clinical Diagnosis of Disease;” for the use of Students and Practitioners of Medicine. Arranged by WILLIAM AITKEN, M.D., &c. Charles Griffin & Co.

FIG. 7.

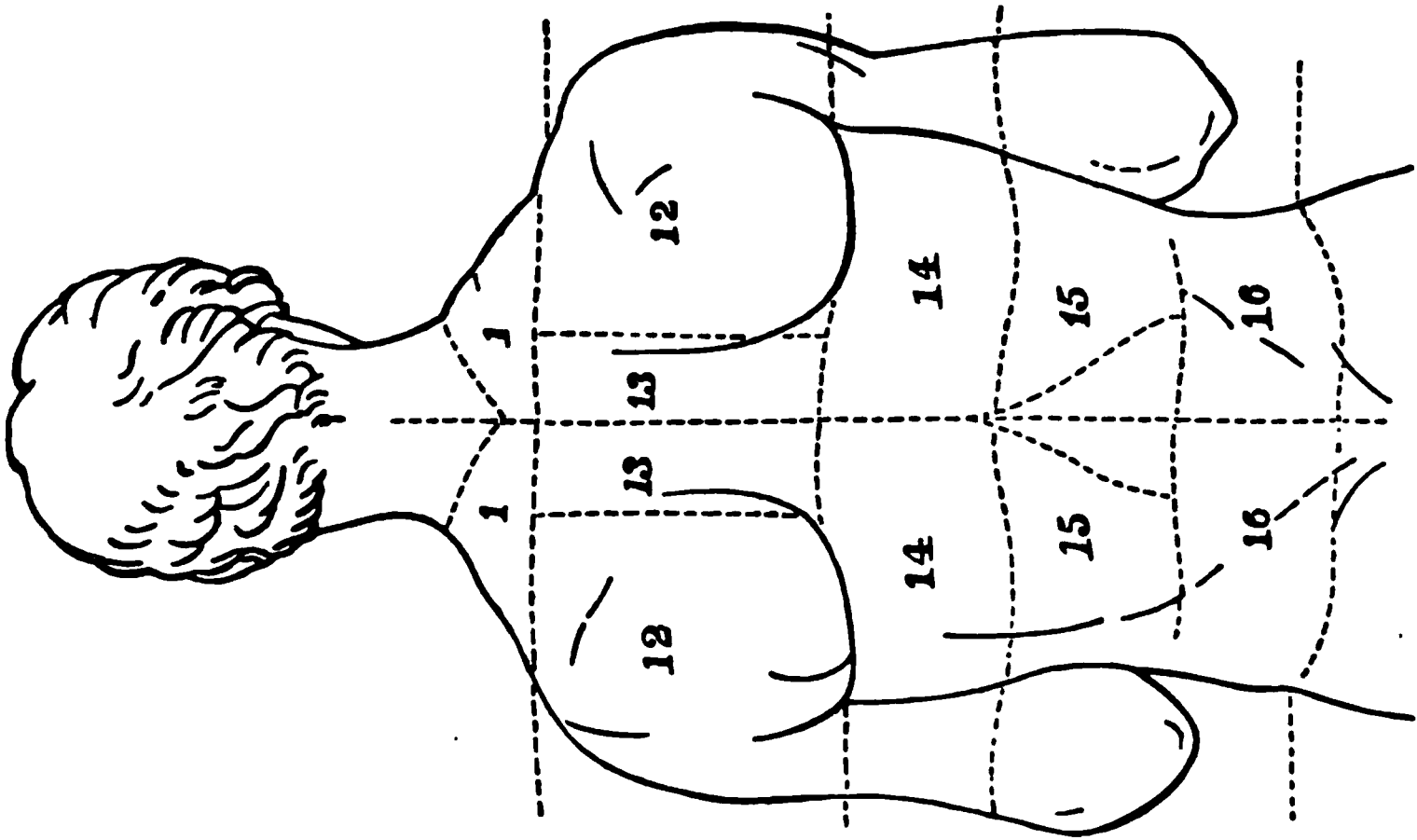


FIG. 6.

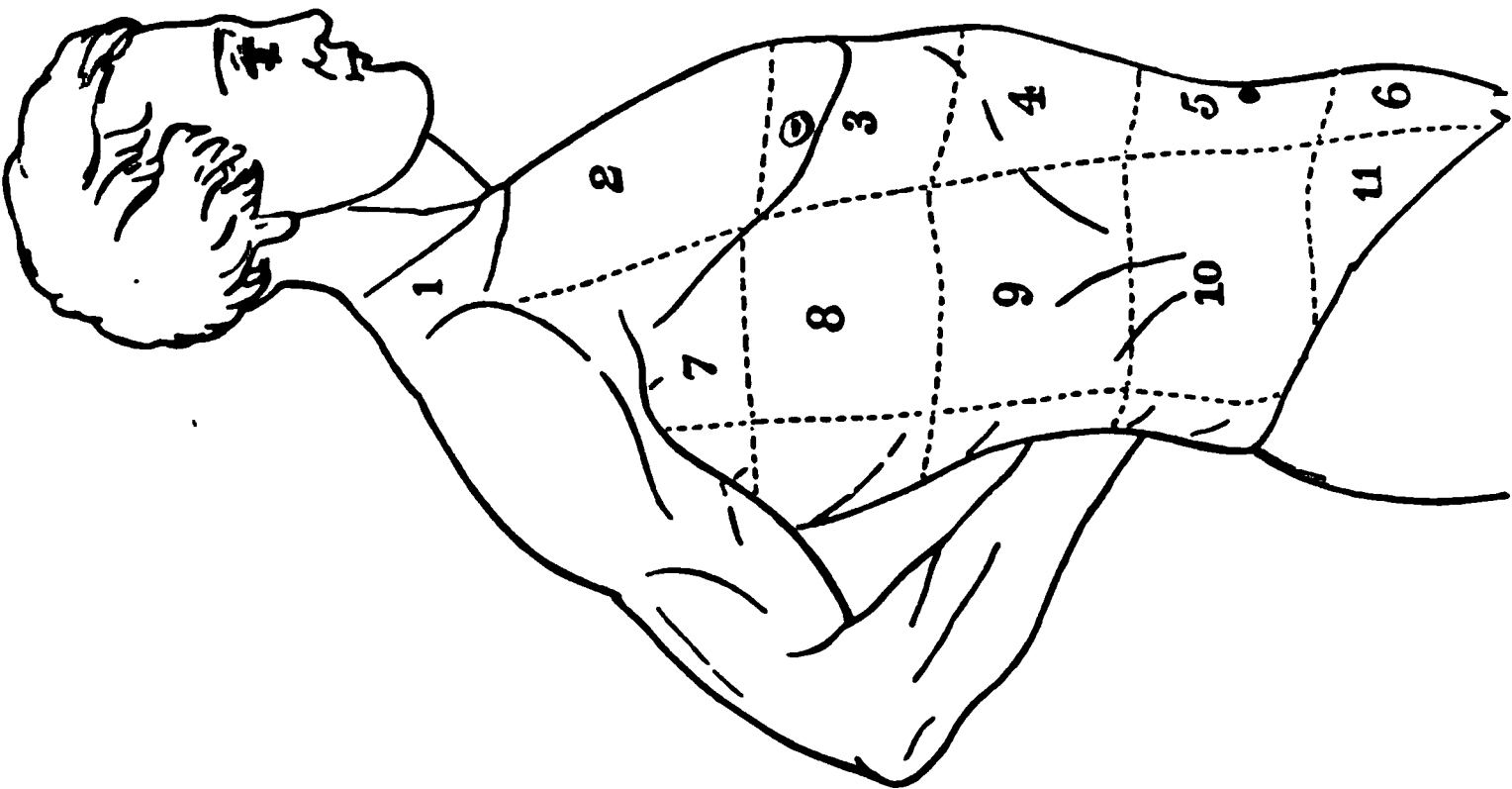
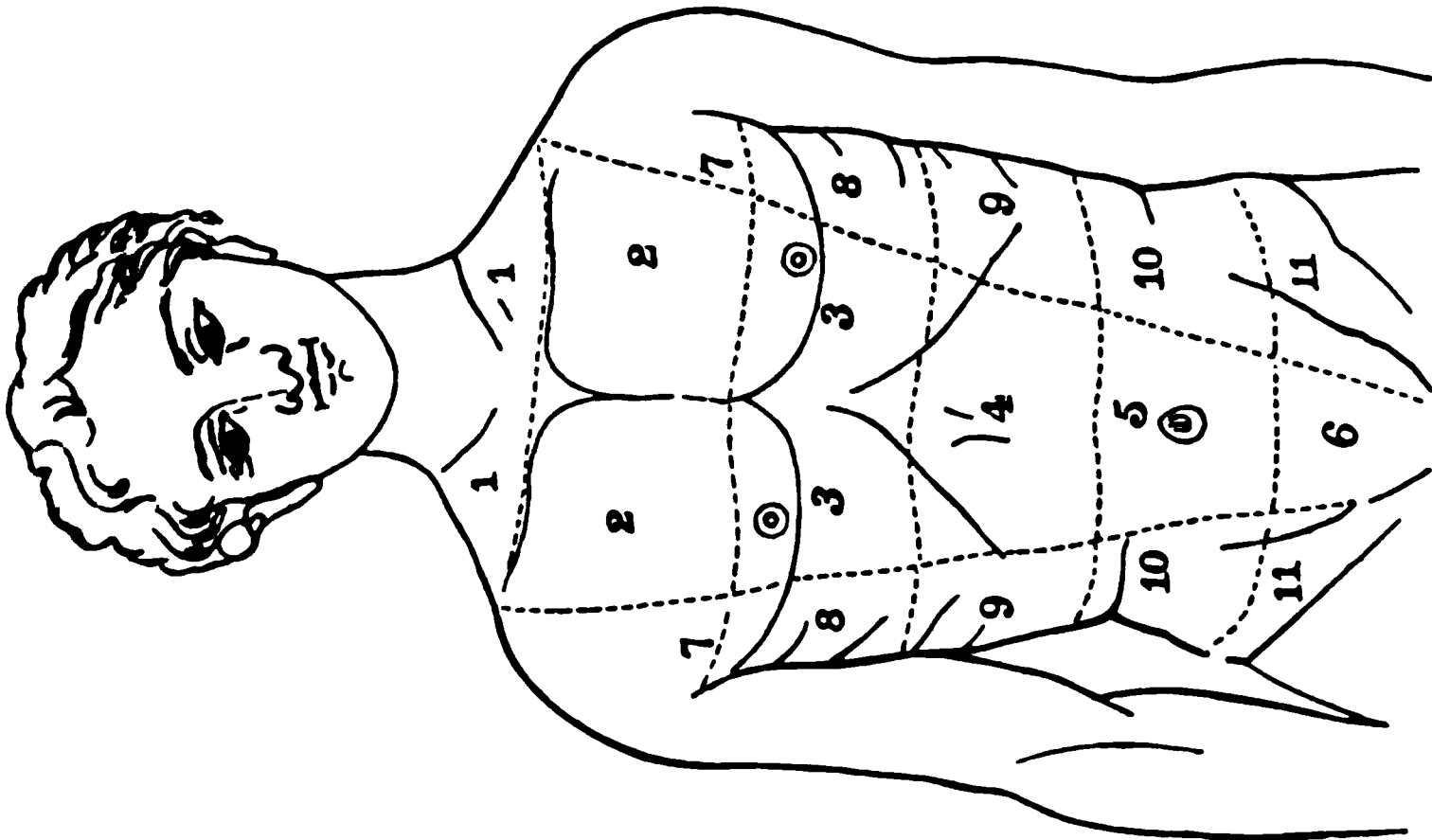
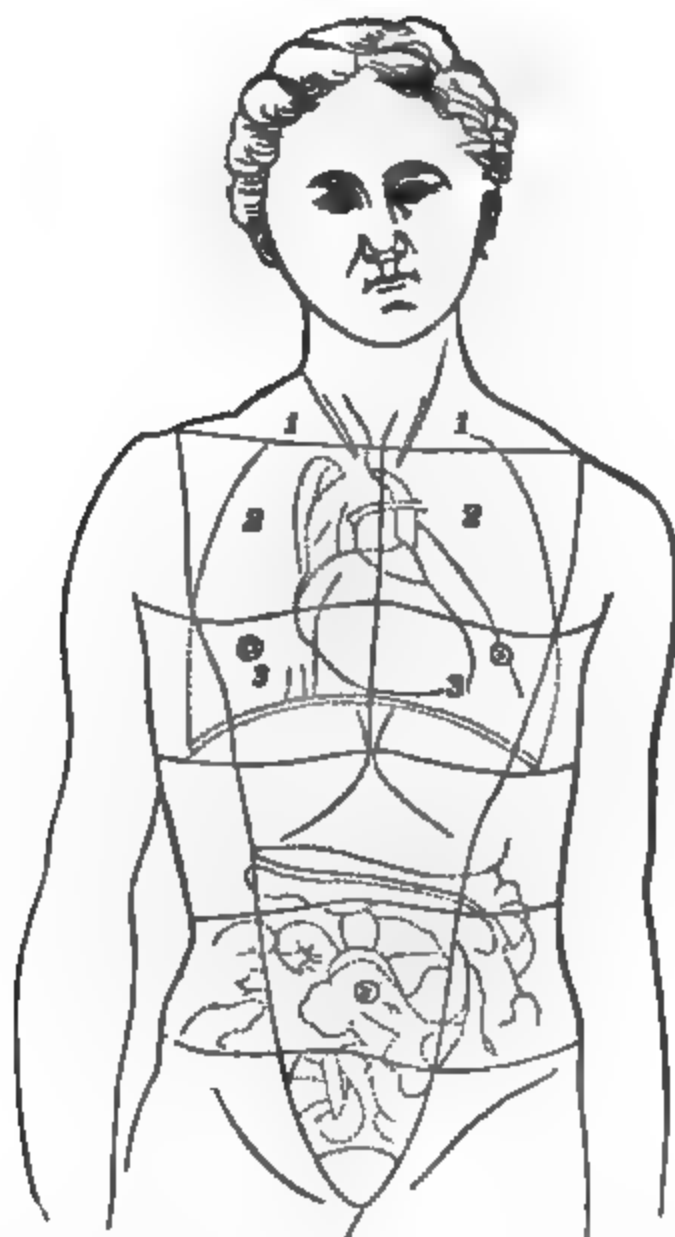


FIG. 5.



left, with portions of the subclavian and carotid arteries, and of the subclavian and jugular veins. The floor of this region internally is formed by the upper surface of the first rib.

FIG. 8.



The **Clavicular Region** is very narrow and oblong, corresponding to the inner two-thirds of the collar-bone. Behind the bone lies on both sides lung-substance; but on the right side, at the sternoclavicular articulation, is the *innominate artery*, and the subclavian artery crosses the region at its outer edge; on the left side the carotid and subclavian arteries pass upwards, almost at right angles to the bone.

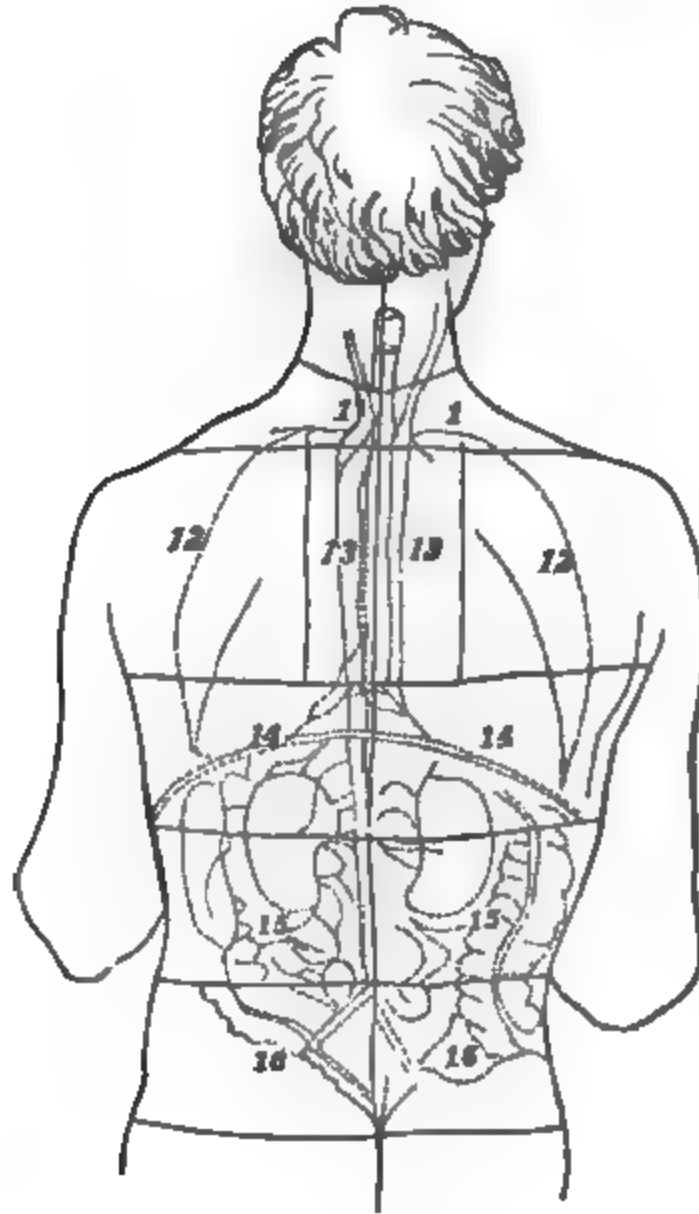
The **Infra-Clavicular Regions** are nearly square, corresponding to No. 2 in the figures already referred to. Each region (right and left) is bounded above by the inferior border of the clavicle; below by the lower border of the third rib, where it joins the cartilages of the sternum; it is bounded on the outside by the vertical line passing from the acromial end of the clavicle downwards towards the external tubercle of the pubes (on either side); on the inner side the subclavian region is bounded by the edge of the sternum.

Within these limits is placed the upper lobe of the lung on both

sides, close to the sternal border of the region. On the right side lie the superior *vena cava*, and a portion of the arch of the aorta. On the left side, close to the sternum, is the edge of the pulmonary artery.

The inferior border of the region on the left side corresponds to a portion of the base of the heart; while part of the right auricle

FIG. 2.



occupies the inferior corner of the region towards the sternum on the right side.

The **Mammary Region** (No. 3, Figs. 5, 6, and 7) has also a square-like form, and is bounded above by the line passing through the lower border of the *third* rib, where it joins the cartilages of the sternum below, by the line passing transversely on a level with the xiphoid cartilage (its upper border); outside by the vertical line passing to the outer tubercle of the pubes (on either side); and on the inner aspect by the edge of the sternum.

The contents of the mammary regions differ greatly on the two sides.

On the right side the lung lies throughout immediately underneath the ribs. Its inferior border turns off almost at right angles



from the sternum, at the cartilage of the sixth rib, whence its thin sharp border gently slopes outwards and downwards, so as to occupy the lower part of the region when the diaphragm is depressed. But when the diaphragm is elevated, the liver rises into this region up to the fourth interspace. The fissure between the upper and middle lobes of the right lung passes upwards and backwards obliquely across the region from about the *fourth* cartilage. The fissure between the middle and lower portion passes in the same direction from the *fifth* interspace. A portion of the right auricle, also a portion of the right and superior angle of the right ventricle, lie between the *third* and *fifth* ribs, close to the sternum, in the right mammary region.

On the left side, at about the level of the *fourth* cartilage, the anterior edge of the left lung passes obliquely downwards, having abruptly left the edge of its fellow on the opposite side, so as to expose a free space of variable size for the heart (Fig. 10). The edge of the left lung thus reaches the *fifth* rib; whence it comes inwards and downwards to opposite the *sixth* rib or interspace, whence it finally passes nearly horizontally outwards and downwards into the lateral region. The anterior point of division of the lobes of the left lung lies about the fifth interspace, below the nipple. The left auricle and the left ventricle, with a small portion of the right ventricle about the apex, lie in the left mammary region; the apex of the heart generally lying immediately above the *sixth* costal cartilage (Fig. 8 and Fig. 16, under the description of the heart, a little farther on).

**The Infra-Mammary Region** has a somewhat triangular form. Its boundary alone is defined by the lower boundary line of the mammary region on each side: below, by the margins of the false ribs; inside, by the xiphoid cartilage or middle line; and outside, by the extension of the line from the acromial end of the clavicle to the outer tubercle of the pubes on either side.

On the right side this region contains the liver, with the edge of the lung overlapping it in front, to a variable extent during full inspiration.

On the left side the stomach and anterior edge of the spleen rise as high as the *sixth* rib in this region; and towards the inner portion of the region the left lobe of the liver lies in front of the stomach.

**The Supra or Post-Sternal Region** is a small hollow, bounded below by the notch of the sternum, and laterally by the *sterno-mastoid* muscles.

The trachea almost completely fills this region; but on the right side the innominate artery lies at the lower angle; and in some persons the arch of the aorta reaches its lower border just at the notch of the sternum, where it may be felt pulsating. The region contains no lung.

**The Upper or Superior Sternal Region** comprehends that portion of the breast bone which is superior to the lower border of the *third* rib.

It covers the left and a small portion of the right innominate

vein. The superior cava runs along its right edge; the ascending and transverse portions of the arch of the aorta; the pulmonary artery from its origin to its bifurcation; the aortic valves near the lower border of the *third* left cartilage at its junction with the sternum, or midway between the mesial line and the left edge of the sternum (the pulmonary valves being a little higher than these, more near the surface, and quite at the left of the sternum); and lastly, the trachea, with its bifurcation, on the level of the *second* ribs. The remains of the thymus gland, with areolar tissue and fat, lie in front of these parts, between the lateral pleural boundaries of the upper V-shaped portion of the anterior mediastinum, thus separating the edges of the lungs above towards their apices.

**The Lower or Inferior Sternal Region** comprehends the remainder of the sternum which lies below the level of the lower margin of the *third* rib. It contains the greater portion of the right ventricle, with the infundibulum of the pulmonary artery, and a small part of the left ventricle. The *mitral valve* is situated towards the upper end of this region, close to the left edge of the sternum, on a level with the *fourth* sterno-costal articulation; the *tricuspid valve* lies nearer the middle line, and more superficially. The edge of the right lung descends vertically along the middle line, and at the upper part of the region is a small portion of the left lung. Inferiorly and deeper seated is a portion of the liver, and sometimes of the stomach, while the line of union of the heart and liver corresponds with where the diaphragm intervenes.

**The Axillary Region** (Figs. 5 and 6, No. 7) extends from the apex of the armpit above to the line continued which marks the inferior border of the infra-clavicular region. In front it is bounded by the posterior border of the infra-clavicular region, and it extends to the external edge of the scapulæ behind on either side. The region can only be brought into view by lifting the arm over the head, or by carrying it away from the side (as in Fig. 6). The region is hidden (as in Fig. 5) when the arm is at rest by the side. It comprehends portions of the upper lobes of the lungs, a great volume of lung-substance, and, more deeply seated, large bronchi.

**The Infra-Axillary Region** (Figs. 5 and 6, No. 8) is bounded above by the region already defined; anteriorly, by the mammary region; posteriorly, by the scapula; and below it extends to the margins of the ribs.

It contains on both sides the lower edge of the lung, sloping downwards from before to behind. On the right side, also, is the liver, between which and the walls of the chest is interposed a thin layer of lung-substance during a full inspiration. On the left side is the spleen and stomach.

**The Supra-Spinous Regions** have the same boundaries as the superior fossæ of the scapulæ, and correspond to the posterior surfaces of the apices of the lungs (Figs. 7 and 9, No. 1).

**The Infra-Spinous Region, sometimes called the Scapular** (Figs. 7 and 9, No. 12). It is identical with the lower fossa of the scapula. It covers lung-substance.

**The Inter-Scapular Region** (right and left) lies between the inner

margin of the scapulæ, divided into a right and a left region by the vertebral column, from the second to the sixth dorsal vertebra.

It covers lung-substance on each side of the mesian line, the main bronchi, and bronchial glands. On the left side is the œsophagus; and from the *third* or *fourth* vertebra downwards is the descending aorta. The bifurcation of the trachea takes place at the middle line between the two regions, but inclining rather to the right side (Figs. 7 and 9, No. 13).

**The Infra-Scapular or Lower Dorsal** is bounded by the continuity of the transverse line, which forms the inferior boundary of the infra-clavicular region, and which, being continued behind, crosses the inferior angles of the scapulæ and seventh dorsal vertebra. It extends below as far as the twelfth rib, corresponding to the transverse line, carried round, which formed the lower boundary of the *infra-mammary* region in front.

As low as the eleventh rib lie the lungs. On the right side, from the level of the eleventh rib, extending downwards, is the liver. On the left is the spleen, occupying some of the outer portion of the region; while the intestines occupy some of the inner part of the region. Close to the spine, on the left side, is the descending aorta; and on both sides, close to the spine, is a small portion of kidney (Figs. 7 and 9, No. 14).

#### (B.) *Situation of the Organs in the Thorax.*

The *outer boundary* of each lung is marked by a line passing obliquely downwards and outwards from a little outside the centre of the clavicle towards the axilla, and then vertically at a variable distance outside the nipple. Each lung rises from half an inch to an inch and a half above the clavicle, the relative height being unequal but variable. The inner margin of each lung passes downwards and inwards from the apex, and meets with the inner margin of the other lung at the middle line, at a point between the first and second cartilages, or at the second.

*The inner margin of the right lung* continues vertically downwards along the centre of the sternum, or inclining a little to the left side, as far as the attachment of the xiphoid cartilage (Fig. 10).

*The inner margin of the left lung* leaves the right at a point between the *fourth* cartilages, or a little higher or lower than this, and passes nearly transversely outwards for a short distance in the direction of the *fourth costal cartilage*. It then runs obliquely downwards and backwards in the course of a line drawn from the centre of the *fourth costal cartilage*, half an inch to one inch inside the left nipple, as low as the seventh rib (Fig. 10).

*The lower boundary of the right lung* passes somewhat obliquely and then transversely from the attachment of the xiphoid to the sternum, across the cartilages of the sixth and seventh ribs backwards to the spine, which it touches on a level with the *tenth, eleventh, or twelfth* dorsal vertebræ (Fig. 9).

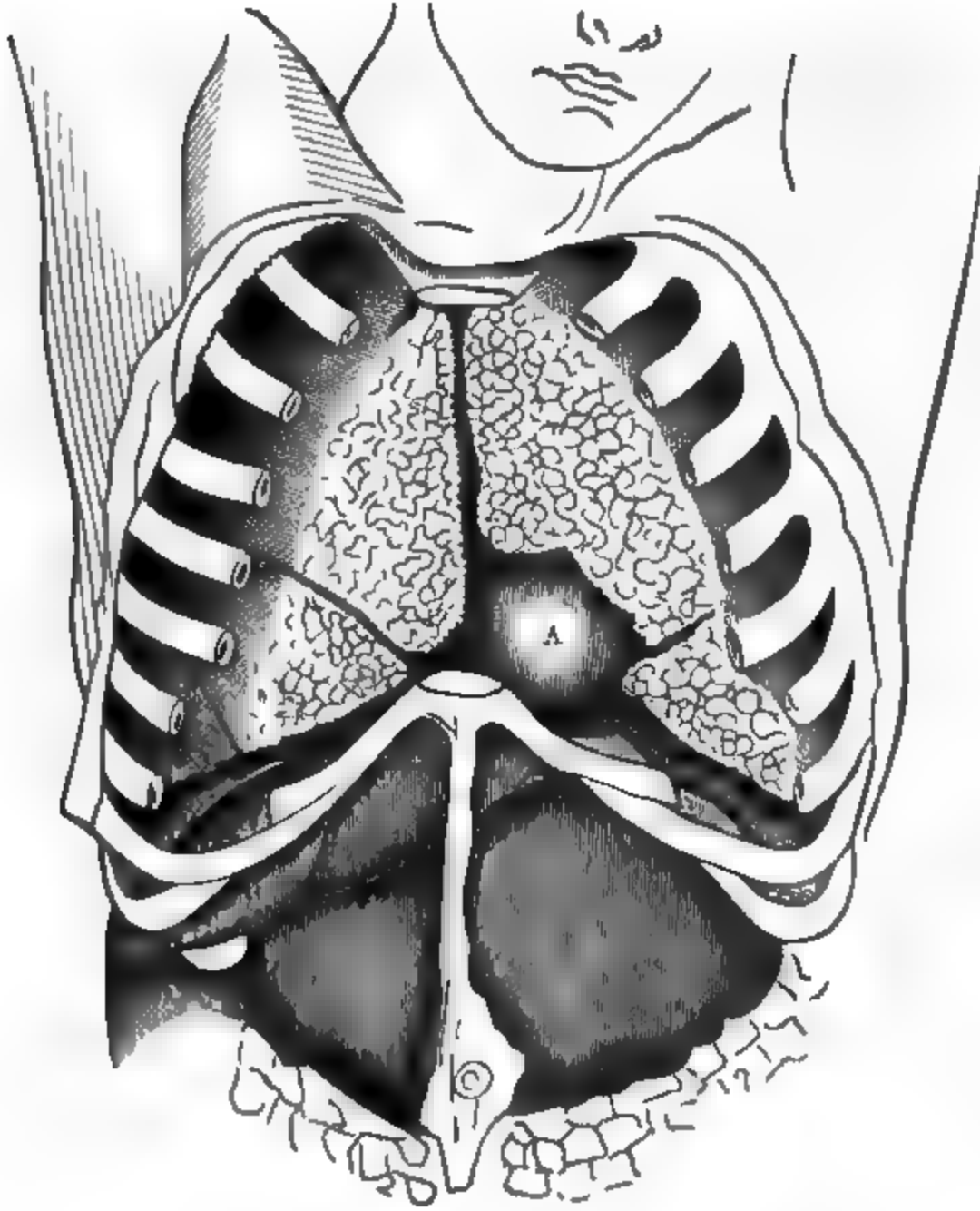
*The lower boundary of the left lung* is a little lower than that of the right, and passes backwards from the point indicated on the seventh

rib, to strike on the spine at a point usually a little lower than that on the right side.

*The apex of each lung* lies beneath the anterior scalenus muscle and the subclavian artery. The apices of the lungs are separated from each other by the œsophagus, the trachea, and the projection anteriorly of the bodies of the *first* and *second* dorsal vertebræ.

*The base of the right lung* is hollowed by the projection upwards

FIG. 10.\*



of the liver, which in the centre of the thorax ascends as high as the *fifth* rib or *fourth* interspace. The liver is also separated from the ribs by the expansion of the lungs between it and the thoracic walls.

*The base of the left lung* may be also pressed upon by the left lobe

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\* Relative positions of the margins of the lungs to each other, to the thoracic walls, and to A, the *præcordial* region, comprehending the right ventricle of the heart, covered by its pericardium (after DR. FULLER. See his work *On Diseases of the Lungs and Heart*).

of the liver; and it is always hollowed out, though to a less degree than the right lung, for the accommodation of the stomach and spleen, and, to some extent also, the left lobe of the liver.

The *heart* lies between the two lungs. The right auricle and a part of the right ventricle are covered by the right lung, the rest of the right ventricle being left bare by the divergence of the left lung from the middle line. The left auricle is covered by the right auricle and by the left lung. The left ventricle lies behind the right ventricle, but projects a little towards the left side, where it is uncovered for a short distance, beyond which its left border is covered by the left lung.

The region corresponding to the portion of the heart uncovered by the lung (Fig. 10) is sometimes called the *præcordial* region or space. The *upper boundary* of this space is where the inner margins of the two lungs separate—namely, at the spot between the *fourth cartilages*. The *outer boundary* of the *præcordial* space is indicated by the diverging line of the inner margin of the left lung passing along the *fourth cartilage*, and then obliquely downwards, inside the left nipple. The *inner margin* of the space corresponds to the nearly straight inner margin of the right lung, behind the sternum, near the middle line. The *lower boundary* of the *præcordial* space is indicated by a line passing from the junction of the sternum to the xiphoid cartilage, directly to the left, or with a slight inclination downwards. Above this line is the right ventricle, and farther out is the apex of the left. Below it is the left lobe of the liver, and the stomach, separated only from the heart by the diaphragm and the pericardium.

The *præcordial* region is thus slightly pyramidal in shape, its base being about two and a half inches long, and nearly horizontal. Its inner margin is about two inches long, and nearly straight; its outer margin is from three to three and a half inches long, and has a sloping direction from the apex to the pyramid (at the middle line between the *fourth cartilages*), to the outer extremity of the line indicating the base.

This *præcordial space* corresponds to the left half of the lower portion of the sternum, and to portions of the cartilages of the *fifth* and *sixth* ribs; and it may reach even to the junction of the cartilages with their ribs. Its inner and outer boundaries can be marked out only by light percussion; and the lower boundary can only be defined with difficulty by the same means.

Below the boundary of the right lung the liver extends to the margins of the right false ribs, or a little below them. On the left side the space between the lower border of the lung and the false ribs is occupied by the left lobe of the liver, the stomach, the spleen, and by a portion of intestine—the colon principally. The left lobe of the liver stretches across beneath the xiphoid cartilage, and below this to a variable extent towards the left side.

### (C.) *Changes in the Position of the Lungs.*

1. *In Health.*—(a.) During inspiration the lungs enlarge in all di-



rections; the apices rise higher and the bases descend lower down; the points of union and of division between the inner margins are in the one case raised towards the apices, in the other case lowered down. The *præcordial* space is thus lessened in size by the advance of the anterior margin of the lungs. (b.) During expiration the state of things is reversed. The lungs fall from each other; but the point of division between the inner margins may be raised to a level with the *third* rib. The area of the *præcordial* space is increased. (c.) The difference between extreme inspiration and extreme expiration is considerable. In extreme inspiration the inferior boundaries of the lung are often from an inch to an inch and a half lower than they are in extreme expiration. (d.) The action of the heart causes a slight difference. Each impulse presses aside the sloping inner margin of the left lung; but this is so instantaneous that it causes no appreciable alteration when the *præcordial* region is mapped out by percussion. (e.) During respiration the thorax enlarges in all directions by the movements outwards and upwards of the superior ribs and sternum, and by the movements downwards and outwards of the inferior ribs. *In women* the movements of the upper ribs are much greater than in men, while the abdominal movements are less. The difference is increased by the use of stays; but it does not appear to be altogether owing to these. In boys the costal movements are often considerable; in old age they are diminished.

2. *Changes by Age.*—In children the still considerably developed thymus gland separates the inner border of the lungs at the top of the sternum; and the point where they come in contact (converging from the apex) is lower than in adults. In children the lungs are also comparatively longer than in adults, and the inferior boundaries are lower down. In old people the lungs often alter considerably in shape, and produce corresponding alterations in position; the lower lobe, particularly in the left lung, becomes more posterior, and the upper lobes or lobe anterior. The lungs at last in old people become even larger above than below; and when mapped out by percussion, their several boundaries are found to have no certain and constant direction.

3. *Changes by Disease.*—One lung being incapacitated, the other lung undergoes supplementary enlargement; the inferior boundary is lowered, its inner margin is pushed to a variable extent across the median line over the heart, diminishing the *præcordial* space from the right or left side, according as the right or left lung is affected. So, also, if one lobe be affected, as by *pneumonia*, *tubercle*, or *cancer*, the other lobe, either upper or lower, as the case may be, is enlarged, and changes its position. In some diseases, as in *emphysema*, the lung enlarges; and if the *emphysema* is general, the lungs may meet each other almost at the top of the sternum; may not separate till on a level with the *sixth* rib; may leave the inferior margins at the *seventh*, or even the *eighth* rib; and may give a pulmonary percussion-note in the posterior lumbar regions, below the ribs altogether. In cases of enlarged heart, or distended pericardium (unless there be coincident *emphysema*, or unless the lungs

are floated forwards by *hydrothorax*), the lungs anteriorly are pushed aside, the point of separation of the inner margin is raised (especially in pericardial effusion), the inner margin of the right lung is thrown to the right side, and the inner margin of the left lung is thrown to the left side. Aneurism of the arch of the aorta, or tumors in the mediastinum, displace the upper portions of the lung; and tumors may even thrust aside and displace the heart. Abdominal diseases, as hepatic and splenic affections, peritoneal effusions, ovarian, uterine, or other tumors, also press up the thoracic organs, and alter their position. By such morbid states the inferior borders of the lungs may be not lower than the *third* intercostal space, or the *fourth* rib; the heart may be thrust upwards and outwards above and outside the left nipple. The student may also be prepared to meet with cases of more or less complete transposition of the viscera. (See a paper on this subject by Professor Allen Thomson, in the *Glasgow Medical Journal*, vol. i; also in the *Lancet* for August 8, 1863, by my colleague, Professor Maclean.)

## SECTION II.—SIGNS OF DISEASE FROM THE SHAPE OF THE THORAX.

The two halves of the thorax are seldom perfectly symmetrical. The right side in the most healthy persons often measures from *half an inch to an inch* MORE than the left; the right *infra-clavicular* space, particularly in right-handed persons engaged in laborious occupations, is apt to be slightly more prominent than the left; the *fourth, fifth, and sixth* left cartilages often project more than the right; the *infra-mammary* and *infra-axillary* regions may be larger on the left than on the right side; the *infra-scapular* region, on the contrary, is usually larger on the right side, or it may be markedly prominent on both. The nipples (which in adult males are above the upper margin of the *fourth* rib, near its junction with the cartilages) are always equidistant from the middle line, but the left is somewhat lower than the right;

Any marked changes beyond those now noted indicate disease either of the spine, as in *curvature*; or of the spine and ribs, as in *rickets*; or of the organs in the thorax. If *spinal curvature* and *rickets* are determined not to be present, any general expansion of one side as compared to the other, or general contraction, or (what is much more common) local expansion or bulging, or local contraction, depression, or excavation, become very important signs.

The existence of such conditions is ascertained by the eye—namely, by “*inspection*,” and accurately by measurement with calipers or with tapes, or ingeniously devised instruments which have been named “*stethometers*” or “*chest measurers*.”

General expansion of the thorax is usually produced by effusions of fluid into the pleura, or by extreme cancerous infiltration, or by emphysematous lungs. Much more rarely chronic consolidation will cause general or partial *bulging*; while tubercular deposition at its earliest period has been said also to cause some degree of local prominence; but *retraction* usually follows more or less complete

impairment of the functions of the lung, or of a part of it, as in softened tubercle or cancer, and in the period of absorption of pleuritic fluid which has firmly compressed the lung. Heart diseases give rise to local bulging only occasionally in the cardiac region. Aneurism of the aorta may cause bulgings, while tumors may produce both bulgings and retractions according to circumstances (*MS. Notes of Dr. Parkes' Clinical Course at University College*).

### SECTION III.—PHYSICAL EXAMINATION OF THE CHEST.

**I. By Simple Inspection of the Form of the Thorax.**—It is necessary to note,—

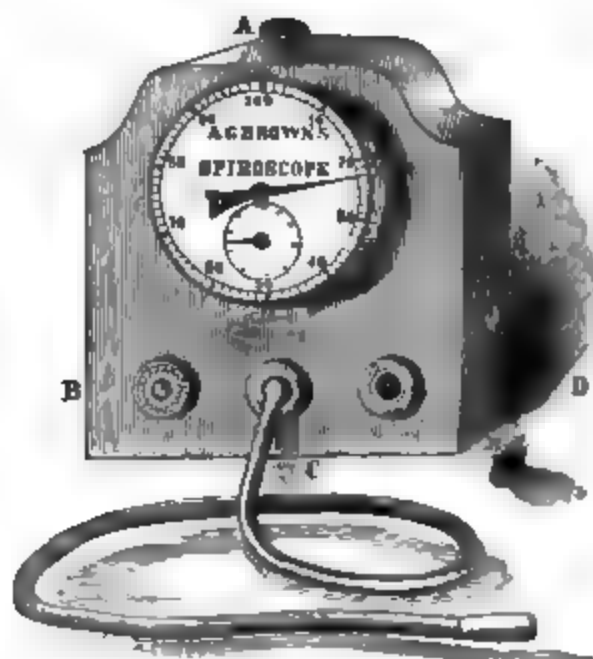
(a.) Its general shape; and especially as to the condition of the *supra-* and *infra-clavicular* spaces in respect of flatness, fulness, retraction, or bulging in these regions; the condition of the hollow above the notch of the sternum (the supra-sternal space); the form of the clavicles and their curvature; the height and breadth of the shoulders; the form of the sternum, as to whether or not it bends outwards or inwards; the curves of the spine; the position of the scapulæ; prominence of their inferior angles, and the firmness or laxity of the *latissimus dorsi* muscle; the distance of the scapulæ from each other and from the middle line; contracted contour or expanded form of the lateral regions; the width, depression, or bulging of the intercostal spaces; and distance of the nipples from the middle line.

(b.) Obtain a *general notion as to capacity or size of the thorax, relative to the height, weight, and age of the individual, allowing for fatness or emaciation* (Spirometry; see previous pages, under “*Scrofula*,” p. 215, vol. ii).

[By *spirometry* we measure the amount of air received into the lungs. The instrument of Mr. Hutchinson, mentioned at p. 241, is unwieldy, inexact, difficult to use, and is open to other objections. More available instruments, to determine the vital capacity of the lungs, have been recently invented. Mr. Coxeter's *spirometer* is compact. It is a bag of India-rubber cloth large enough to hold the air which may be expired by a person of large pulmonary capacity, fitted with a glass mouth-piece. An aperture communicates with another bag, the meter, holding, when filled, fifty cubic inches of air. Both the reservoir and the meter are provided with stop-cocks. The patient breathes into the first bag, with as prolonged an expiration as possible, and the stop-cock is closed. The air is then measured by refilling the meter until all the contents of the reservoir are expelled. Dr. W. E. Bowman's *spirometer* is on the same principle as Hutchinson's, but simplified and less cumbrous and expensive; it may be made of tin or of glass, and is said to answer the purpose very well (*Canada Lancet*, 1863). Mr. A. Gardiner Brown's *spiroscope* is a new and efficient instrument for ascertaining the breathing capacity. It is a wet meter, 6½ inches square, having a dial with two registers, revolving from left to right, marking in a complete revolution 100 and 1000 cubic inches respectively, and a few feet of vulcanized India-rubber tubing to breathe through. Its advantages are facility of management, compactness, portability, security of contained fluid, and it may

be used several times by the same person without readjustment. The air is measured at its initial temperature. The patient should be taught to practise a powerful inspiration, and as complete an expiration as possible, before noting the mean numbers registered in several trials. It should be placed at a convenient height for a person sitting or standing.

[FIG. 11.\*



The *hæmadynamometer* may also be used to determine the inspiratory and expiratory force. According to the investigations of Dr. W. A. Hammond (*Treatise on Hygiene*, 1863), men of five feet eight inches have the greatest amount of inspiratory power, raising the column of mercury two inches by inspiration and about three by expiration.

The *Lung Tester* of A. P. Barnes (to be had of Messrs. Codman & Shurtlef, of Boston), is the simplest and cheapest of all spirometric instruments. It consists of a cylindrical bag of India-rubber cloth, closed at each extremity by a disc of wood, and furnished with two metallic tubes; one tube enters laterally at the bottom, and is about three inches long; the other, vertical, is twelve inches long, and graduated, and inserted in the centre of the upper disc. A flexible tube of proper length, with a mouth-piece, is stretched over the outer aperture of the lower metal tube, and through this a forced expiration is made; the expired air fills, more or less, the bag, and the vital capacity is recorded on the upper tube, which is forced up, as the bag expands. The bag is inclosed in a tin cylinder, shut at both ends, with two holes for the tubes.

The *atmograph*, invented by Dr. J. Burdon Sanderson, of the Hospital for Consumption, London, is intended to record the respiratory movements in the same way as the sphygmograph does the minute differences and peculiarities of the pulse. A disc-shaped bag of thin India-rubber has cemented to the opposite sides two horizontal brass plates. The lower of these plates is glued to a board, and the upper is supported by

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\* [Fig. 11. The nuts A and B are to be unscrewed and boiled water is to be poured in at the orifice A, until it begins to trickle from the opening B; leave it for half a minute and then replace both the nuts with their leather washers. The instrument is now ready for use, and it will not need refilling for two or three months unless constantly used. The openings C and D are for the entrance and exit of the expired air. The free end of the tube is to be stretched over C, and the nut D should be removed.—EDITOR.]

a horizontal brass lever, against which two small springs act in opposite directions, also keeping the sides of the bag in position. This lever is attached to another, which bears a pencil at its extremity, by which the movements of the lever are traced on a revolving cylinder. A T-shaped tube communicates with the interior of the bag, the patient breathing through the horizontal bar. The changes of tension of the air in the bag, and their velocity, are recorded by the lever on the cylinder. The relative duration and intensity of the respiratory acts are thus registered, and an approximate estimate may be made of the amount of air breathed.

(1.) The tracings require no correction for instrumental error. (2.) It not only registers the relative duration and relative intensity of the respiratory acts, but the absolute quantity of the air breathed. (3.) It is so little disturbing to the patient that it may be kept in operation during sleep.]

(c.) Observe the thoracic movements, and estimate in seconds the time taken to complete the inspiratory and expiratory acts; compare the movements of the two sides of the thorax, and also the abdominal respiratory movements with those of the thorax, so as to notice if either takes an undue share in the work of respiration. If the ribs scarcely move, and the parietes generally of the thorax remain at rest, while the surface of the belly rises and falls alternately with the respirations, the act is called *abdominal respiration*, because the abdominal muscles seem to take the larger share in its performance; but if, on the other hand, no motion of the abdomen is visible, the act of respiration is then said to be *thoracic*. Observe whether the whole acts of respiration are quicker or slower than natural—i. e., more or less than *eighteen* to *twenty* per minute, or one to every four arterial beats. Note their frequency per minute. Observe whether they are calm, easy, and fully drawn, or short, forced, hurried, and incomplete, attended by indications of pain, checked or partially arrested by cough. Notice whether respiration is performed through the mouth or nose, or both; and whether the *nares* dilate and contract at each respiratory effort, with any constrained movement of forcible expansion. Note any sensible odor or vapor of the breath, and also its temperature.

II. **By Measurement.**—The use of tape or calipers in deep and medium inspiration and expiration will detect any differences between the two sides, or undue differences in the size of the chest at different times. There is about one inch of an average difference in favor of the right side of the chest compared with the left, and which is consistent with a normal state of the region. A convenient plan is the double tape, originally suggested by Dr. Hare. The double tape is formed by joining two common measuring tapes together, so that the beginning of each may be in the centre of the tape when joined. By putting the point of junction of the two tapes upon the spine, and holding it there tightly, the ends of the tape can be carried round the body at any point, and the circumference of each side read off simultaneously. By taking the size at full expiration and full inspiration, the extent of *expansion* is de-



terminated, as well as the absolute and relative size of each half of the thorax.

The measurements (besides the circular) which are most useful are the distance of the nipples from the middle line—their distance from the sterno-clavicular articulations of each side—and the distance of the centre of the clavicle from the lowest point of the false ribs in the vertical line.

The general expansion of a side of the chest is best learned by the use of the double tape, or by the use of some of the "*chest-measurers*" about to be noticed. The difference in the measurement between the fullest inspiration and the fullest expiration gives the general expansion of the lung. In health both lungs expand nearly equally from *three-quarters of an inch to an inch and a half*; or the right may expand a little more than the left. If there is any deficiency of expansion, there must be disease of some kind, but the nature of the disease cannot be known without additional signs.

Local expansion is most accurately determined by the eye, the hand, and the chest-measurers of Drs. Quain, Sibson, or Leared; the exact levels of the measurements being always noted. All these instruments essentially consist of dials, with indices, moved by mechanism, connected with tapes passing round one or both sides of the chest—so many revolutions of the index indicating on the dial so many tenths and hundreds of an inch of expansion of the chest. Dr. Leared's instrument indicates differences on the two sides (*Med. Times and Gazette*, August 2, 1862); and Mr. Henry Thomson, of University College, has also suggested a simple addition to the tape measure, by which it is made differential.

[Dr. Alfred C. Carroll, of New York, has had made by Tiemann & Co., a *stethometer*, which is ingenious, simple, and exact. There is an outer case, marked with a scale of three inches, *a b*, within which moves a slide

FIG. 12.



*c d*, bearing a gnomon or indicator, *d*. To the end of this slide is attached an ordinary measuring tape, the first three inches being cut off, so as to render the scale continuous, which is passed around the chest and drawn through the catch at the opposite end of the instrument. As the lungs are inflated, the slide is drawn out and the indicator shows the exact amount of expansion. A hard-rubber ring, *f*, sliding easily over the scale, may be used to render the stethometer self-registering. An elastic band, *e e*, draws the two portions of the instrument together; but this had better be removed before applying the instrument, as it may hinder the full expansion of the chest-wall (*New York Medical Journal*, vol. vi, 1868).]

Dr. Sibson gives the following numbers as denoting the movements of various part of the thorax in health:

(1.) The sternum and the first *seven* ribs in tranquil breathing advance forward from .02 to .07 inches. The left *fourth, fifth, and sixth* cartilages, and the *sixth* rib, move less than on the right side, on account of the position of the heart. In forced inspiration the movement forwards is from *half an inch* to *two inches*. (2.) The expansion of the *eighth* and *tenth* ribs varies from .05 to .1 inch. During deep respiration it is increased, but is less than that of the first five ribs. (3.) The abdomen moves forwards in tranquil inspiration from .25 to .3 inches. In deep inspiration the movement amounts to about *one inch*.

There is, however, very great variations in different persons; and "*chest-measurers*" must all be submitted (as *percussion* and *auscultation* are) to the test of comparison between the two sides—the difference which the heart causes between the two sides being remembered (Parkes, *MS. Notes*).

[The instruments above referred to measure magnitudes, movements, and capacity, but cannot measure deviations from the natural configuration of the thorax made by angles and curves formed by the bones composing the chest, by the junction of one bone with another, and of bones with cartilages. Though these departures from healthy configuration are of frequent occurrence in all parts of the chest, they are most common at the upper and front part, at the junction of the costal cartilages with the sternum, and at the junction of the costal cartilages with the ribs. These deviations are best determined by the *stetho-goniometer* of Dr. Scott Alison, which judges accurately the angles, and curves, and depressions of the chest.]

It is necessary in all these observations so to divert the patient's attention as to cause him to look away from the instruments, otherwise the movements of the chest may become so affected as to vitiate the results.

III. **By Palpation**, or the application of the hand. This method affords more extended information to that suggested by simple inspection, such as of the intercostal distances, lateral and antero-posterior; expansion of the chest in the acts of respiration; appreciation of *vibrations* communicated through the walls of the thorax. The palm of the hand applied to the chest in a healthy state during the act of speaking will appreciate a most delicate *vibratile tremor* (commonly called *fremitus*), and more marked according to the *graveness*, *coarseness*, and loudness of the speaking voice. The intensity is generally greater in front than behind, on the right side than on the left, and is stronger towards the sternal than the humeral halves of the region below the clavicles. In disease the *fremitus* on the two sides of the chest must be compared. It is usually *increased* by whatever consolidates the fine vesicular texture of the lung, without obliterating the bronchial tubes. It is *diminished* by the intervention of liquid or air between the lung and the thoracic walls; also by such extensive consolidation of the lung-substance as to fill up the smaller bronchial ramifications leading

to the air-vesicles. *Vocal fremitus*, or *vocal vibration*, is observed by placing the hand over the surface of the chest of a person speaking. It is a delicate vibration, easily deadened by the too forcible pressure of the hand. It is more marked in adults than in children, and in males than in females. It is more intense in long-chested than in short-chested persons; and markedly so in thin than in fat people; and is stronger in recumbency than in the sitting posture.

A *rhonchial fremitus* may sometimes be heard, when certain *rhonchi* throw the bronchial tubes into vibrations sufficiently strong to be felt on the surface of the chest. The sibilant, sonorous, and mucous rhonchi have all this property. *Friction fremitus* may be felt by the hand, when the gliding motion of the pleural surfaces upon each other is attended by a perceptible vibration, by the collision and friction of plastic matter upon the surfaces.

Ordinary fluctuation may sometimes be detected by the hands, combined with shaking or suddenly altering the position of the chest, as by *succussion*.

The hand is also used to appreciate the action of the heart. The heart, after full *expiration*, is felt to beat between the cartilages of the third and fifth ribs, and at the neighboring part of the sternum, generally immediately below and to the outside of the left nipple. After a full inspiration it may be felt as low as the sixth rib. The frequency of the respirations may also be ascertained by the hand, applied to the surface below the clavicles in the female, and below the epigastrium in the male (WALSHE).

IV. **By Percussion.**—The position of the organs, as stated in the previous section, is ascertained during life principally by the signs derived from percussion; that is, from the nature of the sound produced by striking over the lung either “immediately,” as when the chest is gently tapped with the hand or a light hammer; or “mediately,” as when a flat body is placed upon the chest, which is then struck by the finger or the hammer. The main object of percussion is to determine the comparative density of subjacent parts.

*Mediate* percussion is employed almost to the exclusion of immediate. The body which is interposed between the hammer and the chest is called a “*pleximeter*,” and consists simply of a flat piece of ivory or wood, or a piece of India-rubber, or of the index finger of the left hand laid flat upon the chest. The hammer is made of a piece of whalebone, or of light steel, armed at its head with a piece of India-rubber; or the flat edge of the stethoscope encircled by a piece of India-rubber; or it consists of a light thimble covered with vulcanized India-rubber; or the points of the index finger, or middle fingers of the right hand, may be used for the purpose of a hammer, the nails being cut close.

*Mode of Percussing.*—The pleximeter is to be placed flat upon the chest, and held close to the walls. It is then to be struck perpendicularly, and with gentle or moderate force. If the fingers of the right hand be used as the hammer, the blow should be given from the wrist only, the elbow and shoulder being quite motionless. The force of the blow must vary according to circumstances. In the supra-clavicular spaces the finger, if it be used as a pleximeter,

should be pressed closely into the triangular space, and the blow should fall in the direction of a line passing to the centre of the chest,—namely, downwards, forwards, and inwards. If the blow be directed too much forward or backward, it strikes on the clavicle, or the parts at the back of the neck, and the “pulmonary percussion-note” is not obtained. The clavicles are best percussed *immediately*, first at the sternal and then at the acromial end. In the *supra-spinous* spaces the blow should fall downwards, inwards, and forwards. Over the rest of the chest the pleximeter may be placed in any direction, provided the precaution is taken that corresponding points shall be struck on the two sides; as a slightly different note is given out, especially in thin people, by the intercostal spaces and the ribs. If the spaces are struck on one side, they should also be similarly, or with like force, struck on the other.

The “*pulmonary percussion-note*” is the sound given forth by striking on a part of the chest-wall below which lies a considerable depth of lung. The sound produced does not depend upon the proper tissue of the lungs, but upon the vibrations of the air they contain, and also upon the vibrations of the walls of the chest. The pulmonary substance vibrates but little; it is non-sonorous, and acts rather as a damper of sound. The qualities of sound to be noted are—(1.) *Amount in intensity of resonance*, commonly called *clearness*; (2.) *Duration of the sound*; (3.) *Pitch*; (4.) *Volume of sound*.

The intensity of sound is diminished by the lessened vibrations of the walls of the chest, as in pleuritic effusions; or by the lessened vibration of the air, the air being lessened in amount; or by increase in volume of the lung-substance, as by exudation.

The physical conditions which lessen the quantity of air are—(1.) Consolidation of lung-substance from any cause, such as exudation, tubercle, great congestion, or œdema; (2.) Compression of the lung from fluid in the pleura, or by tumors.

When the pulmonary percussion-note is exaggerated, it may become what is commonly termed “*tympanitic*,” that is, a very clear sound, of less volume, but of higher pitch than the pulmonary percussion-note. It is similar to the sound obtained by striking the abdomen when the intestines contain air, or a bladder moderately distended with air. It is generally caused by an increased quantity of air in the lung-tissue or in the pleura, as in pulmonary emphysema, or when cavities exist in the lungs in pneumothorax. A very thin and flexible wall will often give a tympanitic character to the pulmonary percussion-note. There are some exceptions to these statements, namely—(1.) When the air is not apparently in excess, a thin layer of sound lung full of air, lying over hepatized lung, will give forth the sound; (2.) The upper lobes of the lung when hepatized in the lower lobes will also give forth the sound; and (3.) When the lung floats on the surface of pleuritic fluid. *Tubular* sounds are produced when the lung is consolidated, covering bronchial tubes. In pleurisy the intensity of sound is diminished from both causes.

The sound is intensified or made more clear by whatever increases the vibrations of the walls of the chest, as in thin children,

or in persons becoming thin, and by increased vibrations of air in the lungs.

[The sound elicited by percussion of the chest in health varies with the region explored. The anterior regions give a much clearer sound than the posterior, on account of the difference in the thickness of the walls. But the degree of resonance varies in the several sub-regions anteriorly. In the supra-clavicular region the sound is very clear, and becomes almost tympanitic as the clavicle is approached. Over the clavicle the sound is clear in the middle third; in the inner third it is of high pitch; whilst towards the scapular end it is duller. In the infra-clavicular region—between the clavicle and the superior margin of the fourth rib—it is very clear and typically pulmonary; and perhaps somewhat less clear and shorter, but of higher pitch, on the right side. From the fourth rib downwards to the sixth or eighth rib on the right side, where the liver is reached, the sound, on strong percussion, is less clear, till it abruptly becomes flat. On the left side, from the fourth to the sixth rib, and from the nipple to the sternum, the sound is dull from the presence of the heart, but somewhat lessened in extent in cases where the lung overlaps the heart, as during inspiration, and in pulmonary emphysema. Below this the sound varies in different parts of the region, owing to the projection of the left lobe of the liver, the spleen, and the stomach, being dull on the inner and outer portions of the region, and often tympanitic, or of a ringing metallic tone, between these, from gas in the stomach. Laterally, the axillary regions on both sides are highly sonorous; the right infra-axillary region is clear to near the sixth or eighth rib, when it becomes suddenly dull on account of the liver, the line of hepatic flatness varying during inspiration and expiration; on the left side the percussion-sound is more or less dull from the position of the spleen, but when the stomach is distended with gas, it is tympanitic. Posteriorly, the scapular regions give a clear sound on percussion, though much less than the corresponding anterior and lateral regions; the infra-spinatal sub-region is more sonorous than the supra-spinatal. The infra-scapular regions give a very clear percussion-sound; on the right side the line of hepatic flatness is at or near the eleventh rib, but strong percussion will detect some dullness for an inch or more above; on the left side there may be tympanitic resonance from gas in the stomach, about the seventh rib, and at the ninth dullness from the spleen. In the inter-scapular regions the clearness and duration of sound are considerably lessened with increased resistance, owing to the mass of muscles.

**Auscultatory Percussion** was proposed by the late Dr. G. P. Camman, and Prof. A. Clark, of New York (*New York Jour. of Medicine*, 1840). The chest is struck, whilst the ear is applied to the stethoscope in contact with the chest. By this means, it is thought, percussion-sounds are more distinctly heard, and their special characters more accurately distinguished. It is most useful in nicely determining the boundaries of the solid organs, as the heart, liver, and spleen. Dr. Flint says: "By means of auscultatory percussion, using Camman's stethoscope, the pectoral extremity being brought near to the open mouth of the patient, amphoric and cracked-metal resonance may often be ascertained when otherwise they would not be heard" (*Pract. Treat. on Phys. Exploration of the Chest, &c.*, 2d ed., 1866).]

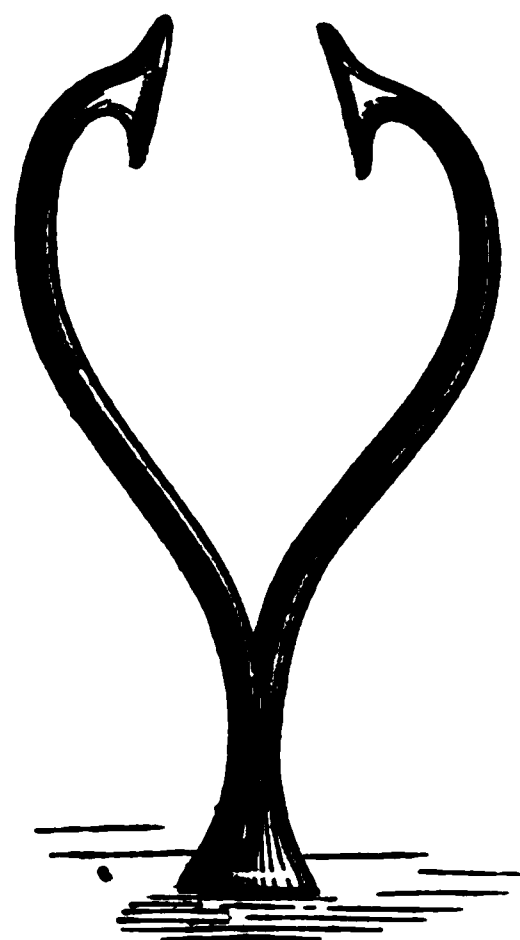


V. **By Auscultation**, which implies "the act of listening," and is termed pulmonary or cardiac according as the sounds listened to relate to the lungs or to the heart. For this purpose the ear may be applied to the surface directly, merely interposing, for several obvious reasons, a fine towel or thin piece of linen or calico between the chest of the patient and the ear of the observer; or an instrument called the *stethoscope* may be used, and which is especially necessary when the sounds to be heard are limited to a small and definite region, as in listening to the sounds of the heart. The ear-piece of the stethoscope ought to fit the ear of the observer; or the ear-piece may be removed, so that the end of the stethoscope may be introduced into the orifice of the ear. It is important that the ear be well fitted. The extremity applied to the patient should be about an inch and a quarter in diameter.

A double or bin-aural stethoscope is also an instrument of great value in the differential diagnosis of sounds associated with lesions in the thoracic organs, especially when the sounds are obscure from their lowness. One form of the instrument seems to be an efficient magnifier of low sounds (LEARED), and another form of the instrument—that of Dr. Scott Alison—is extremely useful in cardiac diagnosis when the action of the heart is rapid or irregular (GAIRDNER). When the first sound is indistinct at the apex, or cannot be identified with the apex beat, or when the second sound is indistinct, or when it is audible only at the base, the first sound being audible only at the apex, the difficulty of recognition of the two sounds is very considerable. It is in such circumstances that the double or bin-aural stethoscope of Dr. S. Alison is of great value, for it enables the sounds to be identified at the points where they can be heard best; and it brings them into accurate relations with each other, because of the two ears receiving at the same time the sound from two points of the *præcordial* region (GAIRDNER).

Dr. Arthur Leared, of London, devised and exhibited a double stethoscope in the Great Exhibition of 1851 (Class X, No. 620), where it attracted little attention by the profession in this country. It consists (Fig. 13) of two tubes, one for each ear, made of *gutta percha*, the elasticity of which, acting as a spring, keeps the tubes firmly applied to each ear. The thoracic ends of the tubes are fitted into a hollow cylinder or cup, which is applied to the surface of the chest. Dr. James E. Pollock, of London, called the attention of the profession to an American double stethoscope, in the *Lancet* of April 12, 1855, at the request of his friend Dr. Coulson, neither of

FIG. 13.\*



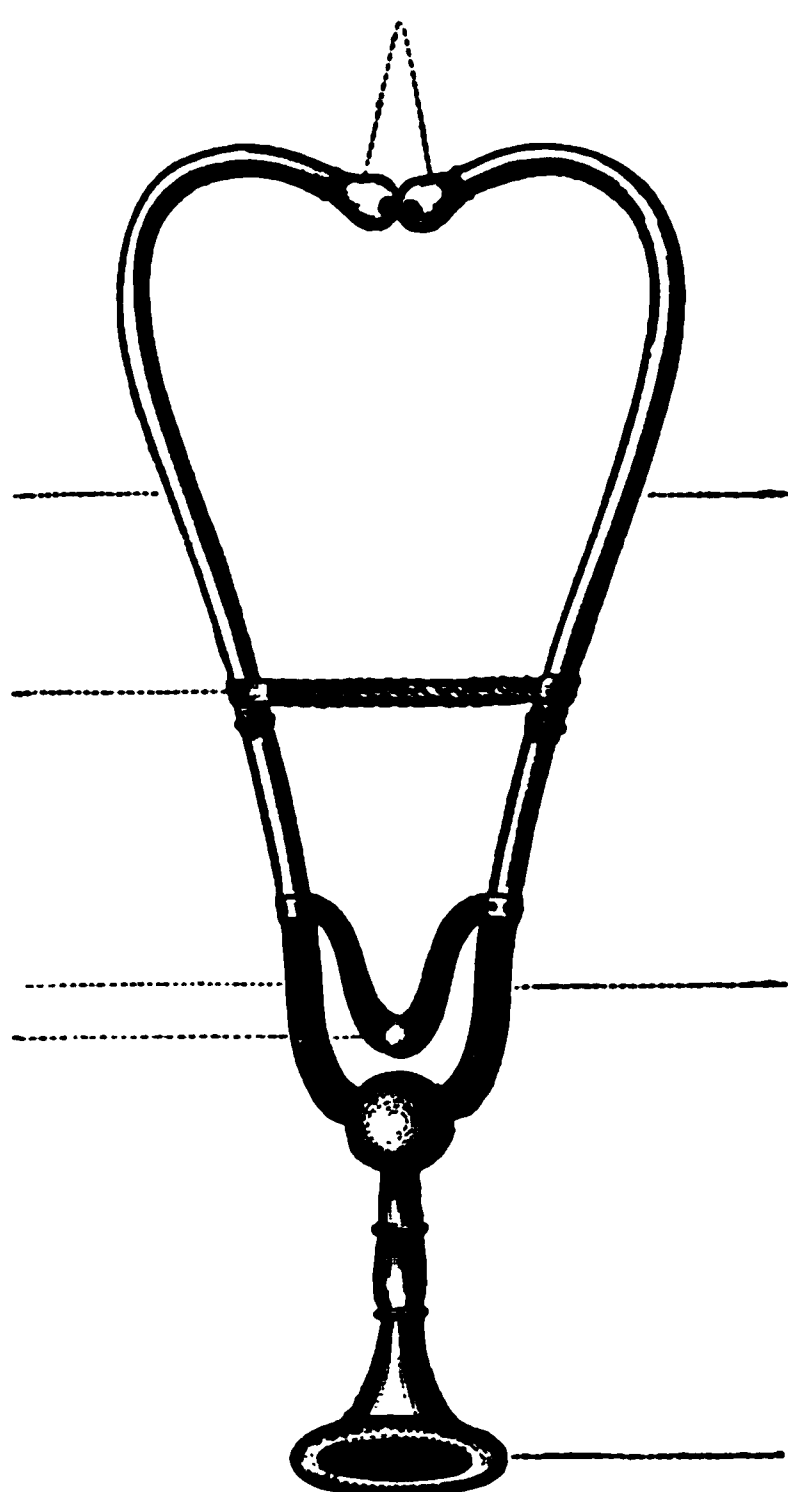
\* The Original Double Stethoscope of Dr. Leared.

whom were aware that Dr. Leared had devised and exhibited such an instrument in 1851.\*

\* From discussions which followed in the medical journals, I have been led to make a statement in previous editions which does an injustice to Dr. Camman; and I am glad to be able to insert this note from the American reprint of this text-book. The Editor of the reprint writes as follows:

"The writer (Dr. Clymer) is indebted to Dr. James R. Leaming, of New York, the literary executor of the late Dr. Camman, and his clinical assistant at the time named, for the following facts: In the spring of 1852, Dr. H. W. Browne brought one of Marsh's patent double stethoscopes to Dr. Camman's class at the Northern

FIG. 14.<sup>1</sup>



Dispensary. Dr. C. at once said that the principle was not a new one, and that he had at the time one of Landouzy's poly-stethoscopes, sent to him from Paris, which was on the bin-aural principle. He also observed that it was a shame for a medical man to patent an instrument intended for the profession. He then proposed to his assistants to begin a series of experiments for the purpose of perfecting a stethoscope which would render the bin-aural principle practically available. With the assistance of Drs. H. W. Browne and C. P. Tucker, and the mechanic employed, the instrument known as 'the self-adjusting bin-aural stethoscope' was in the summer of 1852 perfected, and freely given to the profession without annoying restrictions. Soon afterwards he received a letter from Dr. Marsh, threatening a prosecution for the infringement of his patent. He took this letter to his lawyer, A. P. Mann, Esq., and authorized him to answer it, denying Marsh's claim to the discovery of the bin-aural principle; and, as the two instruments were widely different in construction, refusing to make any terms or compromise, and expressed a willingness to contest the case at law if necessary. Thus not only did Dr. Camman not patent the admirable mechanism of his stethoscope, which was clearly his own invention, but placed it freely in the hands of the profession, and stood ready to defend his right to do so. He had not been in Europe since 1830, and consequently could not have carried home Dr. Leared's idea (1851); nor is it probable he ever heard of Dr. Leared's

stethoscope. He never called it 'Camman's Double Stethoscope;' this was done by the instrument-maker.

"It has been said that he did not appreciate the value of his own instrument, possibly because he did not assert his claim to its originality, and from the fact that after the first year of its introduction he used it sparingly, finding that its constant application to the ear sensibly dulled the delicate acuteness of hearing so prized by the expert.

"To understand the *rationale* of this effect, it is well to premise that the double stethoscope conveys the same sound-impression through a distinct channel to each ear, just as the stereoscope presents an individual picture of the same object to each eye; and the effect in each case is to produce in the brain a clearer perception of the entirety of the sound heard or the object seen, than can be done by one ear or one eye alone; but no instrument can do this perfectly unless both ears or both eyes are

<sup>1</sup> Self-adjusting Bin-aural Stethoscope of Dr. Camman.

Another form of double stethoscope has been devised by Dr. Scott Alison, and named by him the "differential stethoscope." Like Dr. Leared's instrument, it has a tube for each ear; but each tube has a separate cylinder or cup, which admits of being applied to any part of the chest. Dr. Leared's instrument enables us to hear sounds emanating from any given portion of the chest with both ears simultaneously; while Dr. S. Alison's instrument enables us to listen to the sounds emanating from two different parts of the chest at the same moment, so that we may compare the sounds at any two points, or on opposite sides of the chest, by a series of consecutive observations. If two sounds exist, one at each point, and if they differ in intensity, the weaker sound is eclipsed or nullified.

All forms of stethoscope require much practical study and experience to use them with success. The ear-piece should fit the ear exactly in all forms of stethoscope; "and, therefore, when a well-made stethoscope has been selected and an ear-piece chosen which fits the ear comfortably, the student should keep to that one, and familiarize himself with its use" (FULLER).

The main object of the stethoscope is to *circumscribe* and localize the sounds that we hear; the chest end of the instrument should therefore be as small as possible, in order the better to appreciate the precise seat of the greatest intensity of sound. To ascertain this, the instrument should be "worked towards the sound," and then "from the sound," right and left, up and down, till its end is on the exact spot whence the sound proceeds in its greatest intensity. By working the stethoscope round and round, and gradually contracting the circle, the *area* of the sound can be ascertained; and hence so far the lesion producing it can be localized (HYDE SALTER).

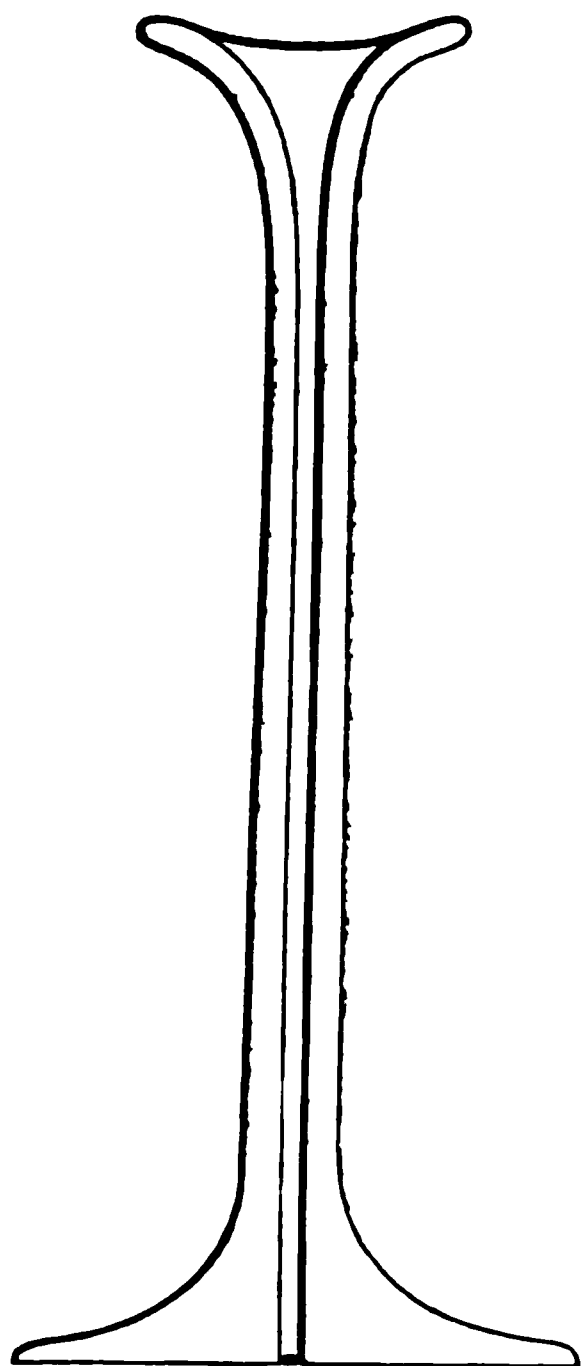
Four circumstances affect the quality of a stethoscope: (1.) *The material which allows the least amount of sound to be lost, and least of all perverts or modifies the sound*, is the best for a stethoscope—namely, some *porous* wood which is a good conductor of sound. Cedar and deal are the best woods for the purpose. The denser the wood the more are the sounds apt to be modified; therefore ebony is to be condemned. (2.) *The stethoscope should be of one piece of wood*—turned, in fact, out of a solid block. It ought not to be

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equally good. In ordinary sight or hearing, the eyes and ears being dual organs, one rests while the other is in action. Any one looking through the stereoscope a short time will be sensible of fatigue and temporary impairment of sight; and should it be too long and too constantly continued, the injury would probably be permanent; and the rule holds equally good in using the bin-aural stethoscope, for sight and hearing are cognate senses. It was a practical knowledge of the evil effects of a too constant application of the double stethoscope that caused Dr. Camman to restrict its employment, believing it more important to preserve the delicate sensitiveness of the expert ear, than that the sound-impression should be made with its utmost power. It is gratifying to know that there are those in England who appreciate Dr. Camman's unselfish labor. Dr. Alison, in his work *On the Chest*, pp. 322, 323, and 324, gives a description of this instrument, accompanied by a wood-cut, and accords to Dr. Camman the full credit of rendering the bin-aural principle available. In describing the construction of his own instrument—the differential stethoscope—he acknowledges his indebtedness to Dr. Camman in adopting the mechanism of his instrument, which he pronounces beautiful."

part ivory and part cedar. (3.) *As to length and thickness*, the length most convenient is that which permits the instrument to be carried in the crown of one's hat; and it may be solid if the wood is very porous; but generally it is hollow. (4.) *The chest end* should be small, not larger than *one inch and a quarter* in diameter; because the smaller it is, the greater is its localizing power, and the narrower are the limits within which the seat of any particular sound can be determined. The chest end of the stethoscope should also be narrow, and smoothly rounded over the edge. (5.) *The ear-piece* should be *large and flat*, to secure apposition and occlusion; but in this respect each man must fit his own ear (Hyde Salter "On the Stethoscope," *Brit. Med. Journal*, January 31, 1863). The wood-cut (Fig. 15) represents the section of a good stethoscope as given by Dr. Hyde Salter. It is half the "natural" diameter—i. e., one-eighth the size. The ear-piece is flat and broad, and the most careless application of the ear would produce perfect occlusion; the

FIG. 15.\*



chest end is small, with a narrow and rounded edge. The measurements are, length, seven inches; diameter of ear-piece, three; diameter of chest end, one and a quarter; circumference of shaft, one and a quarter.

Dr. C. L. Hogeboom, of New York, has suggested an improvement in all stethoscopes, by stretching evenly and tensely over the pectoral extremity a piece of parchment, so as to be in contact with the skin, and sufficiently firm and elastic to compress the tissues and transmit sonorous vibrations. He claims that it intensifies the sounds, and that their source is more circumscribed. In Camman's stethoscope it lessens the roaring. To auscultation of the larynx and trachea it is peculiarly adapted, as well as to that of the heart (*New York Med. Jour.*, vol. iii, 1866).

To become familiar with the knowledge to be acquired by means of auscultation, much time and labor must be devoted to its practice, alike on persons in health and in disease. A verbal description of the sounds to be learned is difficult, because the impressions made

on the senses of one person cannot be communicated exactly by language to another; and the distinctions which subsist between the sounds heard in health and those in disease are not yet regarded as similarly significant by all; nor is their individual importance yet clearly determined in relation to practice.

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\* Section of a good form of Stethoscope (HYDE SALTER).

[**Auscultation** is either *immediate* or *mediate*; *immediate* when the ear is placed in direct contact with the chest, and *mediate* when a conducting medium—the stethoscope—is placed between the ear of the listener and the chest, through which the sounds produced within the chest-walls are transmitted. Immediate auscultation is simple and easy, and the chest can be more quickly, and, in many cases, as satisfactorily, explored as by mediate auscultation. In females the anterior regions of the chest, and the supra-clavicular and axillary regions in both sexes, are better examined with the stethoscope. The delicacy, fastidiousness, or filth of the patient, may make the mediate method more desirable. When it is needful to localize precisely the morbid sounds, and when heteromorphisms exist, the stethoscope should be used. Children are frightened by the production of an instrument, and for them, as a general rule, the naked ear is preferable. Each method has its advantages, and neither should be exclusively and indiscriminately used; and the student should make himself familiar with both kinds. The distinctness and precision with which the sounds are heard depend on the training of the auscultator. In practising auscultation both patient and explorer should be placed in a position where there is the least constraint for both. The chest should be free of all clothing, and a thin towel, for the sake of cleanliness or decency, should be thrown over it. When possible, the patient ought to sit in a high-backed chair for an examination of the anterior regions, the arms hanging loosely at the sides. To explore the posterior regions, the position in the male may be reversed, with the body slightly bent forwards, and the arms lightly crossed; the female may sit in this position of the body and arms on a stool, or sideways on a chair. For examination of the lateral regions, the arms must be raised over the head. When the patient cannot leave the bed, he should, if able, sit up; if too feeble, a partial exploration of the posterior regions may be made by turning him over on his side. Fatigue and annoyance must be carefully avoided. In *immediate* auscultation the ear should be firmly and accurately applied to the surface, the pressure not being too light or too strong, and the examiner must not stoop, or bend his head too much; this rule applies to both methods of exploration. Both ears should be drilled, so that they may be used indiscriminately. In *mediate* auscultation the stethoscope is to be evenly and closely applied to the surface of those parts of the chest to be examined, but not with too much pressure: the least tilting is to be avoided.

To practise understandingly auscultation, the examiner must be familiar with the natural respiratory and vocal sounds, in order that he may detect any modifications of them, which constitute the physical signs of disease. The healthy *vesicular murmur* is a diffused, soft, breezy, sighing sound, not to be described, whose probable site of production is the air-sacs of the lungs (HYDE SALTER), and is divisible into two times, or sounds, inappreciably separated,—the *inspiratory* and *expiratory murmurs*. In a certain number of persons—about one-third—the expiratory murmur is wanting most frequently on the left side; it is from three to five times shorter, less intense, and of lower pitch than the inspiratory murmur; its duration is increased in old persons, while its intensity is less; it is more prolonged in the right clavicular region than in the left (GERHARD, LOUIS). The intensity of the vesicular murmur varies in different healthy persons of the same age, sex, conformation, and like general condition; but age and sex modify it: it is loud and well-marked in infancy and childhood,—*puerile respiration*; becomes qualified in adult age, and in old persons is frequently very feeble,—



*senile respiration*; generally it is greater in the female than in the male, especially in the upper chest regions. With regard to the degree and character of the inspiratory murmur in the several regions of the chest,—in the clavicular regions it is generally stated to be of greatest intensity on the right side, but Dr. Flint says, from his own observations, that though its pitch is higher on the right side, its intensity is almost invariably greater on the left. Not unfrequently the expiratory murmur is prolonged on the right side to quite or nearly the length of the inspiratory, and its pitch on that side is sometimes higher. Dr. Flint also asserts, on the right side the two murmurs are occasionally separated by an appreciable interval. In the sterno-clavicular portion of the infra-clavicular region, it is apt to be notably modified by sounds produced in the superficial bronchial tubes and trachea. In the *mammary* and *infra-mammary* regions, the inspiratory murmur is appreciable, but less intense, lower in pitch, and softer, than in the clavicular regions, and the expiratory murmur is rarely heard. In the *supra-spinal scapular* region the respiratory murmur is less intense than anteriorly, and the inspiratory murmur is sometimes more marked on the left side, while the expiratory is more prolonged on the right. In the *infra-spinal scapular* region, the murmur is more intense than in the upper region, but less in degree than in front. In the *interscapular* region, owing to the nearness of the large superficial bronchial tubes, the murmur is decidedly bronchial, like that heard over the inner third of the clavicle. In the *axillary* regions the inspiratory sound is very loud, especially in the upper part, and the expiratory murmur is much more frequently distinct than in the middle- and inferior-anterior or posterior regions.

When the stethoscope is placed over the supra-sternal fossa, a sound like that produced by air driven forcibly through a tube of a certain calibre is heard, and may be divided into two times, one coincident with inspiration, the other with expiration, and separated by a brief interval. The quality of both sounds is peculiar and characteristic, and is said to be *tubular*; the *inspiratory* sound is of higher pitch than that of vesicular respiration, and the *expiratory* is intenser, longer, and higher in pitch than the inspiratory; this is *tracheal respiration*. *Laryngeal respiration* is said by many writers to differ greatly from tracheal respiration, but according to the observations of Dr. Flint, the difference is limited, as a general rule, to intensity; in other respects they are essentially the same (*loc. cit.*, p. 131).

**Auscultation of the Voice.**—The sounds of the voice are transmitted through the chest, modified by the size of the tubes, and the nature of the substance through which they pass, and become signs of the condition of the organs transmitting them. In vocal auscultation Camman's stethoscope or the naked ear should be used, and the patient instructed to count *one, two, three*, slowly, distinctly, and moderately loud, and to repeat these numbers as often as is necessary. Over the trachea the voice seems concentrated and coming into the ear of the explorer; it is more or less distinct, resonant, and gives the sensation of a peculiar shock and fremitus; this is called *tracheophony*. The phenomenon of the direct entrance of articulated words into the ear at the point auscultated is called *pectoriloquy*. When the stethoscope is placed over the thyroid cartilage, and the patient is directed to speak, the voice will be found to be transmitted generally with less intensity, shock, and vibration than from the trachea; this is named *laryngophony*. On listening over the chest, either immediately or mediately, to the sound of the voice, articulated words are not heard, and the resonance is less intense than

over the trachea, is more diffused, seems farther from the ear, and usually the shock is wanting; and over certain parts of the chest, in many instances, there is no fremitus; but differences exist in different persons, and in the several regions, and in the corresponding regions of the two sides. Over the first divisions and subsequent larger subdivisions of the trachea,—the larger bronchial tubes,—as on each side of the upper part of the sternum, at and between the scapulæ, and in the axillæ, there is still considerable resonance, though diffused and distant, and frequently some fremitus; this is natural *bronchophony*, or bronchial resonance.

The *whispered voice* and its modifications, have been little studied by writers. Dr. A. Flint has lately called attention to the subject, and thinks that the sounds made by whispered words will prove to be an “important addition to the physical signs available in diagnosis.” A whispering sound is the sound produced by a forcible act of expiration (*Physical Exploration of the Chest*, 2d ed., p. 147).

**Auscultation of Children.**—Very young children should be held in the nurse’s arms, so that the part of the chest to be examined may be applied against the ear of the auscultator; or the examiner may place his hand beneath the chest of the child and raise it,—the head and pelvis not being supported, the chest becomes prominent, and the ear is conveniently applied. The manner in which the child breathes should be first carefully noticed, that errors may not be made, and the sounds which are produced in the pharynx and nose may be set aside. To ascertain the amount of vocal resonance the chest should be explored while the child is crying, the cry replacing in young children the voice of the adult. The maximum of intensity of the respiratory murmur is, anteriorly from the clavicle to a little below the nipple, on the right side, and not quite so low down on the left, and posteriorly the inferior part of the interscapular region; the minimum, anteriorly, is the præcordial region, and posteriorly the supra-scapular regions.

**Auscultation in Disease.**—The modifications of the *respiratory murmur* in diseased conditions of the lungs are changes in its duration and intensity, rhythm, and special character.

Duration and Intensity,	{ Exaggerated or puerile. Weak or senile. Suppressed.	
Rhythm, . . . . .	{ Incomplete. Jerking. Divided. Prolonged expiration.	
Character, . . . . .	{ Harsh. Bronchial or blowing. Cavernous. Amphoric.	{ Diffused. Tubular.

(1.) *Exaggerated*, called also *supplementary* (ANDRAL), *puerile* (LAENEC), and *hypervesicular respiration*, is simply increased intensity or loudness without change in quality, pitch, or rhythm. It has been stated that the degree of the respiratory murmur varies in different persons, and in the same regions of the two sides of the chest, and this should be remembered in estimating its value as a sign of disease.\* When accompanied by a greater number of respirations than natural, decidedly

\* [According to Walshe and Fournet, when exaggerated respiration is a sign of disease the excess of intensity is on the side of expiration, whilst in health it is on the side of inspiration.]

heightened in the anterior and superior regions, or diffused over one side, it is indicative of disease in a portion of the lung more or less distant, or of the opposite side. It is most frequently met with in solidification of the lung, as in pneumonia, large tubercular deposits, carcinoma, pulmonary apoplexy, or where a considerable portion of lung is deprived of air from a mechanical cause, as the pressure of an enlarged bronchial gland or air-tube, blocking up of the bronchi from plastic exudation, and in pleurisy.

(2.) *Weak or senile respiration*, tokened by diminished intensity, or feebleness of, the respiratory murmur, its other characters remaining the same, happens in a number of pulmonary disorders, and is a sign of much value, often marking the site of the lesion. It may be close to the ear, *superficial*, or more or less distant, *deep-seated*. It is generally persistent, though it may be intermittent. The expiratory sound is rarely heard, except in emphysema, where along with feebleness of the murmur it is often distinctly prolonged. The murmur may be actually diminished in degree from imperfect production, or the effect may be caused from its reaching the ear through some solid or liquid between the lung-surface and the chest-walls. It is caused by (1) an obstruction in the air-tubes, as in laryngeal disorders or foreign bodies in a primary bronchus, narrowing of the bronchi from inflammation and its products, spasm or permanent contraction of the air-tubes, and their compression by tumors, enlarged bronchial or lymphatic glands; (2) obstruction of the air-cells, in pulmonary tuberculosis, œdema, pneumonia, hemorrhage; (3) overdistension of the air-cells, in emphysema; (4) gaseous, liquid, or solid effusions into the pleural sac; (5) impaired thoracic movement caused by pain, as in acute pleurisy and intercostal neuralgia, or from paralysis of the respiratory muscles, as in general paresis and hemiplegia. When weak respiration is limited to the apices of the lungs, and is accompanied by diminished resonance, it generally denotes tubercles; heard in the anterior, superior, and middle regions, with increased resonance, it is a sign of emphysema; and at the base of one or both lungs, and remote, with more or less dulness on percussion, it indicates pleuritic effusion.

(3.) *Suppressed respiration* is the entire absence of the respiratory murmur. It may be permanent or intermittent; and is met with in the same pulmonary conditions as weak respiration. It is chiefly valuable as a sign of pleuritic effusion, single or double.

(4.) *Incomplete respiration* is lessened duration of the inspiratory murmur, and is of two kinds: (*a*), when its beginning is abridged (*deferred inspiration*), and it is not heard until an appreciable interval elapses after the commencement of the inspiratory act and the air-cells are fully distended, and then as a short wheeze, as in emphysema, bronchitis, pleuritic effusions; (*b*), or it may be initially evolved, but abruptly stopped before the inspiratory act is ended, with a sort of hitch (*unfinished inspiration*); its site is in the bronchioles.

(5.) *Jerking, interrupted, wavy, cogged-wheel, respiration* (*inspiration entrecoupée, respiration saccadée*) is when the respiratory murmur, instead of being continuous, is broken and whiffy. When partial, it is the result of incomplete dilatation of the air-cells from some cause or other. Its site is almost always the apex of the lung anteriorly, and oftener on the right than the left side. It is thought by many to be a valuable sign of incipient phthisis. When general, it is due to sudden arrest in the dilatation of the chest-walls from pain or deficient innervation, as in asthma, pleurisy, plurodynia, palsy. It is also met with in pleuritic adhesions.

(6.) *Divided respiration*,—a distinct interval elapsing between the

inspiratory and expiratory murmurs,—is caused by over and permanent dilatation of the cells hindering the expulsion of the air, and is usually heard in the middle regions anteriorly, as in emphysema.

(7.) *Prolonged expiration* may be classed under the modifications of rhythm, when it is the only or chief alteration in the respiratory murmur, and there is no change in degree or pitch; it is a diagnostic sign of much significance if properly estimated. Natural prolonged expiration happens in a certain number of persons, but only on the right side. Its value as a morbid sign is measurably coincident with its site. When heard over a limited space in the upper chest regions it may indicate tuberculosis; when more general, and heard in the middle regions, emphysema; or it may token only temporary obstruction or compression of the air-cells.

(8.) *Harsh, rude, broncho-vesicular* (FLINT), *vesiculo-bronchial*, (DA COSTA) *respiration* has less of the vesicular quality than the healthy respiratory murmur, being less soft, of higher pitch, more blowing, and not so equable. In inspiration, the vesicular and tubular qualities are united, the duration short, the pitch more elevated, and the intensity variable; expiration, sometimes wanting, is, when present, always prolonged, of higher pitch, and sometimes of greater intensity than inspiration, to which it succeeds after an appreciable interval (FLINT). It denotes a certain amount of pulmonary condensation, from deposition or compression, and happens in phthisis, pneumonia, pulmonary apoplexy, cancer, fibroid degeneration, melanosis, œdema, and in pleural effusions of a certain amount. For its presence, the degree of condensation of the lung should not be sufficient to abolish vesicular respiration, which must to some degree, however slight, be heard in inspiration.

(9.) *Bronchial, or blowing, respiration* is in all respects the same as natural laryngo-tracheal respiration, and in certain pulmonary disorders takes the place of vesicular respiration. Its production and course are rapid. Inspiration is tubal, of short duration, incomplete, and of high pitch. Expiration is nearly or quite as long as inspiration, and sometimes longer, of greater intensity, and higher pitch. From the sudden interruption of the inspiratory murmur, a distinct interval occurs between it and the beginning of the expiratory. It is always associated with condensation of the lung-tissue, and is heard in phthisis, pneumonia, pleurisy with effusion, uniform dilatation of the bronchi with induration of surrounding tissue, &c. It may be *diffused* or *tubular*.

(10.) *Cavernous respiration* resembles the sound produced by blowing into a hollow space; inspiration is of slow production, of low pitch, and not tubular in quality; expiration is of lower pitch than inspiration. Its common site is the superior regions. It betokens a pulmonary cavity, or globular bronchial dilatation.

(11.) *Amphoric respiration* gives the sensation of blowing into a large cavity with thick walls filled with air, and is imitated by blowing gently into a narrow-necked glass bottle. It is distinctly metallic and musical. It is caused by the air in the bronchial tubes acting on the air in the cavity. It may accompany both respiratory sounds, but is most often heard in inspiration; it is generally circumscribed or only partially diffused. It is heard in pneumothorax with pulmonary fistula, and large tubercular cavities.

**Rhonchi.**—In pulmonary disorders we meet with certain accidental sounds, caused by more or less disturbance of the natural respiratory process, and produced either in the air-tubes, the air-sacs, or in cavities formed in the lung-tissue in the course of disease; these endo-pulmonary sounds

are called *rhonchi*, *râles*, or *rattles*, and may be dry, moist, or of an indeterminate character.

Dry or Vibrating Rhonchi,	{	High pitch, . . . .	Sibilant.
	{	Low pitch, . . . .	Sonorous.
Dry or Vibrating Rhonchi,	{	Sibilant, . . . .	High pitch.
	{	Sonorous, . . . .	Low pitch.
Mucous Rhonchi, . . . .	{	Crepitant, . . . .	Very fine.
	{	Moist crackling, . .	Less fine.
	{	Mucous, . . . .	Unequal, fine and coarse.
	{	Gurgling or cavernous,	Very coarse.
Intermediate Rhonchi, . .	{	Clicking, . . . .	{ First dry, then thickly
	{	Crumpling,	{ moist (gummy).

Exo- or peri-pulmonary sounds happen in disease of the investing membrane, and these, from their site and genesis, are called *pleural friction-sounds*.

Pleural Friction-sounds, . .	{	Grazing.
	{	Rubbing.
	{	Grating.
	{	Creaking.
	{	Crumpling.

**Changes of Voice.**—The modifications of the voice which occur in disorders of the lungs and pleura are valuable signs. The natural tracheal or laryngeal voice may be heard in unnatural sites, or natural bronchial resonance may be materially altered in intensity, pitch, concentration, and apparent proximity to the listening ear, or it may be diminished or suppressed. According to Dr. Flint, the *whispering voice* may also undergo changes. These unnatural changes of the voice may be ranged under the following heads:

Intensity, . . . .	{	Exaggerated resonance.
	{	Bronchophony.
	{	Exaggerated bronchial whisper, and
	{	whispering bronchophony.
	{	Diminished or suppressed resonance.
Character and pitch,	{	Pectoriloquy, . . . . . { Cavernous.
	{	Cavernous whisper. { Amphoric.
	{	Ægophony.

**Heart's Sounds.**—When the lungs are healthy, the intensity of these sounds is directly as the distance of the point at which they are examined from their centre of production. In some diseases of the lungs the conducting power of the media being changed—increased or lessened—the intensity of heart's sounds will be increased or lessened.]

With the view of affording the means for a comparative study of the auscultatory phenomena, the following tables, relative to the natural sounds in health, and the altered or morbid sounds in disease, are compiled from the writings of Walshe, Thompson, Wood, and Bennett; and it is hoped they may furnish an outline to guide the student in appreciating the morbid states of the lungs and heart.

The arrangement in a tabular form has been preferred, because it is believed the description of the sounds are more easily studied and compared with each other by this mode of arrangement than any other.



T A B L E I.  
THORACIC SOUNDS OF RESPIRATION AND OF THE VOICE HEARD IN HEALTH.

Sounds of Respiration.	Synonym.	Character of the Sounds.	Common Site of Production.
<b>Vesicular.</b>	Respiratory murmur of inspiration and expiration.	<i>Inspiration.</i> —A soft diffused murmur of a gentle breezy character, increased in intensity with rapidity and force of respiration, and prolonged by a full inspiration. <i>Expiration.</i> —Slightly harsher and more hollow, weaker, and shorter, usually not above one-fourth the length of inspiration.	Entrance of the air and its expulsion from the air-cells and terminal portions of the bronchi—vibrations of tissue therewith connected.
<b>Puerile.</b>	Loud Vesicular murmur.	The respiratory murmur of children and women louder than that of adults, but with characters as above.	
<b>Bronchial or Tubal.</b>	Tracheal.	<i>Blowing</i> as of air passing quickly through a tube; higher in pitch than the vesicular sounds; more rapidly evolved. <i>Expiration</i> as long, or nearly so, as <i>inspiration</i> ; generally a perceptible interval between <i>inspiration</i> and <i>expiration</i> .	Sites corresponding to the bifurcation of the tracheæ, the upper part of sternum, and between the scapulæ.
Sounds of the voice through the lungs. <b>Natural Bronchophony.</b>	Vocal resonance. Pectoral Vocal resonance.	Obscure, thrilling sound of the voice, diffused, and conveying the idea of distant origin. Articulation sometimes appreciable. Resonance more marked in males than females, and in adults than children; is only markedly present over the first bone of the sternum and in the inter-scapular regions.	The voice in articulation, passing down the trachea and bronchi, is obscured, intercepted, weakened, and diffused, by passing through the spongy pulmonary vesicular tissue to reach the surface of the chest.

TABLE II.—THORACIC SOUNDS OF A MORBID TYPE, SOMETIMES CALLED RALES BY FRENCH, AND RHONCHI OR RATTLES BY ENGLISH AUTHORS, EVOLVED DURING THE ACTS OF RESPIRATION.

A.—IN THE PULMONARY SUBSTANCE.

English Name of Sound (THOMSON).	Synonym.	Character of Sound conveyed by	Relation to Inspiration and Expiration.	How and Where Produced.	Diseases with which it is most usually associated.
I. Bubbling. <i>a. Bubbling rhonchi or rattles.</i>	Mucous rhonchus or râle.	The bursting of bubbles of some size, unequal and varying in num- ber, modified by coughing and expectoration.	Coexisting with both.	Bubbling of air through liquid (mucus, blood, or pus) in bronchial tubes of the size of a crowquill, and heard in the central or middle part of the lungs.	Bronchitis after se- cretion has become es- tablished.
<i>b. Small bubbling rhonchus or rattle.</i>	Subrepitant or sub- mucous râles or rhon- chi.	The bursting of more minute bubbles, producing weaker sounds.	Coexisting with both movements, but pre- dominating during in- spiration.	Bubbling of air through more or less viscid fluid in minute bronchial tubes, as at their peripheral distri- bution.	Capillary bronchitis of both bases of lungs, tubercular bronchitis of apex, resolution of pneumonia.
<i>c. Gurgling rattles.</i>	Cavernous rhonchi or râles.	The bursting of bubbles ob- viously of large size, with a hol- low gurgling sound, or a metallic sound if the bubbles be small.	Coexisting with both acts.	Caused by the bursting of bubbles in a hollow space, inclosed by more or less dense and smooth walls— the more so, the more perfect the gurgling.	Excavations from tu- bercle or other causes, dilatations of bronchi, pus in the pleura, with a bronchial fistula.
Clicking, or Crackling.	Humid crackling, hu- mid crepitation, hu- mid crackling rhon- chus.	Successive clicks, few in num- ber, and tending to pass into the bubbling rhonchi, especially dur- ing expiration.	Coexisting with both acts, but more regular and distinct during in- spiration.	Mechanism of its production ob- scure. It probably originates in the interior of softened tubercles, which have just commenced to communi- cate with the minute bronchi.	Generally in direct connection with tuber- culous exudation which has commenced to soften.
	Dry crackling rhon- chus, dry crepitation.	A succession of three or four minute, dry, short, sharp, crack- ling sounds, permanent in many cases when once established, and tending to pass into the clicking sound. At first it may disappear for a day or two and again recur.	Coexisting exclu- sively with inspira- tion.	Mechanism of their production undetermined. Conveys the im- pression of being evolved at a dis- tance from the surface, and in the great majority of cases is found in the <i>infra-clavicular</i> and <i>supra-cla- vicular</i> regions.	Generally observed on the eve of the soft- ening process in tuber- cles.

III. Crepitation.					
a. Primary.					
	Crepitant r�le or rhonchus.	The idea of crepitation, like that produced by rubbing slowly and firmly between the finger and thumb a lock of one's hair near the ear (WILLIAMS). An immense number of sharp sounds, conveying the notion of minute size and dryness.	Coexisting exclusively with inspiration, and at first towards its close only.	Probably due to the sudden and forcible expansion of delicate tissue, altered in its physical properties by the inflammatory state, and which probably undergoes minute ruptures.	Primary idiopathic pneumonia, or the pneumonic state established round tubercles. The crepitation of early pneumonia.
b. Secondary.	<i>Rhonchus crepitans</i> <i>rednx.</i>	Crepiti of a bubbling nature, slowly evolved, few in number, and unequal or dissimilar and irregular in occurrence.	Audible in expiration as well as inspiration, but pertaining specially to the latter.	Probably due to the bubbling of air through fluid contained in the minute bronchi.	Coexisting with the resolution of pneumonia.
	Sonorous r�le.	A musical sound of a vibratory, deep, or grave tone, attended with fremitus of the walls of the thorax over a variable surface. <i>Snoring</i> , <i>humming</i> , <i>cooing</i> , and <i>bass notes</i> are its varieties.	Coexists with inspiration and expiration, but especially marked in the latter, to which it may be limited.	Arises in the larger bronchi, and suggests the idea of vibrations.	Essentially associated with bronchitis.
b. Sibilant <i>rhonchus</i> .	Sibilant r�le.	A high-pitched, whistling sound, of variable intensity and duration, and irregular recurrence. <i>Clicking</i> , <i>whistling</i> , and <i>hissing</i> varieties are described.	Coexistent with inspiration and expiration, especially marked in the former, but occasionally limited to either.	The influence of the passage of air on a local accumulation of viscid mucus.	Pulmonary emphysema and bronchitis.
B.—ASSOCIATED WITH THE MOTIONS OF THE PLEURA.					
V. Friction.	Grazing, rubbing, grating, creaking, are varieties of this sound.	The sensation of friction by a series of abrupt jerking sounds, rhythmical with respiration, few in number and superficial in seat, limited in extent, attended with fremitus, palpable to the hand, and perceptible to the patient.	Invariably heard in inspiration, or in both respiratory acts.	The rubbing of two opposed serous surfaces together, attended by inflammation.	Pleurisy, or any cause of roughness on the surface of the pleura.

T A B L E I I I.

THORACIC SOUNDS OF A MORBID TYPE EVOLVED DURING THE ACT OF ARTICULATION OF THE VOICE.

Name of the Sound.	Character of the Sound.	Physical Conditions under which it is supposed to be Produced.	Diseases with which it is commonly Associated.
I. <b>Bronchophony.</b>	Exaggerated resonance of the voice, unattended with articulation, diffuse or concentrated, and rarely producing any tactile sensation to the ear.	Increased density of the pulmonary tissue surrounding pervious bronchi, with enlarged calibre and hypertrophy of the substance of the bronchi. The more homogeneous the consolidation the better probably is the sound transmitted.	Hepaticization of lung; dilated bronchi; pleurisy, with effusion, when hepaticization coexists.
II. <b>Pectoriloquy,</b> or Pectoriloquous Bronchophony.	Complete transmission of articulated words from the walls of the chest into the ear; the resonance being generally circumscribed and limited in extent.	When solid masses of lung lie between a large bronchus and the parietes; when a moderate-sized excavation exists, with smooth and dense internal surface.	Various morbid states in which such physical conditions may exist.
III. <b>Amphoric resonance.</b>	A ringing metallic sound reverberating through a cavity, and resembling that produced by speaking into a broad-mouthed empty pitcher.	The voice reverberating in a large cavity, communicating with a bronchus by a small aperture.	Chiefly in phthisis.
IV. <b>Ægophony.</b>	A sound—vibratory, tremulous, cracked, and irregular, limited in its seat, comparable to the bleating of a goat, or to the voice of the exhibitors of Punch; synchronous with the articulation of each word, or following like a feebly whispered echo from a distant source. The sound appears to flutter tremulously about the applied end of the stethoscope.	When a stratum of fluid contained in the pleura compresses the lung, the voice in the bronchial tubes is thus rendered more distinct, by the compression of the pulmonary texture, and is thrown into vibration by the layer of fluid.	A tendency to it during the early period of pleuritic effusion, equally diffused and small in amount.

## SECTION IV.—RELATION OF THE PARTS OF THE HEART AND GREAT BLOODVESSELS TO THE WALLS OF THE THORAX.

A knowledge of the exact position of the several parts of the heart, particularly of its valves and orifices, and of their relation to fixed points on the surface of the chest, is essential to accurate diagnosis. The size of the heart, and of its several parts, may thus be relatively determined in the living subject: so also its relative position, and any amount of displacement it may have undergone, and whether or not its valves or orifices are diseased. The base of the heart being the most fixed part, is the most convenient from which to trace the outline of the heart, and to determine what parts correspond to certain fixed points upon the surface of the chest.

The heart is situated obliquely in the cavity of the thorax, from above downwards, from before backwards, and from right to left. It lies behind the middle and lower bone of the sternum, also behind the cartilages of the third, fourth, and fifth right ribs, near the sternum, and the cartilages of the third, fourth, fifth, and sixth ribs on the left side, in front of the bodies of the sixth, seventh, and eighth dorsal vertebræ. It rests immediately above the diaphragm upon its cordiform tendon, the serous layer of the pericardium only being interposed.

Owing to the obliquity of its position, the *line of the base of the heart* looks upwards and backwards towards the right shoulder. *The apex* points downwards and forwards towards the space between the cartilages of the fifth and sixth ribs on the left side, where its impulse may be felt during life. *The base of the heart* is on a line with the interval between the cartilages of the second and third ribs.

*The region of the heart's superficial dulness* is known as the *præcordial* region, the limits of which, as already defined, correspond to a vertical line through the centre of the sternum; and about the middle of the bone, nearly on a line with the cartilage of the fourth rib, the edge of the left lung separates from this middle line, and passes obliquely to the left side—thus exposing a small portion of the pericardium (Figs. 5, 6, *ante*), which is uncovered by lung. The exposed surface has a triangular shape; the apex above, the base below. The parts of the heart thus exposed beneath the pericardium are a part of the left ventricle, near its apex, and a portion of the apex of the right ventricle. This triangular *præcordial* space is on a plane below the nipple and the fourth rib. Its base is on a line with the cartilage of the sixth rib; its right boundary nearly a vertical line through the centre of the sternum; its left boundary is an oblique line through the cartilages of the fifth and sixth ribs on the left side. Within these limits the heart is in contact with the parietes of the chest, yielding a characteristic sound to percussion.



*The sac which incloses the heart* has a pyriform shape, the base below, the apex above, exactly the reverse of that of the heart. Thus, the *base of the pericardium* is on a line with the upper part of the xiphoid cartilage; *its apex* is a short distance above the origin of the large vessels, and generally on a line with the articulations of the cartilage of the second ribs with the sternum; but may extend as high as the level of the articulation of the first ribs with the sternum. The sac is wider at the centre (corresponding to the greatest transverse diameter of the heart) than it is at the base; and towards its centre it extends more towards the left side.

*The line of the base of the ventricular portion* of the heart is from three to three and a half inches below the clavicles (left and right respectively), and on a line with the junction of the cartilage of the third left rib and fourth right rib with the sternum.

*The line of the base of the left ventricle* rises as high as a line drawn across the junction of the cartilage of the *third left* rib with the sternum—i. e., about three inches below the clavicle on that side.

*The line of the base of the right ventricle* corresponds to a line across the upper margin of the junction of the cartilage of the *fourth right* rib with the sternum—i. e., about three inches and a half below the clavicle on that side.

*The impulse of the apex of the organ* is to be felt between the fifth and sixth left ribs, near where the body of these ribs joins the cartilage. The apex is a little below the fifth left rib, slightly to the left of its junction with its cartilage, and on a line with the articulation of the xiphoid cartilage with the sternum. The nipple in the male has been considered to be a useful guide. It is said to be upon the fourth rib, or over its upper or lower edge, a little more than an inch to the left of the junction of the rib with its cartilage. The edge of the left ventricle reaches the nipple on the left side.

*The length of the ventricular portion* of the heart is determined by the length of a line drawn from the middle of the sternum, between the cartilages of the third ribs, to below the fifth left rib, slightly to the left of its junction with the cartilage.

The greater part of the *right ventricle* lies behind the sternum: at its upper part it extends slightly to the right of this bone, a small portion extending under the cartilages of the fourth and fifth right ribs close to the sternum. Its apex is to the left of the sternum, a little above the apex of the heart; and a part of the right ventricle extends under the cartilages of the fourth and fifth right ribs, close to the sternum. The inferior margin of the right ventricle is nearly on a line with the junction of the xiphoid cartilage and the sternum. The anterior wall lies immediately under the sternum. It is overlapped at its upper portion by both the right and the left lung.

The *left ventricle* is covered by the left lung; and all its anterior surface is to the left of the sternum, extending from the cartilage of the third left rib to the interspace between the fifth and sixth

left ribs, near where the cartilage joins the body of these ribs. It lies between the sternum and the nipple on the left side, to which its left margin reaches.

The *right auricle* lies to the right of the sternum, entirely covered by the right lung. Its appendix lies behind the cartilage of the third right rib, its tip rests against the right side of the ascending portion of the arch of the aorta, and is on a line with the pulmonary valves.

The *left auricle* is entirely covered by the left lung. Its appendix is the only portion seen when the pericardium is laid open. It lies behind the cartilage of the third left rib, close to the sternum, resting against the left side of the commencement of the pulmonary artery.

The line of the base of the auricles is on a line with the interval between the junction of the second and third ribs with the sternum, the greater portion of it being under the sternum.

**Relative Position of the Orifices of the Heart.**—The *right auriculo-ventricular orifice* lies behind the centre of the sternum, on a line with the lower margin of the articulation of the cartilages of the fourth rib with the sternum.

The *left auriculo-ventricular orifice* is on the same level, but on a plane posterior to the right. It lies behind the cartilage of the fourth left rib, near to or behind the sternum.

The *valves of the pulmonary artery* are on a line with the space between the cartilages of the second and third ribs to the left of the sternum, and very close to this bone. In some instances they may lie a little lower down—namely, on a line with the junction of the cartilage of the third left rib with the sternum, and immediately under it.

The *aortic valves* lie behind the sternum, on a line with the junction of the cartilages of the third rib with the sternum, and towards the left edge of this bone. When the valves of the *pulmonary artery* are situated lower down, the *semilunar valves* of the aorta will be lower also, and on a line with the interval between the insertion of the cartilages of the third and fourth ribs.

A line drawn across the inferior margin of the third ribs corresponds to the *base of the valves of the pulmonary artery* and to the *free border of the aortic valves*.

The *right ventricle* ascending higher than the left, the *orifice of the pulmonary artery* is on a plane higher than that of the aorta; hence the *pulmonary orifice* is the highest up, as well as the most anterior, of all the orifices of the heart.

The *aortic orifice* lies behind the pulmonary orifice, but on a lower plane.

The *left auriculo-ventricular orifice* is immediately behind the aortic orifice, but on a lower plane.

The *right auriculo-ventricular orifice* is nearly on the same plane as the left, but more anterior, about three-quarters of an inch lower than the pulmonary orifice.

The *ascending portion of the arch of the aorta* curves to the right

of the sternum, between the cartilages of the second and third ribs. In this part of its course it is still within the pericardial sac, and (in the dead subject) lies at the depth of an inch and a half from the surface, the margin of the right lung and the pericardium being between it and the parietes of the chest.

The *transverse portion of the arch of the aorta* crosses the trachea at the centre of the first bone of the sternum on a line with the lower margin of the articulation of the cartilages of the first ribs with the sternum, and at a still greater depth from the surface.

The *arch of the aorta* approaches most closely to the parietes of the chest at the point at which the *arteria innominata* comes off—i. e., on a line with the junction of the cartilage of the second right rib with the sternum.

The *origin of the pulmonary artery* is on a line with the junction of the cartilages of the third ribs with the sternum. The tip of the left auricle rests against its left side. The pulmonary artery ascends about two inches before it divides; and at that point a portion of the margin of the vessel comes to the left of the sternum between the cartilages of the second and third ribs. The division of the artery is on a line with the upper edge of the cartilage of the second ribs where they join the sternum.

The *ascending vena cava* passes through the diaphragm by an opening, which corresponds to the upper part of the xiphoid cartilage.

In various morbid states the apex of the heart is formed by the *right ventricle* alone, or by the right and left together: in hypertrophy of the right side, for example, associated with *bronchitis* or with *emphysema*. When the right ventricle chiefly enters into the formation of the apex of the heart, the apex is then broad and rounded, rather than of the normal conical form, and is evidence of long-standing pulmonary obstruction (BELLINGHAM, BOUILLAUD, FULLER, PEACOCK, WALSHE, WILKS).

#### SECTION V.—DIMENSIONS OF THE ORIFICES OF THE HEART.

The healthy working of the heart is consistent rather with its relative size, and the relative dimensions of the orifices to each other, to the size and weight of the body generally, the condition of other organs, such as the lungs, the liver, the stomach, the spleen, and the kidneys, than with the absolute size of the heart itself.

The *tricuspid orifice* ought to be larger than the *mitral orifice*, and *half as large again* as the *aortic orifice*.

The *mitral orifice* ought to be larger than the *orifice of the pulmonary artery*, and larger than the *orifice of the aorta* by *one-fourth*.

The *orifice of the pulmonary artery* ought to be larger than the *orifice of the aorta* by about *one-eighth* (WILKS).

The *mitral orifice* tends to diminish under all forms of disease which diminish the supply of blood coming from the lungs to the

left side of the heart—*e. g.*, smallness of the chest and lungs compared with the size of the person, and imperfect respiratory acts. Such forms of contraction are independent of organic disease of the part, and are unlike induration, which implies increased development of tissue round the orifice, which leads to its contraction. Under this latter condition ulceration may ensue, when the anterior curtain is generally partially destroyed, when large soft vegetations or concrete fibrin are deposited from the blood, and are found adhering to the diseased parts.

If the *mitral orifice* is relatively small and contracted, the left auricle may become surcharged with blood. Its walls are then apt to become thick, tough, and hypertrophied, in place of being uniformly thin. Pulmonary obstruction is associated with this condition, and enlargement of the right side is a subsequent effect.

Aortic obstruction or contraction enlarges relatively all other parts of the heart.

The orifices of the right side of the heart exceed in size those of the left, and more especially the *auriculo-ventricular orifices*, compared with the arterial orifices; but in advanced life the *aortic orifice* tends to exceed the *pulmonary*.

The researches of Bizot likewise show that the size of all the orifices of the heart is relatively greater in the male than in the female, and that their size progressively increases as life advances in both sexes.

The following are the average dimensions of the orifices of the heart given by Bizot:

	Male.	Female.
Right auriculo-ventricular orifice, . . . .	54.12 lines.	48.25 lines.
Left auriculo-ventricular orifice, . . . .	45.17 "	41.8 "
Pulmonary orifice, . . . . .	82.2 "	80.7 "
Aortic orifice, . . . . .	81.15 "	28.4 "

SECTION VI.—RELATIVE WEIGHT OF THE LUNGS AND HEART.

The absolute weight of the lungs and of the heart increases as life advances, and is greater in the male than in the female at all ages (Bizot, Boyd, Peacock).

The following are the records given by Dr. Boyd in his paper already referred to:

TABLE SHOWING THE RELATIVE AVERAGES OF BODY-WEIGHT, AND THE WEIGHT OF THE LUNGS AND HEART, AS TO AGE AND HEIGHT.

Age and Sex.		Body-weight.	Body-height.	Weight of Right Lung.	Weight of Left Lung.	Weight of Heart.
Years.		Lbs. Oz.	Inches.	Ounces.	Ounces.	Ounces.
1 to 2	Male, . . . . .	14 6	28.5	4.12	8.99	1.66
	Female, . . . . .	18 2	27.7	8.58	2.95	1.47
2 to 4	Male, . . . . .	20 0	81.6	5.45	5.08	2.14
	Female, . . . . .	18 7	81.6	5.04	4.24	2.11
4 to 7	Male, . . . . .	25 8	87.5	6.27	6.01	2.77
	Female, . . . . .	24 9	87.0	5.52	5.45	2.8
7 to 14	Male, . . . . .	42 6	47.0	10.14	10.38	4.25
	Female, . . . . .	88 6	45.0	8.82	8.17	4.88
14 to 20	Male, . . . . .	68 0	60.5	20.4	19.67	7.61
	Female, . . . . .	68 14	57.7	17.52	15.08	8.46
20 to 30	Male, . . . . .	92 14	66.75	32.84	30.09	10.06
	Female, . . . . .	86 18	62.0	21.89	16.71	9.08
30 to 40	Male, . . . . .	98 8	66.5	28.47	24.29	11.86
	Female, . . . . .	87 0	62.0	18.74	17.64	9.45
40 to 50	Male, . . . . .	102 0	66.8	31.21	28.68	11.53
	Female, . . . . .	84 9	62.0	19.73	17.47	9.6
50 to 60	Male, . . . . .	102 0	66.0	30.82	26.29	11.83
	Female, . . . . .	86 0	62.0	19.48	17.08	10.44
60 to 70	Male, . . . . .	103 18	65.7	28.52	24.16	12.94
	Female, . . . . .	86 14	61.5	20.32	16.59	10.64
70 to 80	Male, . . . . .	106 18	65.7	18.77	17.11	13.14
	Female, . . . . .	80 4	61.0	16.76	14.59	10.10
Above 80	Male, . . . . .	99 0	66.7	30.46	24.30	12.1
	Female, . . . . .	79 5	60.0	18.22	15.23	10.27

The thickness of the parietes of the heart varies also with age; those of the *left* ventricle are thicker in the male than in the female at every age, and the thickness increases as age advances. The parietes of the right side increases, but in a much less ratio.

The thickest part of the parietes of the left ventricle is at the centre, next at the base, and it is thinnest at the apex (the mean thickness is under *six* lines). The thickest part of the parietes of the right ventricle is at the base, where it is above *two* lines. The thickest part of the *septum ventriculorum* is at its centre.



## SECTION VII.—MODE OF EXAMINATION OF THE HEART.

In order to determine the nature, the situation, and extent of morbid changes in the heart, it is necessary to be able to recognize readily any alteration in the heart's impulse, either as regards its strength or the situation in which it is felt—it is necessary to be able to detect any difference in the extent and degree of the heart's superficial dulness, or any change in the character of its sounds different from those which are normal.

Inspection, palpation, percussion, and auscultation are all therefore capable of affording valuable assistance in arriving at a diagnosis; and the following statements are condensed from the admirable writings of Drs. Bellingham, Fuller, Gairdner, Stokes, and Walshe.

The inspection of the external surface of the thorax, and the application of the hand to the *præcordial* region should never be omitted. Positive information is obtained by these two methods of examination, which mutually assist one another.

The exact point at which the apex of the heart comes in contact with the parietes of the chest may be determined simply by inspection. The *strength* or *feebleness* of the impulse of the heart is to be determined by the application of the hand.

By inspection it is ascertained whether the two sides of the thorax are symmetrical; and (in connection with cardiac diagnosis) whether there is any bulging in the *præcordial* region, or any unusual pulsation at any part of its parietes in the large arteries which come off from the arch of the aorta, as well as in the jugular veins or epigastric region.

By the application of the hand—palpation—the force or impulse of pulsation is determined, the frequency or slowness of the heart's action is judged of, and the regularity or irregularity of its movements. We may likewise appreciate by this means any *tremors* or *frictions* which accompany its action in the pericardium. To determine the impulse of the heart, the hand must be placed directly upon the surface of the chest; but *mediate* palpation may be used by placing one end of the stethoscope over the part where the impulse is, when the extent to which the instrument is elevated, and the force with which this is accomplished, will give an accurate idea of the strength of the heart's impulse, especially in hypertrophy, or in hypertrophy with dilatation of the ventricles.

In healthy persons with well-formed chests the impulse of the heart is so slight as not to be perceptible to the individual himself; and it is felt only at one spot—namely, between the cartilages of the fifth and sixth ribs on the left side—*i. e.*, from one to two inches below the nipple, and to its sternal side. When the parietes of the chest are much loaded with fat, the impulse is scarcely perceptible to the hand; while in thin persons it is evident to the eye.

The impulse is somewhat stronger in the erect than in the recumbent posture. A forced inspiration diminishes it, and causes it to

be felt lower down than usual; while a deep inspiration elevates the ribs, without raising the heart in the same degree.

In a forced expiration the impulse is more perceptible, and is felt higher up. In examining the heart, it is therefore necessary to make the patient vary his position, and to examine the heart both during inspiration and expiration. Calmness and tranquillity on the part of the patient must be obtained, because mental excitement, as well as exercise or exertion, increases the impulse of the heart. A diminished impulse, circumscribed or feeble, is due to feebleness of the action of the heart from disease or alteration of its muscular tissue—as in softening or fatty degeneration of its tissue, or general debility of the system; or owing to disease in the lungs or pericardium—as from effusion into the sac—when the apex of the organ may be prevented from coming in contact with the parietes of the chest. The impulse is also diminished in cases of attenuation of the walls of the ventricles, with dilatation of their cavity. Emphysematous lungs may likewise overlap the heart, and prevent its impulse from being felt. Increased impulse of the heart is generally due to some morbid state of the heart itself. It is stronger than natural in hypertrophy of the walls of the left ventricle, and is greatest in hypertrophy with dilatation of the ventricles. In such cases the impulse is slow, gradual, heaving, double, and occasionally so violent as to shake the bed on which the patient rests. This slow, progressive, heaving impulse, is produced by no other cause than hypertrophy with dilatation of the ventricles of the heart; and in such cases the extent of surface over which the impulse is felt is much increased, and the whole side of the chest is sometimes elevated by the action of the organ. The double impulse which can be felt is due to the fact that the *diastole* as well as the *systole* of the ventricles is accompanied by an appreciable impulse. Adhesions of the pericardium to the pleuræ of opposite sides may so bind down an enormously large heart that its impulse will not be felt.

*The situation of the impulse of the heart* may be altered by displacement of the heart itself, as in cases of empyema, towards the left side when the right pleural cavity is distended with fluid, and to the right side when the left pleural cavity is distended with fluid. The impulse may then be felt on the right of the sternum. In cases of ascites the heart may be pushed upwards, and its impulse felt on a plane higher than natural; so also in cases of ovarian and other abdominal tumors, hysterical tympanitis, or in advanced stages of pregnancy. In emphysema of both lungs the heart is displaced downwards, so that the impulse is then felt sometimes as low down as the epigastric region. When fluid is effused into the pericardium, the site of the impulse is somewhat elevated; and as the amount of fluid increases, the impulse becomes weaker, unequal, undulatory, or irregular; and when the effusion is very considerable the impulse will be altogether absent.

In hypertrophy of the left ventricle, with dilatation of its cavity, the impulse is felt lower down than natural, more to the left side, and occasionally on a line vertically from the axilla.

In hypertrophy with dilatation of the right ventricle, the impulse

is felt lower down, and more to the right side than natural, and not unfrequently on a line with the xiphoid cartilage.

In the former case (hypertrophy with dilatation of the *left* ventricle), the impulse is progressive, heaving, and strong, elevating the hand or stethoscope of the observer, and felt over a very much larger surface than natural; in the latter case (hypertrophy with dilatation of the *right* ventricle), the impulse is felt over a more circumscribed space, and is neither heaving, prolonged, nor very strong. But the downward displacement of the heart is not due *only* to the hypertrophy. Three causes conspire to produce it: one is the hypertrophy of the organ; another is the dyspnœal inflation of the lungs; and another is the flattening of the diaphragm, which always exists where there is an abiding source of dyspnœa. This descent and flattening of the diaphragm is due to the instinctive efforts made in all forms of dyspnœa to obtain more air. The large heart is thus pushed downwards by the dyspnœal lung inflation, and pulled downwards by the diaphragm (Hyde Salter, *Brit. Medical Journal*, February 8th, 1862).

*Turgescence and pulsations in the jugular veins* are to be appreciated by inspection and palpation. They are signs which accompany advanced stages of some cardiac diseases. Turgescence is the most common, and occurs in cases in which an impediment exists to the free passage of the blood through the right side of the heart; and in such cases, when the tricuspid valve imperfectly closes the right auriculo-ventricular orifice, and so permits regurgitation into the auricle at each systole of the right ventricle, we have pulsation of the jugular veins as well as turgescence. Pulsation is always most evident immediately above the clavicles, and may extend half-way up the neck.

When the *mitral valve* permits regurgitation, a "purring tremor" may be felt when the hand is placed over the region of the *mitral valve*; and a similar tremor may be felt over a dilated aorta when the valves are patent, or when a communication exists between a vein and an artery. The jarring pulse in the radial artery, in cases of aortic valve patency, is an instance of the "purring tremor" felt in an artery of small calibre.

**Percussion.**—The whole of that portion of the anterior wall of the chest behind which the heart is situated is sometimes called the *præcordial region*; but *anatomically* it is a region more limited, corresponding to that part of the pericardium of the heart uncovered by lungs (Fig. 10, *ante*, p. 535).

Both regions, however, are definable by the limits of a dull sound on percussion and more slight percussion.

The portion of heart uncovered by lung seldom exceeds *two inches* in any direction. It has a triangular shape, the base below, the apex above. It consists of a portion of the apex of the right ventricle, and of part of the left ventricle near its apex, and is on a plane below the nipple and the *fourth* ribs. Its base is on a line with the cartilage of the sixth rib. Its apex is at the point where the margins of the opposite lung begin to separate from one another—i. e., immediately below the *fourth* rib. The triangular

boundaries of this anatomically true *præcordial space* are constituted—(1.) On the right side of the thin edge of the right lung, by nearly a vertical line through the centre of the sternum; (2.) On the left side of the thin margin of the left lung, by a more or less oblique line through the cartilages of the *fifth* and *sixth* left ribs; (3.) Below, it is bounded by the diaphragm.

Over this region, to slight percussion, a sound less dull than that yielded by the liver is elicited, and on stronger percussion a difference of sound can be detected where the thin margin of the lungs covers the heart. The mode of percussing this region, so as to mark the lung part and the heart part, is to lay one finger over the decidedly dull part, and another over the slightly resonant edge of the lung, when, by striking the two fingers alternately, the arched line along which the organ lies in contact with the walls of the chest may be traced with accuracy, unless fat obscures the resonance (HOPE).

Another means of estimating the size of the heart is by *auscultatory percussion*. This requires two competent persons to determine the result, and is managed as follows: "A solid cedar cylinder, six inches in length and one inch in diameter, cut in the direction of the fibres, and with an ear-piece attached, is applied to the centre of the *præcordial* region, while the ear is applied to the other end: percussion is then made by another person from the point near where the cylinder is applied towards the limits of the heart in every direction. So long as percussion is made over the body of the heart, a distinct sharp shock is felt directly in the ear; but as soon as the limits of the heart are passed, this sharp shock immediately ceases, even in passing from one solid organ to another in contact with it, as from the heart to the liver" (Drs. CAMMANN and CLARK). Practice will enable a discrimination to be made between the characteristic sound of the heart and the diffused shock produced by striking the ribs.

The mean diameter of the healthy adult heart (not the anatomical *præcordial* region only) has been found to be as follows:

			Inches.	Lines.
Vertical diameter,	{	Male, . . .	4	0
		Female, . . .	3	7
Transverse diameter,	{	Male, . . .	4	4
		Female, . . .	4	1
Right oblique diameter,	{	Male, . . .	4	10
		Female, . . .	4	10
Left oblique diameter,	{	Male, . . .	3	10
		Female, . . .	3	7

Generally the region of this *deep-seated dulness* extends transversely from the left nipple to a little to the right of the sternum, and vertically from the third to the sixth ribs. The true anatomical *præcordial space* may be diminished if the lungs are largely developed, and may disappear if the anterior margins of the lungs are emphysematous, so that their edges meet in front of the organ.

The region of the heart's superficial dulness is increased whenever the heart is enlarged, or whenever fluid to any amount is

effused into the sac of the pericardium. If the walls of the ventricles are hypertrophied, or if their cavities are dilated, the dullness will extend over a wider surface, and its extent is in some degree a measure of the increased size which the organ has attained. The enlarged heart pushes aside the lungs, and a larger portion of it comes in contact with the parietes of the chest. In hypertrophy with dilatation the heart attains the largest size that it is capable of, and the præcordial region may yield a dull sound over a square surface of from two to six inches. When hypertrophy predominates over dilatation, the space which yields a dull sound is wider from above downwards. When dilatation predominates over hypertrophy, the region in which a dull sound is yielded transversely is wider. When there is fluid in the pericardium, a larger surface than natural in the *præcordial* region yields a dull sound; the degree of dullness is more pronounced, and the sensation of resistance considerably greater than in the former case.

The situation of the dullness, its amount and degree, are the guides as to its probable cause. When it is caused by enlargement of the heart, the site of the dullness is lower down and more to the left side than when it depends upon liquid effused into the pericardium. If a large amount of fluid is contained in this sac, a dull sound may be elicited by percussion as high as the articulation of the second rib with the sternum, and even in some cases as high as the first rib. The degree of dullness over fluid is also much more marked than over the heart itself, and the resistance to the finger is greater. Solidification of lung in the immediate vicinity of the heart will cause an apparent extension of the cardiac dullness.

**Auscultation and the Sounds Associated with the Action of the Heart.**—Auscultation of the heart, like percussion, may be either mediate or immediate; but generally there are many objections to immediate auscultation. In the case of females it is indelicate; in dirty persons it is disagreeable; while in contagious diseases it is not without risk; besides, there are some situations in which either the ear cannot be applied, or in which the stethoscope is much more convenient. In cases of valvular disease of the heart the stethoscope, and especially the forms of the double stethoscope already described, are absolutely necessary in many cases.

The phenomena of the heart's action, as observed by the eye on the heart of a living animal exposed to view, or as appreciated by the ear and hand applied over the cardiac region of man, are of the most definite nature; and in determining the alterations of sound produced by disease, it is necessary to bear in mind the occurrence and sequence of the following incidents.

The two auricles of the heart contract at the same instant of time, and the contractions of the right and left ventricles are also simultaneous. The contraction of the ventricles follows immediately that of the auricles. The relaxation of the fibres of each part of the heart follows immediately their contraction, and a short but distinctly appreciable period of repose intervenes between the relaxation of the ventricles and the secondary contraction of the auricles. Each complete revolution of the heart is thus accompa-



nied by two successive sounds, separated from each other by intervals of silence. These two sounds *are unlike*; and the two periods of silence *differ in duration*. Within the limits of health these sounds have a variable duration, and limit of surface over which they are heard, as well as of intensity, and of a certain *rhythm*. The *rhythm* of the heart is maintained when its two sounds succeed each other, and are followed by an interval of repose, which varies in length according to the duration of the previous systole, and according to the rapidity with which the sounds succeed each other.

The first sound is coincident with the systole of the ventricles, the impulse of the apex against the side of the chest, and the pulse of the large arteries. It is sometimes also called the *systolic* or *inferior* sound of the heart. It should be listened to over the apex of the heart.

The second sound is synchronous with the diastole of the ventricles, the recedence of the heart from the side, and the pulseless state of the large arteries. It is sometimes also called the *diastolic* or *superior* sound. It should be listened to at *mid sternum*, near its left edge, and on the level of the second interspace.

A very short silence (which only becomes obvious when the pulse does not exceed sixty in a minute) succeeds the first sound; but a distinctly appreciable period of silence and repose, of some duration, succeeds the second sound.

The *first sound* is of a prolonged and dull character compared with the *second sound*, which is more quick, short, and clear, and bears a close resemblance to that produced by *lightly tapping, near the ear, the knuckle of a bent finger with the soft extremity of a finger of the other hand* (HOPE).

The Line of Transmission of these sounds respectively it is of the greatest practical importance to observe; because, being so constant in health, any variation indicates some modifying cause.

The *first sound* passes slantingly upwards to the left acromial angle, growing weaker and weaker on the way. Its intensity diminishes much more on the way to, and at, the right acromial angle. The propagation backwards of the first sound is thus clearest and fullest to the left,—so that, while audible at the left back, it may be inaudible at the right.

The *second sound* has the region of the base for its centre; and in nine people out of ten is heard more clearly at mid sternum, *on the level of the second interspace*, than at any point of the præcordial region (WALSHE). It radiates towards the right and left acromial angles, and with greater clearness to the left than the right, while posteriorly it is heard at the surface with less clearness and distinctness on the right side than on the left.

Next to the lines of propagation of the sounds being determined as above indicated, it is necessary to analyze the sounds, and compare them,—(1.) At both sides of the apex and region of the base; (2.) At base and apex on the same sides of the organ; (3.) At base and apex of opposite sides. In any one of these regions, compared with the other, the sounds in health are found to vary so materially

in positive and relative properties that any single description of them cannot be given.

The natural sounds of the heart thus indicated may be of abnormal character, as regards *intensity, pitch, duration, quality, rhythm, reduplication, and apparent distance*, at each of the following points: namely, at the third interspace, and along the third rib for the distance of three inches from the left edge of the sternum, *for all sounds*; at the point where the apex beats, *for mitral sounds*; at the left of the sternum, and over the ensiform cartilage (if the apex be not in this position), *for tricuspid sounds*; at third left costal cartilage and adjoining part of the sternum, *for both aortic and pulmonary sounds*; at second right and second left costal cartilages, *for the sounds of aortic and pulmonary orifices respectively* (*What to Observe*, p. 43).

The term "*sound*," in reference to cardiac diagnosis, is understood to refer to the natural sounds of the heart, either normal or modified in character as detailed above.

It is important to observe the relationship of the sounds of the heart to the pulse. The first sound anticipates, by a very short but appreciable interval, the pulse at the wrist. An interval consistent with health is undetermined; but it may be stated generally, that if the diastole of the most distant arteries, such as the posterior tibial, behind the inner ankle, or the arteries on the dorsum of the foot, is so much retarded as to become synchronous with the *second sound*, the state indicates disease. It is a frequent attendant on insufficiency of the aortic valves.

When the sounds of the heart, after a certain number of perfectly regular beats, are suspended during the time usually taken to perform an entire revolution of the cardiac functions, the sounds of the heart are said to *intermit*. A sudden pause or silence then occurs, and such an intermission sometimes happens more or less regularly, or after a fixed number of regular beats. This constitutes *intermittence* or *intermission* of the heart's action. It may occur in individuals otherwise in perfect health; but it is common in diseased states of the valves or orifices of the heart, where some impediment exists to the direct passage of the blood, or where regurgitation is permitted.

**Morbid Sounds of the Heart, or Murmurs.**—The term "*murmur*" is applied to a sound superadded to the normal sound of the heart, and which may occur with one or more of the natural sounds. The term was first employed by the late Sir John Forbes, and has been very generally adopted since. These may be so obscured, or even obliterated, that the *murmur* or *morbid sound* is alone heard. According to their supposed seat of production these *murmurs* are,—(1.) *Endocardiac*, sometimes called *valvular*; (2.) *Pericardiac*, also called *exocardiac*.

All endocardiac or valvular murmurs yield a "*blowing*," "*rough*," "*rasping*," "*sawing*," "*booming*," or "*bellows*" sound, as in the whispered expressions of the words "*who*" or "*awe*," the double letter "*ss*," or the single letter "*r*;" and there are certain spots where they may be heard in their greatest *maximum force*; namely,

—(1.) A few lines above the left apex; (2.) Just above the ensiform cartilage at mid sternum; (3.) On the level of the third interspace; and (4.) At the junction of the third left cartilage with the sternum.

The physical causes which may explain the *mechanism* of these murmurs are due either—(1.) To pure constrictions of natural orifices; (2.) To pure widenings of natural orifices; (3.) To pure roughness of surfaces; or (4.) To the association of the latter condition with either of the two former.

When murmurs are due to such single or combined mechanism, they are said to be *organic murmurs*, to distinguish them from *inorganic* murmurs due to certain morbid causes not yet well understood.

These *inorganic* murmurs are connected—(1.) With certain states of the blood, as in *spanæmia*; or (2.) With dynamic or functional action of the heart itself.

It is of the greatest importance to determine, therefore, the nature of a *murmur*, especially as to whether it is really *organic* or *functional*.

In this investigation the essential points to be inquired into, to guide the diagnosis, are as follows: (1.) Observe the relationship of the murmur to the systole or diastole; that is, the *rhythm* of a murmur, or the position, in point of time, which it holds during the different physiological acts which constitute a complete cardiac pulsation—namely, the *contraction*, the *dilatation*, and the *period of rest* of each of the cavities; (2.) The spot of its maximum intensity on the surface of the chest; (3.) The direction in which the murmur is transmitted; (4.) Its quality and pitch; (5.) State of the natural sounds of the heart which may remain; (6.) Presence or absence of any audible phenomena in the arteries or veins, or both; and (lastly) The duration and clinical progress of the case.

Each orifice of the heart may be the seat of two murmurs, *constrictive* and *regurgitant*, *with* or *against* the current; and thus *eight murmurs* are the total number the occurrence of which is possible. The essential characters of these murmurs are condensed as follows from Dr. Walshe's *Practical Treatise on Diseases of the Lungs and Heart*; and from Professor W. T. Gairdner's paper, entitled "A Short Account of Cardiac Murmurs," *Edin. Med. Journal*, Nov., 1861.

In determining the attributes of a cardiac murmur, the first step in the inquiry is to determine which is the *second* sound of the heart, and which is the *first*. In the *rhythmical* succession of the heart's actions the phenomena which we can appreciate externally are a little later than the commencement of the heart's action. *Before there is either sound or impulse the contraction has already taken place*; and whatever the pathological origin or seat of the murmur may be, those which immediately succeed the first sound and the impulse correspond to the period of ventricular contraction; and those which succeed the second sound correspond to the period of ventricular dilatation.

Dr. Gairdner gives the following classification and account of them:

1. An *auricular systolic* murmur is one which *precedes and runs up to the FIRST SOUND* of the heart, which is in all probability produced in one or other of the auriculo-ventricular orifices, inasmuch as it coincides with the forcible emptying of the auricles into the ventricles through these orifices. Its reasonable interpretation, therefore, is *obstruction to the current of the blood ENTERING a ventricle*. If the left auriculo-ventricular orifice is affected, the murmur will be found to have the character of a *mitral murmur*, and to have its area at A (Fig. 16). If, on the contrary, the *tricuspid* orifice be obstructed, the murmur will occupy the triangular area C (Fig. 16).

2. A ventricular systolic murmur *succeeds and runs off from the FIRST SOUND*; and it may be produced either in the *auriculo-ventricular* or in the arterial orifices. In either case it coincides with the *emptying* of the ventricles; and, therefore, if *auriculo-ventricular* in origin, it is a *murmur of regurgitation*; if, on the other hand, it is of *arterial origin*, it is a *murmur of obstruction*.

A ventricular systolic murmur may thus have four distinct solutions among organic valvular diseases. If the area be *mitral*, it is a *murmur of mitral regurgitation*. If the area be *aortic*, it is a *murmur of aortic obstruction*. If the area be *tricuspid*, it is a *murmur of tricuspid regurgitation*. If the area is that of the origin of the *pulmonary artery*, it is a *murmur which indicates pulmonic obstruction*.

3. A *ventricular diastolic murmur* succeeds and runs off from the second sound, and may be produced either in the *auriculo-ventricular*, or in the *arterial* orifices. In either case it coincides with the *filling* of the ventricles; and, therefore, if *auriculo-ventricular* in origin, it is a *murmur of obstruction*; and if *arterial*, it is a *murmur of regurgitation*.

A *ventricular diastolic murmur* may thus have four distinct solutions among organic valvular diseases. If its area is *mitral*, it is a *murmur of mitral obstruction*; if its area is *aortic*, it is a *murmur of aortic regurgitation*; if its area is *tricuspid*, it is a *murmur of tricuspid obstruction*; if its area is of the origin of the *pulmonary artery*, the murmur denotes *regurgitation from the pulmonary artery*.

The most frequent combinations of these murmurs are those which denote—

1. Combined aortic obstruction with regurgitation, indicated by ventricular systolic and ventricular diastolic murmurs.

2. Mitral obstruction and regurgitation, indicated by *auricular systolic* murmurs, sometimes by ventricular diastolic and ventricular systolic murmurs.

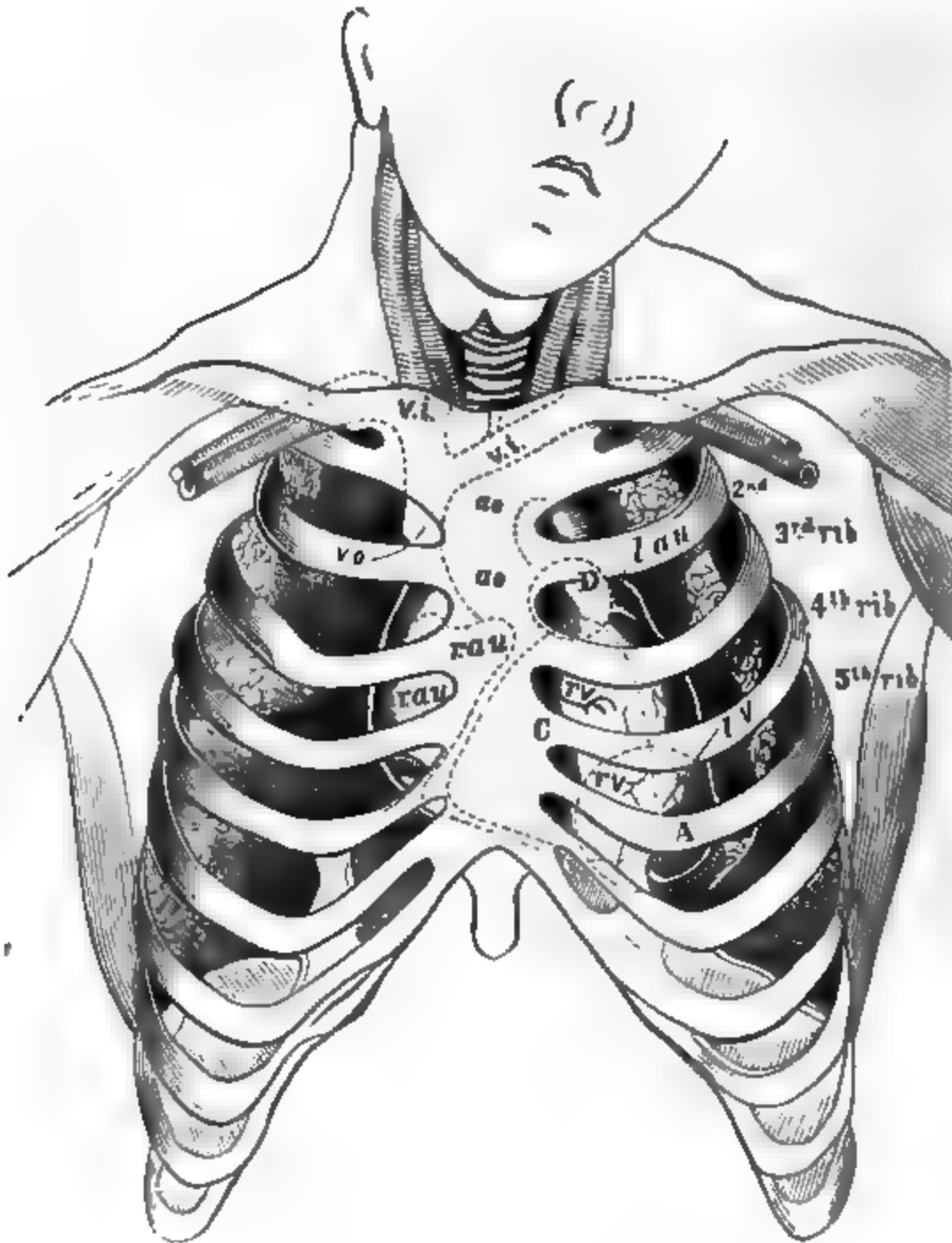
3. Various combinations of the two preceding forms, the aortic and mitral valves being both diseased.

4. Mitral obstruction with dilated right ventricle, and consequently tricuspid regurgitation, indicated by *auricular systolic murmur*, heard over area A, Fig. 16; and ventricular systolic murmur, heard over area C.

The rarest of all murmurs are those which denote *obstruction of pulmonary artery*, and those of *tricuspid obstruction*. These murmurs are still more rarely observed singly, being usually in combination with diseases causing murmur on the left side of the heart.

In a very able communication to the Hunterian Medical Society of Edinburgh, Dr. J. Warburton Begbie has recently called atten-

FIG. 16.\*



tion to "the diagnostic value of an accentuated cardiac second

\* The Heart: its several parts and great vessels in relation to the front of the thorax. The lungs are collapsed to their normal amount, as after death, exposing the heart. The outlines of the several parts of the heart are indicated by very fine dotted lines. The area of propagation of valvular murmurs is marked out by more visible dotted lines. A, the circle of mitral murmur, corresponds to the left apex.

The broad and somewhat diffused area, roughly triangular, is the region of tricuspid murmurs, and corresponds generally with the right ventricle where it is least covered by lung. The letter C is in its centre.

The circumscribed circular area, D, over which the pulmonic arterial murmurs are commonly heard loudest. In many cases it is an inch, or even more, lower down, corresponding to the *conus arteriosus* of the right ventricle, where it touches the walls of the thorax.

The internal organs and parts of organs are indicated by letters, as follows: *r. au*, right auricle, traced in fine dotting; *ao*, arch of aorta, seen in the first intercostal space, and traced in fine dotting on the sternum; *vi*, the two innominate veins; *rv*, right ventricle; *lv*, left ventricle.



sound" (*Edin. Med. Journal*, June, 1863). This accentuated second sound—equivalent to an intensified or greatly pronounced sound—is heard in instances of aortic aneurism and aortic dilatation, associated with atheromatous degeneration, as well as in some cases of hypertrophy and dilatation of the left ventricle. In a case of aortic aneurism, the second sound of the heart has been observed so intensified or accentuated over the base of the heart as at once to be recognized even by *tyros* in the art of auscultation. When this sound occurs, which is of a *booming* or *ringing* character, it is to be presumed that the aortic valves are competent. If they were insufficient, a diastolic murmur would be the result, as the prominent physical sign, and apt to cause the most skilful physicians to overlook the existence of aneurism.

Modern diagnosis localizes murmurs chiefly from the observation of the areas of transmitted sounds already indicated. There are four distinctive areas to which murmurs arising at these orifices may be propagated.

The accompanying wood-cut (modified from those of Professors W. T. Gairdner, of Glasgow, and Luschka, of Tübingen), Fig. 16, p. 576), indicates the areas.

1. **Murmur connected with the Mitral Valve, Orifice, or neighboring portion of the Left Ventricle**, may be the result of inefficiency of the valve, by changes in its structure, or from roughness of its edges, as by vegetations, shortening of the *chordæ tendineæ*, or fibrinous coagula amongst them. It may also result from simple roughness or deposit on the under surface of the valve without positive insufficiency. It is a *ventricular systolic murmur*, of maximum force, heard at and immediately above, or to the outside of the *left apex*, and which may completely or partially cover the first sound of the heart at the left apex, but which may also preserve its natural characters towards the base.

This *systolic murmur* is faintly or wholly inaudible at the right apex, the mid sternal base, the pulmonary and aortic cartilages. It is more or less clearly audible about and within the inferior angle of the left scapula, and beside the dorsal vertebræ from the sixth to the ninth. This *murmur* is rarely of high pitch; and once established it is permanent.

To find the area of this murmur it is requisite to determine the exact seat of the apex beat, the patient lying a little to the left side, or even on the face. If there is no distinct apex beat, find the most remote point downwards and leftwards at which the impulse of the heart is discernible; test this point by percussion, to observe if it corresponds with the margin of the cardiac dulness; test it also by auscultation, to hear if the *first sound* is conveyed thither with special distinctness. If a murmur concurs in position with the seat of these different phenomena, and if its seat of diffusion is *round this point nearly in a circle* (Fig. 16), it is *probably of mitral origin*.

2. **Murmur associated with the Tricuspid Valve** may be due to regurgitation, or to the sharp collision of blood among thickened and roughened *chordæ tendineæ*. It too is a *ventricular systolic murmur*, heard of maximum force *immediately above or at the ensiform carti-*

*lage*; inaudible, or nearly so, at the left apex, and very faintly, if at all perceptible, in the left vertebral groove opposite the lower angle of the scapula. It originates in the right ventricle; and when due to regurgitation, there is distension and pulsation of the auricle, vena cava, innominate and jugular veins, the distension of the latter being visible. It is generally a soft *murmur*, of low pitch, and rarely masks the systolic sound completely. It is a rare *murmur*, and often escapes detection from two causes—namely, a powerful mitral *murmur*, with which it is usually associated, or a deep-seated venous *hum*.

Professor W. T. Gairdner does not consider that *tricuspid murmurs* are rare, at least those of regurgitation. The area of tricuspid valve murmurs is over the right ventricle, where it is uncovered by lung—*i. e.*, at the lower part of the sternum, and over the whole space between this and the seat of the mitral murmur. It is usually but little audible above the level of the *third* rib, and is thus distinguished both from the pulmonic, and still more from the aortic murmur. Its area is indicated by the triangular space in Fig. 16; but in cases of considerable hypertrophy and dilatation of the right side of the heart, especially in connection with emphysema (when the ventricle pulsates in the epigastrium), the murmur is heard loudest towards the xiphoid cartilage, and along the margin of the seventh left costal cartilage.

3. **Murmur connected with the Aortic Valve** habitually signifies a rough constriction of that orifice, and in rare cases has been traced to fibrinous coagula impeding the egress of the blood. It likewise is a *ventricular systolic murmur*, heard of maximum force at *mid sternum, opposite the third interspace, or upper part of the fourth rib*. It abruptly loses force between this point and the left apex, where it may be almost inaudible. Faintly perceptible at the second left cartilage, it is clearly audible at the second right cartilage, the notch of the sternum, and the left vertebral groove, opposite the second, third, and fourth dorsal vertebræ, thence rapidly losing strength downwards. It originates at the aortic orifice, and disappears about the sixth dorsal vertebra. It is propagated into the arteries of the neck. It is a high-pitched, harsh, loud, and prolonged *murmur*. The concurrence of ventricular hypertrophy increases its intensity and prolongs its duration the more contracted the orifice is. The area of this murmur corresponds generally to the regions of the sternum, and is often absolutely loudest close to the xiphoid cartilage.

4. **Murmur connected with the Orifice of the Pulmonary Artery** may indicate obstruction or simple roughness in its valves, or pressure on the vessel by adventitious masses in the pericardium. It is a *ventricular systolic murmur*, heard of maximum force at the *sternal edge of the third left cartilage*, or a little lower down, and imperceptible in the back. It is rarely met with.

5. **The Murmur indicative of Obstructive Narrowing of the Mitral Valve** is a *ventricular diastolic murmur*, heard in maximum force *immediately above and about the left apex*.

6. **The Murmur which indicates the probability of Tricuspid Narrowing or Obstruction** is also a *ventricular diastolic murmur*, and is heard in maximum force at the *ensiform cartilage*.

7. **The Murmur which indicates Regurgitation at the Aortic Orifice** is likewise *ventricular diastolic*, and is heard of maximum force at mid sternum, opposite the third interspace or fourth cartilage; and it is often carried down loudly to the left apex. It is usually of an inspired, blowing character, sometimes almost hissing, rarely rough, and completely fills up the interval of repose and silence which ought to follow the second sound. It differs from *constrictive* aortic murmur in being heard with almost as much intensity *about the ensiform cartilage as opposite the third interspace*. When it covers completely the second sound of the heart at the point of its maximum intensity, the valves may be presumed to be utterly incompetent.

8. **The Diastolic Murmur connected with insufficient Pulmonary Valves** is so rare that it is only mentioned here to complete the notice of cardiac murmurs which may be heard.

The relative frequency of intra-cardiac organic murmurs Dr. Walshe states to be in the following order, commencing with the most common,—namely, *mitral regurgitant*; *aortic constrictive*; *aortic regurgitant*; *mitral constrictive*; *tricuspid regurgitant*; *pulmonary constrictive*; *pulmonary regurgitant*; *tricuspid constrictive*. These may, however, be variously associated together; and when they coexist they are to be distinguished by the *rhythm*, the *pitch*, and the *character* of the aspiration.

The point at which a murmur is produced being in the majority of cases one of the four valvular orifices, all doubtful murmurs should be tested in the first instance on the supposition that they are valvular. With this view the most important practical points to be determined are,—(1.) The actual size and position of the heart, and the relation of its several parts to the thoracic walls (as described in the previous sections); (2.) The anatomical *præcordial* space must especially be accurately defined; (3.) The exact point of the apex beat is to be determined; (4.) The character of the impulse both of the right and left ventricle should be carefully studied; (5.) Determine by careful stethoscopic observation the exact seat and the limits of the diffusion of the murmur actually under observation.

**The Pericardial Murmurs** consist of *friction* or rubbing sounds, analogous to those already described in the pleuræ (Table, p. 559), and result from the movements of two opposed surfaces on each other, having been rendered dry or rough by change of tissue or exudation. Pericardial murmurs are almost limited to cases of inflammation of the pericardium. These friction-murmurs are generally double, and are sometimes louder during the diastole than the systole of the ventricles. They appear to be superficial or near, and are seldom audible beyond the limits of the *præcordial* region. They never replace the ordinary sounds of the heart, and are entirely independent of them. Their duration is usually short, frequently ceasing entirely after having been heard for a few days,

and not unfrequently changing their character and seat within the period that they are audible. A peculiar vibratory thrill, sensible to the hand laid upon the parietes, frequently accompanies them.

#### SECTION VIII.—SIGNIFICANCE OF THE PULSE IN CARDIAC DISEASE.

Certain forms of cardiac disease are capable of impressing peculiar and well-marked characters upon the pulse. The pulse ought to correspond with the ventricular systole, and with the first sound of the heart; and when the heart, the arterial system, and the blood are each in a normal condition, the force, the strength, the frequency, and the fulness of the radial pulse may be taken as a measure of the strength or feebleness of the systole of the left ventricle, of the rapidity with which the movements of the heart are performed, and of the amount of blood transmitted at each systole of the left ventricle. The beat of the pulse in the radial artery ought to be a little—a very little—later than the ventricular systole. The interval is almost imperceptible, unless the pulse is unusually slow. In the dorsum of the foot the interval is more easily appreciated. It is advisable to place one hand upon the præcordial region, or to auscultate the region of the heart, while the finger is on the radial pulse, to determine these points in all cases.

*In softening of the heart* the pulse is sometimes much less frequent than the cardiac systole, because the impulse fails to be transmitted (FULLER, J. C. B. WILLIAMS, GAIRDNER).

*In hypertrophy of the left ventricle*, when the parietes are increased in thickness, the systole is strong in proportion, the blood is propelled into the aorta with increased force, and the radial pulse is strong and hard. Its velocity may not be increased, but the systole takes a longer time to be completed, and the pulse will “dwell longer under the finger.” When dilatation is combined with hypertrophy, so long as the circulation continues free, the pulse will be full, or of larger volume, because the amount of blood propelled at each systole will be larger; but when dilatation is combined with attenuation, or if dilatation simply prevail, the radial pulse will have nearly opposite characters to those stated. It will be soft and weak.

*The pulse in aortic regurgitation* acquires a peculiar character. It is jerking and receding, though regular; while the pulsations of the arteries of the upper extremities and of the neck are visible, as if “leaping.” It has been named a “locomotive” pulse by Bellingham and Todd—*i. e.*, the arterial tubes are SEEN to move by elongation—“leaping forth at each beat of the heart.” This is sometimes termed the “pulse of unfilled arteries.” In well-marked examples it appears as if the blood was divided into separate little balls, which pass in rapid succession under the finger. The sensation is better *seen* and felt in a large artery, such as the brachial, and two or more fingers should be laid on the line of artery. The arteries, when tortuous especially, appear like worms under the skin, wriggling into tortuous lines at each pulse (WILLIAMS). This kind of pulse is also sometimes observed in *aneurism of the ascending or*

*transverse portion of the arch of the aorta*, as well as in cases of disease of the aorta itself, when it has become rigid, elastic, and dilated.

*Intermission of the pulse* indicates the slightest degree of derangement of the heart's action. It is not uncommon in persons advanced in life, in gouty subjects, and in derangement of the digestive organs with flatulence. It is also met with in cases of disease of the valves, or of the muscular tissue of the heart.

*An unequal pulse* is one in which some of the pulsations are strong, and others weak.

*An irregular pulse* is one in which a few rapid beats are succeeded by one or more slower beats, and when the interval between them is different.

*An unequal and irregular pulse* are much more unfavorable signs than a simply *intermittent pulse*. Both are met with in the same cases—in certain diseased states of the valves at the left side of the heart, or in morbid conditions of the muscular tissue.

*In contraction of the left auriculo-ventricular orifice*, the pulse, in addition to being weak and intermittent, will become small, irregular, and unequal, although the heart's action continues to be strong. "The heart may often beat so violently as to shake the patient in his bed, while the pulse is small, weak, and irregular. It appears as if there were two *pulses*; one is slow and deliberate for two or three beats, succeeded by three or four rapid and indistinct pulsations" (ADAMS).

*In mitral regurgitation*, when the closure of the valve is very imperfect, the pulse becomes weak and small, and will intermit if the circulation is hurried; and when a considerable quantity of blood is permitted to regurgitate into the auricle, the pulse will also become irregular and unequal.

*In contraction of the aortic orifice* (when it becomes extreme only) the pulse becomes small, and intermittent or irregular, resembling the pulse of considerable constriction of the mitral orifice. When the contraction is slight, it is neither weaker nor smaller than natural, and is perfectly regular.

*In degeneration of the muscular tissue* the pulse in the advanced stages is small, weak, irregular, and unequal, sometimes slow, and the impulse of the systoles fails to be propelled.

*In pericarditis with copious liquid effusion*, the pulse presents somewhat similar characters.

*During the formation of fibrinous concretions within the cavities of the heart* the pulse suddenly becomes small, weak, intermittent, and irregular.

## SECTION IX.—USE OF THE SPHYGMOGRAPH.\*

The *Sphygmograph* was originally devised by M. Marey to determine various points in the physiology of the circulation of the blood; and, as an instrument of the greatest accuracy, it could not fail to become of great value as an aid in determining the nature

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[\* See the First American edition of this work, 1867, vol. ii, p. 597; and *New York Med. Jour.*, vol. iv, 1867, p. 267.]

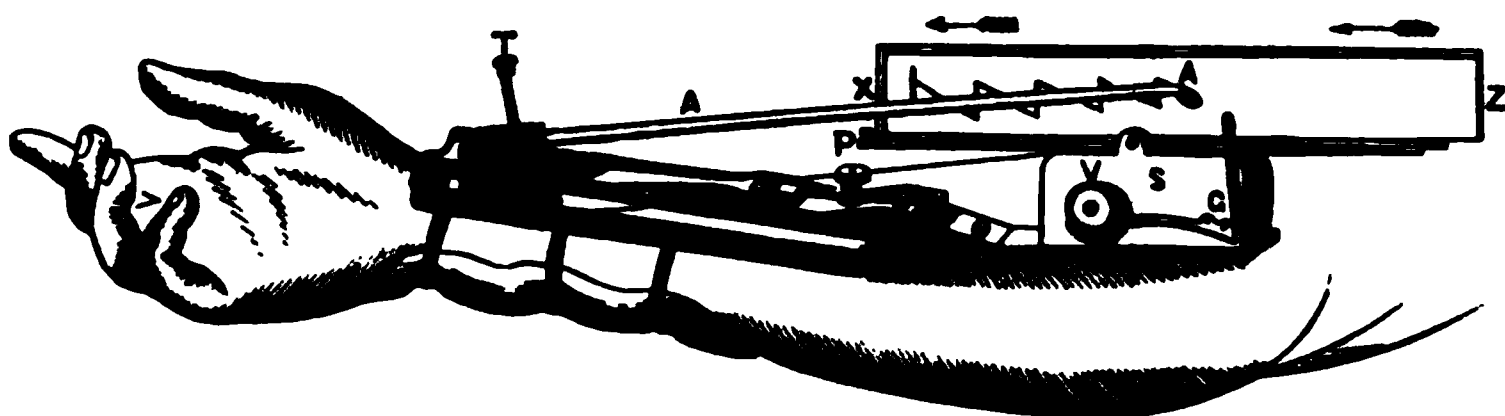




this movement is obtained by means of a very light lever, A, which moves upon a pivot, c. The elevation of the spring is transmitted to the lever, very near to its centre of movement, by means of a bar of metal, B E, which moves round the point E; this bar terminates in a vertical plate, B D, and is pierced by a screw, T. When the screw acts upon the spring, the connection is established between the spring and the bar, and the movements of the spring are transmitted to the bar, and through its vertical plate to the lever. In order to insure the transmission of the movements, the plate B D must be in contact with the under surface of the lever; by means of the screw T we can arrange this, and regulate the interval between the point of the plate B D and the under surface of the lever. In order that the lever should not be projected too much upwards by sudden movement, and also that it should overcome any slight friction experienced in the paper at its terminal point A, a small spring, Y, rests upon its fixed extremity, and presides over its descent. The screw, P, enables us to regulate the amount of pressure exercised upon the artery by the spring, I.

Fig. 18, modified from Marey, shows the instrument placed upon the arm over the radial artery in the position for use. The lever, A, is here

FIG. 18.



Sphygmograph applied to the arm.

seen to carry at its free extremity a little pen, which, filled with ink, registers its movements upon the paper which covers the plate x z; this plate is moved at a uniform rate in the direction indicated by the arrows by means of watch-work placed beneath in the case, s. Ten seconds are occupied by the passage of the plate. The button, v, enables us to wind up the watch-work; and the small regulator, g, starts the plate, or stops its motion, as desired. The application of the instrument Dr. Foster has found much facilitated by the use of elastic bands, instead of a silk lace, as recommended by Marey. These bands embrace the arm, and are hooked on to the small projecting points on the metal framework, as seen in the diagram. The addition of a pad, suggested by Mr. Waters, to the under surface of the arm, renders the instrument more easy to the patient, and prevents any pressure from the bands.

[Several very important changes in certain details of Marey's Sphygmograph have been made recently. The French instrument had certain faults which rendered its use by one not perfectly skilled in its application quite uncertain, and a deceptive record very likely. The several changes which have been made in the instrument in England by Mr. Berkely Hill, Dr. Burdon Sanderson, and Dr. F. E. Anstie, have removed many of these difficulties, made it of relatively easy application, and insured more uniform and reliable results. The improved instrument is shown in Fig. 19, and is described by Dr. Anstie in the *Lancet*, No. xxxv, 1868.]

The chief annoyance in Marey's instrument was the clumsy method of *adjustment*, by which much time was lost. It is necessary that there should be a uniform position of the hand and arm, that the limb should be solidly maintained in that position, and that the fastenings should be such that when once the tactile spring has been so placed on the artery as to receive its fullest pulsations, the tracing may be immediately taken. Mr. Hill has secured these objects by the pad on which the arm rests, and the elastic bands by which the sphygmograph is held in place (Fig. 19). The hand is bent back over the thick end of the cushion, and fastened by an elastic strap across the palm, which strap should be always drawn tight enough to make the knuckles touch the table or bed. By this means the radial artery is well thrown out.

FIG. 19.



Hill's, Sanderson's &amp; Weiss's Improved Sphygmograph.

The new apparatus for the *graduation* of the pressure of the tactile spring upon the artery, the principle of which originated with Dr. Burdon Sanderson, has put a new value upon the sphygmograph, increasing indefinitely the precision of its record. He found the large centre button or screw (r Figs. 17, 18), devised by Marey to regulate the amount of pressure which the spring exercises upon the artery, cannot be used so as to moderate pressure in uniform gradation, even for high pressures, while for the weaker pulses it does not provide the means of adequately weakening the spring. Dr. Sanderson adopted the principle of *fixing* the centre button r at a definite pressure of 400 grammes, which he found was the best maximum for the most resistant pulses; and when this pressure required to be lessened, it was done by putting pads or blocks of different thicknesses under the wrist-end of the instrument, and screwing down the free end of the spring in a corresponding degree. The distance between the tactile spring and the writing lever, having been measured in tenths of an inch, when, say 300 grammes pressure was on (for there are few pulses for which 300 grammes is not enough pressure) was remeasured after the free end of the spring had been extended sufficiently to develop the maximum pulse. The proportion between the two distances showed the amount to which it had been lowered—*e. g.*, if at 300 grammes, the distance was  $\frac{2}{3}$ ths of an inch, then an increase of the distance to one inch would mean a reduction of pressure to 150 grammes; and so on.

The mechanical application of this principle was done by Weiss &

Son, and Mr. Meyer, of London. Instead of the inconvenient and inexact blocks or pads, an apparatus was devised to regulate the extension of the free end of the spring, of which Weiss's modification is represented at G, Fig. 19. By turning the screw at G, after the sphygmograph is fastened on the arm at 300 grammes pressure, the instrument can be separated from the artery exactly to such distance as shall insure that when the free end of the spring is screwed down so as again to rest on the artery, the maximum pulse-curve will be secured. By measuring *the distance between the tactile spring and the horizontal brass rod immediately above it* (Fig. 19), before and after the reduction of the pressure, two numbers are got, the proportion of which to each other shows the strength of pressure which produces a maximum pulse-curve. The measuring instrument is simply a little piece of boxwood, of about one inch and a half in length, graduated in tenths of an inch, and set in a little wooden handle. Dr. Anstie has added a scale, which, under certain conditions, shortens the process of measuring the pressure.

The value of being able to mathematically estimate the resisting power of a pulse, which is due to Dr. Sanderson, is very great, and its practical purposes numerous. If, for example, on examining a patient with febrile symptoms, no matter how caused, it is found that the pressure best suited to bring out the maximum pulse-curve is very low, say 100 to 120 grammes, it is pretty certain that the case will require stimulants. If, on the other hand, the pulse requires 200 grammes power to bring out its maximum trace, then it shows that stimulation is not yet needed.

Another improvement is the abolition of the check-spring, which used to guard the ascent of the writing lever, which was found to be useless, and even to cause false tracings. Finally, another source of fallacy, the pen, ink, and paper of the writing apparatus, has been got rid of, and glass slides, blackened with the smoke of paraffine, and a writing lever armed with a needle-point, have been substituted. Friction is thus reduced to a minimum, and the resulting traces are delicate and beautiful. They may be preserved by covering the slide with photographic varnish.]

The sphygmograph, when in action, gives an exact representation of the pulse form—the frequency of the pulsations, and their regularity. It enables us to see at a glance any peculiarity in the entire series, or in any single pulsation. A trace, as below, is com-

FIG. 20.



posed of a series of curves, each of which corresponds to a complete revolution of the heart, and is called a *pulsation*. Each pulsation is composed of three parts—the line of *ascent*, the *summit*, and the line of *descent*. The line of *ascent* is produced during the flow of blood into the arterial system after each cardiac systole; and tells us, by its form, the manner in which the blood enters the vessels. The more rapid the afflux, the more quickly the pressure in the arteries will be elevated, and the more vertical will be the ascent of the lever. When the entry of the blood is slow, the line of ascent will be traced by the lever obliquely, sometimes in a curved form. The line of ascent, in certain morbid conditions, exhibits a mixed form



—the first part of the trace being vertical, the latter part curved. The *summit* of the pulsation corresponds to the duration of the arrival of blood in the artery, and designates the period during which the entrance balances the onward flow—in other words, the afflux and efflux are exactly equal. This period varies in length: it may be so short, that the summit becomes a mere mathematical point between the lines of ascent and descent; or it may be so long as to render the summit a horizontal line of some length. In the latter case, the lever traces the horizontal line, whilst the entry of blood into the vessel and its passage onwards mutually balance one another, and this line indicates the duration of the cardiac systole. The summit of the pulsation, when of any length, is not always horizontal; it may be formed by an ascending or descending plane,

FIG. 21 \*



according as the afflux predominates over the onward flow, and *vice versa*. The line of *descent* corresponds to the fall of the pressure in the arterial system, and is synchronous with the interval between the closure of the sigmoid valves and the next ventricular contraction. This line, by its obliquity, marks the celerity of the fall of the pressure within the vessels, and indicates the facility with which the blood passes on in its course. The amplitude of the pulse is indicated by the obliquity of this line. The form may vary very much; sometimes it is purely oblique,

at others a curve convex upwards, and occasionally one or more undulations may be seen in it. This last peculiarity is often very

\* A typical radial pulse-trace, enlarged (after Dr Foster). (a to b) Line of ascent; (c) First secondary wave, d) Second secondary wave, or true diastole; (e) Third secondary wave, not generally seen. The notch in which c is placed corresponds to closure of the aortic valves. The first secondary wave, c, should be situated at the junction of the *upper third* with the *lower two-thirds* of the line of ascent, a, b, about the level of the dotted line. From a to f the ventricle is contracting or contracted and f marks the closure of the aortic valves. The fourth wave, e, is seldom marked, but the three others can generally be distinguished, except when the tension is very high. The line of ascent corresponds to the ventricular systole, and marks the flow of blood into the aorta and great vessels.

The line of descent is generally broken by the occurrence of several secondary waves, which correspond to the vibrations in the blood column, alternately to and from the heart. The summit of the trace is usually followed by a small notch, and then the trace rises again to form the first secondary wave, c. The relative position of this wave to the summit part is of most practical importance to observe.

The deep notch in which the third secondary wave, e, is situated, and which precedes the second secondary wave, d, is of great importance, as it marks the closure of the aortic valves. It corresponds with that reflux of blood towards the heart which forces together the lappets of the valves; and the second secondary wave, or diastole proper, corresponds to the vibrating motion towards the periphery given to the blood by the sudden flapping together of the aortic valves, and the prevention of regurgitation into the left ventricle.

From a to the deep notch preceding d, corresponds to the period of the heart's systole and measures the duration of each cardiac contraction. From the deep notch preceding d to the beginning of the next line of ascent marks the period of the heart's diastole (Dr Foster's MS. Notes).



marked, and in some cases is perceptible to the finger; it has been termed *dicrotism*. By this term we should only understand undulations occurring in the line of descent. Similar peculiarities have been pointed out in organic diseases of the heart; but in these cases the dicrotism belongs to the period of ascent, and is due to very different causes. The sphygmograph enables us to recognize in which period the peculiarity occurs. Thus, conditions which are associated with great elasticity of the arterial walls, as dilatation of the vessels and easy passage of blood through the capillaries, are marked by evident dicrotism in the line of descent. The following trace (Fig. 22), taken during the perspiration of hectic shows this characteristic.

FIG. 22.



Senile change (Fig. 23) in the vessels, and consequent loss of elasticity, is indicated by,—(1.) The absence of dicrotism; (2.) By the great dimensions of the curve; (3.) By the closer proximity not only of the first but of all secondary ascensions to the apex of the curve; (4.) By the extreme predominance of the first secondary wave; and in near relation to this pulse of old age stands the radial pulse of people not far advanced in life, and suffering from hypertrophy of the left ventricle; and next, that which accompanies insufficiency of the aortic valves.

The great parts to be noticed in the senile pulse are the high positions of all the secondary waves in the line of descent, and the enormous size of the first secondary wave as compared with the second secondary wave or true *dicrotism*.

FIG. 23.



In examining a pulse-trace, one should note, in addition to the form of each pulsation, whether the summits of all of them can be joined by a straight line, and whether the bases can be also connected by a similar line parallel to the former. In some instances this ceases to be the case, and a series of pulsations cannot be contained between such imaginary lines. The pulsations become irregular, and the line to join their summits or bases must cease to be horizontal. The line joining the summits of a series of pulsations is the line of the maxima of *arterial tension*. Its value as an indication is not absolute; it only tells us the variations that the arterial tension may undergo during the period of the observation; and it enables us to judge of the relative pressure within the vessels during any of the cardiac contractions registered. This line of greatest tension is of much value, and, with the corresponding line of

tension, should be observed in all cases; as these lines generally undergo parallel deviations, and a glance at either usually suffices to inform us of any change. Different *diseases* cannot be recognized by pulses peculiar to them; but the pathological changes which the normal curve of the radial artery undergoes takes place simultaneously with the changes indicated by the rise or fall of temperature in fever (WOLFF).

The *frequency* of the pulse may be studied by means of the sphygmograph; for, as the plate moves at a uniform rate, and occupies exactly ten seconds in its passage, we can with ease calculate the pulse-rate. Slight variations in frequency, and irregularities that would most probably escape appreciation by the unaided touch, are by this means revealed. The frequency of the heart's action, according to the French physiologist, depends very much upon the state of the circulation in the vessels of the periphery—an easy passage of blood favoring the increased action—a difficult passage (by reason of the greater arterial tension) causing diminished frequency of the ventricular systole. The law is thus laid down by Marey that, in the majority of cases, "*the frequency of the pulse—i. e., the number of contractions of the heart—varies inversely to the arterial tension;*" but Onimus and Viry maintain that "*the number of contractions of the heart varies directly with the initial force.*"

Even if a pulse trace fails to indicate any specific lesion, the sphygmograph is the most exact measurer of tension and index of a *hard* or *soft* pulse. When we find the pulse-trace without any well-marked notch *before* the second secondary wave, and the line of descent forming an almost unbroken oblique line, it indicates *high arterial tension*, with arteries unusually full, giving the free, incompressible hard pulse. On the other hand, when the line of descent falls suddenly, the arteries are insufficiently filled, and the pulse is known as the *soft compressible pulse*.

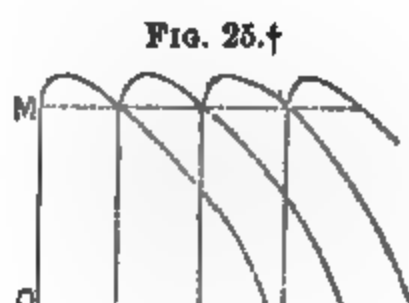
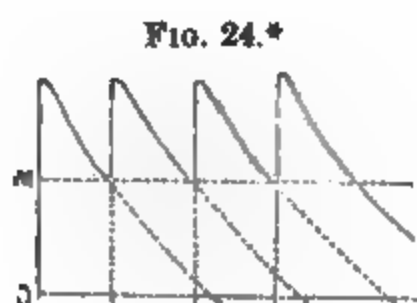
The *force* of the pulse is indicated by the height of the pulsations. The greater the elevation of the lever, the greater the energy of the pulse-beat; and we may say that, in many cases, the strength of the ventricular contraction is expressed by the force of the pulse. This law, however, has many exceptions; and we find that the altitude of the pulse-trace depends on several other conditions. For example:

(1.) The volume of the artery greatly influences the amplitude of the trace. This can be well seen in traces collected from old persons. In senile changes (Fig. 23) the volume of the vessels is increased considerably, and the trace betrays great fulness. Marey believes this to be due, not solely to the hypertrophy of the ventricle which exists in the old, but also to the dilatation of the artery (Fig. 23).

(2.) The state of arterial tension modifies greatly the force of the pulse; and, as the tension is dependent on the state of the capillary circulation, it may be said that in most cases "the force of the pulse is not in relation with the energy of the ventricular systole, but that it is regulated by the state of the circulation in the ultimate ramifications of the vascular system." By means of the manometer, in a great number of experiments this law has been proved to hold

good—a feeble state of arterial tension giving to the finger and the instrument the sensation of increased amplitude. The following diagrams (Figs. 24 and 25) illustrate this (MAREY).

The difference in the amplitude of the traces is very distinct. In the state of feeble tension, or easy passage of the blood onwards,



the lever falls quickly to the point of least tension, and is elevated considerably at each pulsation. In the case of difficult passage of the blood through the capillaries, and consequently of great arterial tension, the lever descends slowly by a line convex upwards; and, long before it has reached a minimum tension equal to that in the former case, the lever is raised slightly by the next pulsation. While the lines of the maxima of arterial tension are the same in both cases, the lines of the minima are very different. On this depends the amplitude of the pulse-trace.

(3.) The duration of the interval which separates the pulsations has also a distinct amplitude of the trace. This is due to the fact that, during a long interval, the blood flowing continually onward lessens the pressure in the vessels, and thus favors the greater amplitude of the next pulsation. This is well seen in a trace (Fig. 26) taken from a patient of Dr. Foster's in the Queen's Hospital, Birmingham.

FIG. 26.



The condition of the vessel itself, as to permeability below the point observed, influences the force of the pulsation by altering the pressure within the artery. Marey has also pointed out that in some cases, where the pulse-beat is almost imperceptible to the finger, the sphygmograph records a considerable amplitude of trace; and *vice versa*. Such cases have been associated with a very slow distension of the vessels.

But it is now shown that the modes of adjusting the instrument have a great influence on the result. For example, by a more accurate apportionment of the pressure of the tactile spring to the power of the pulse, very much larger and more developed curves can be obtained. Under certain circumstances, also, Dr. Anstie has shown that alterations of the weight of the writing lever may greatly increase

\* The form of pulsation in a state of feeble tension.

† Under a state of strong tension.

the size of the curves obtained—a heavy lever suiting a strong pulse, and a light lever a weak one. Drs. Beigel, Sanderson, and Anstie have each shown the advantage of suspending weights in such a position on the writing lever as to exercise more or less pressure; or of weakening the spring by placing pledgets of lint or pieces of wood under the brass work, so as to ease off the pressure in taking the traces of very weak pulses. Also, Dr. Anstie points out that the highly significant trace of delayed systole (represented by the square-headed trace) may be spuriously manufactured by the instrument under either of two circumstances—namely, (a.) Too great friction between the pen and paper; and (b.) Too great resistance by the check-spring. This check-spring Dr. Anstie considers quite useless.

The discrimination of increased arterial resistance, as measured by the exaggeration of the systolic expansion, is the element of diseased action, whose detection and estimation, according to Dr. Sanderson, are the most important purposes to which the sphygmograph can be put. "Anatomical researches lead us to believe it probable that the earliest beginnings of what we may call degenerative disease consist in such structural alterations of the minutest arteries as, by rendering them less pervious to the circulating blood, must inevitably lead to increased arterial resistance estimable by the sphygmograph; and if by such an examination," continues Dr. Sanderson, "we can ascertain that the heart is overtaxed long before any change can be detected by auscultation or percussion, it is obvious that we have made a step forward in practical utility." Thus, he considers it likely that the *sphygmograph* is to be of greater use as an aid in forming an opinion as to the probable duration of life than in any other department of medical practice. There are many persons in whom, in the absence of any other trace of ailment, the pulse-curve indicates that the arterial resistance is excessive. The question is, "Are such persons sound?"

Dr. Anstie, on the other hand, is of opinion that the diseases in which the *sphygmograph* will prove of the greatest diagnostic value are—(a.) In aortic regurgitation, by estimating the amount of valvular imperfection; (b.) In discovering unsuspected commencing cardiac hypertrophy, senile disease of arteries, or capillary disease, dependent on degenerative processes in the ultimate tissues; (c.) Above all, in discovering the existence of intra-thoracic aneurisms, and in deciding the locality of an aneurism; (d.) In aiding prognosis and decisions as to treatment in the course of acute diseases. Dr. Anstie has already made a series of observations in fever, pericarditis, pneumonia, and delirium tremens, which show that in this direction the sphygmograph promises to be of great value, as the best indication of the use or otherwise of certain remedial agents.

## SECTION X.—GENERAL SYMPTOMS OF THORACIC DISEASE.

The elucidation of some of the topics, of which a short outline has been given in the previous sections, claims for the name of Laennec an immortal fame. He discovered how, *by means of auscultation*, disease might be detected. He not only accurately described the sounds heard in diseased states, and compared them with the sounds in health; but by morbid anatomy, in connection with careful clinical observation, he traced the mechanism of those sounds to anatomical and physical conditions of the organs with whose functions they were connected. The immediate effect of this inestimable discovery was to divert the attention of the physician from the study and observation of those vital symptoms and general states of the constitution so pregnant with information when correctly observed and properly appreciated. Now, it was the close observation of these vital symptoms, watching the order and the periods of their manifestations, and the modes in which they were combined, that distinguished the successful practice of our forefathers in medicine—such men as Cullen and the two Gregorys of former days. Disastrous results of treatment were the consequence of the circumscribed study of so-called physical diagnosis; “but what was lost in lives was gained in pathology.” The physician of the present day now knows better. He is a close observer of vital symptoms, of constitutional states, and of physical signs; and with all the delicate instrumental appliances to appreciate those signs, he forms his judgment from the combined evidence of them all.

The general symptoms which express derangement of the pulmonary organs and their functions are *dyspnœa*, *cough*, *expectoration*, *tenderness*, and *pain*. The general symptoms which indicate derangement of the cardiac apparatus are *palpitations*, *sinking*, and *fainting*, combined sometimes also with *dyspnœa*, *cough*, *pain*, and *tenderness*.

The sensation of *dyspnœa* is brought about by an embarrassed or laborious breathing, amounting in severe cases to a sense of suffocation, expressed by the common English phrase, “*want of breath*,” or by the meaning conveyed by the Latin term “*anxietas*,” when the *dyspnœa* is at the point of greatest intensity. It is aggravated by exertion, some positions of the body, and a full stomach. The act of speaking is frequently arrested “*to fetch a breath*,” and the patient who suffers from *dyspnœa* cannot hold the breath, or refrain from the attempt to inspire, as a person in health can. The *dyspnœa* may occur in paroxysms, and the acts of respiration may be painful. The difference between the *dyspnœa* of asthma and that of cardiac disease has been already given. (See p. 187, *ante*, as it is of extreme importance.)

The number of respirations performed in a given time is greatly increased, and often unequally so, when the paroxysm is aggravated. In health, from eighteen to twenty acts of respiration are unconsciously completed in a minute, according as a person is lying, sitting, standing, or walking; and the ratio of the acts of respiration



to the pulse varies in the proportion of one to four (WATSON), or one to nearly six (WILKS); that is, about one complete act of respiration for every *four* or *six* beats of the heart. But so intimately are the functions of the heart and lungs dependent upon each other, that any deviation from these proportions, in the acts of the one or other set of organs, immediately influences the actions of the other.

*Cough, expectoration, and the nature of the sputa*, furnish valuable indications of thoracic disease.

**Microscopical Elements of Sputa consist of**—(1.) Young epithelium-cells—*i. e.*, of mucous corpuscles; (2.) of mature epithelium in the form of pavement, cylindrical, or ciliated bodies; (3.) Cells containing granules, or bodies like cells made up of granules; (4.) Pus-cells; (5.) Colored corpuscles of the blood; (6.) Fibrine, either in the form of flake-like membranes, or in the form of casts of the smaller bronchi and pulmonary air-cells, as in the expectoration of pneumonia. They are sometimes seen as dichotomous cylinders with rounded enlargements, composed of fine filaments, generally covered with granules. They may be met with from the third to the seventh day in pneumonia. (7.) Fat occurs in granules or globules; (8.) Tubercle matter, earthy, calcareous, amorphous, and crystalline particles also occur; (9.) Substances derived from the food; (10.) Carbon and true pigment, free or contained in cells; (11.) Fragments of pulmonary tissue.

**Chemical Characters of Sputa.**—Much attention has not yet been given to the chemical characters of the sputa. Professor Laycock, of Edinburgh, had a chemical analysis made of very fetid expectoration in bronchitis, which demonstrated the existence in it of *butyric* and *acetic* acids. The odor was characteristic of the *butyrates of ethyl*, resembling the smell of may-flower or apple blossoms, combined with an odor of *fæces*. Chemical investigation may thus demonstrate the cause of the excessive fetor in those cases which resemble gangrene of the lungs, if the smell alone is considered (*Med. Times and Gazette*, May, 1857, p. 480). A much more extended analysis has been recently made by Bamberger into the chemistry of the sputa—(1.) In chronic bronchial catarrh; (2.) In bronchial dilatation; (3.) In chronic pulmonary tuberculosis; (4.) In the infiltration of acute tuberculosis; and (5.) In pneumonia. In sputa which is chiefly catarrhal the salts vary but little—the organic matters vary considerably. The insoluble salts form about 4 to 5.5 per cent. of the whole saline contents; the chief amount consists of *chloride of sodium* and *phosphate of potash*. Puriform matter predominating, causes the sputa to contain a greater quantity of organic and inorganic substances; there is considerably more *phosphoric acid* in the ash, considerably less *chlorine*, and less *sulphuric acid*. The ash of pneumonia sputa differs from that of catarrhal in several respects. The alkalies, combined with phosphoric acid (which amount to 10 and 14 per cent. of the saline constituents of catarrhal sputa), are almost entirely absent in pneumonia during the inflammatory period, but the sulphuric acid is remarkably increased. The quantity of chlorine (37 per cent.) is nearly the same as the average in the catarrhal (36 per cent.); and there is not much variation in the in-

soluble salts, except in the phosphates of iron derived from the blood. In the period of resolution the sputa of pneumonia become more similar to the catarrhal; the *phosphoric acid* increases, the *sulphuric* diminishes, and the chlorine reaches a very high amount, while the potash and soda are present in the same relative proportion as in the catarrhal, whereas during the inflammatory period this was inverted. Sugar has been detected during the height of the inflammation; and Dr. Beale has shown that an excess of the chloride of sodium is constantly present in pneumonic sputa. In the sputa of bronchiëktasis, sulphuretted hydrogen, acetic, butyric, and probably formic acids, were detected (*New Syden. Society Year-Book*, 1860, p. 128; Schmidt's *Jahrb.*, band 114, p. 3).

**Sputa Typical of Pneumonia** is characterized by its viscidness, semi-transparency, and tenacity, adhering strongly to the vessel containing it. So tenacious is it, that the vessel may be turned upside down without the sputa becoming detached from the sides. This rusty-colored sputa consists of mucus intimately mixed with blood—not streaked with it, as in bronchitis, but thoroughly mixed and amalgamated with it—so that it acquires a yellowish, or reddish-yellow, or even a red color, according to the quantity of the blood. If the disease be not very intense, the expectoration never attains the degree of viscidness or the depth of color above referred to; but though still tenacious and adherent to the sides of the vessel, moves from one part to another as the vessel is tilted. If the disease progresses to a favorable termination, the sputa become more abundant, less adhesive, and less highly colored, passing through the various shades of orange, until at length they become greenish or whitish, and resemble the expectoration of ordinary catarrh. If the disease be hastening to a fatal termination, the expectoration becomes scanty, less tenacious, and of a darker or dullish-brown hue, resembling the juice of prunes. If the type of inflammation be typhoid, or adynamic, or connected with tubercles in the lungs, the mucus may be tinged, or even streaked with blood; or it may consist throughout of nearly colorless, stringy, and more or less frothy mucus (Fuller *On Diseases of the Chest*).

**Sputa Typical of Gangrene of the Lung**, at first of a muco-purulent character, sometimes tinged with blood, begins to emit a very disagreeable odor; and as soon as a free communication is established between the air-passages and the sloughing tissue of the lung, they not only acquire an intensely fetid gangrenous odor, but assume an appearance more or less characteristic of the disease. They lose their muco-purulent character, and become extremely liquid or sero-purulent, and of a dirty greenish or ash-gray color. At the same time the breath acquires an offensive putrid odor, the pulse feeble and rapid, with evidence of great and increasing prostration (FULLER, *l. c.*).

**Sputa Typical of Acute Bronchitis** appear, after a few days, as a thin, saltish, frothy mucus, sometimes streaked with blood. They increase in quantity, and soon become glairy, semi-transparent, and of a faintly yellowish color. Subsequently they assume a grayish or greenish-yellow tint, and become opaque and viscid. If the

attack is severe, they become muco-purulent, and in some instances may even lose their glairiness, presenting the character of thoroughly opaque mummillated sputa.

In *chronic bronchitis* the sputa may be of the following characters: either (1.) The expectoration of a grayish, or greenish, or yellowish-white muco-purulent matter; or (2.) The expectoration being difficult, the sputa are comparatively scanty, consisting of stringy, tenacious mucus, of a grayish or yellowish-white color, occasionally streaked with blood; at another time, expectoration being easy, the sputa are more copious, muco-purulent in character, of a yellowish-green color, having a faint unpleasant odor; at another time the sputa are profuse, almost wholly purulent, of a nauseous and sometimes a fetid odor, usually running together into one mass, but often remaining separated, and forming distinct mummillated masses; or (3.) There is a profuse expectoration, sometimes to the extent of half a pint in an hour, of a thin, watery, ropy fluid, which varies in opacity, but is usually somewhat transparent, resembling gum-water (FULLER).

**Sputa Typical of Plastic Bronchitis** consist of ordinary bronchitic sputa, or blood-tinged mucus, with fragments of white fibrinous matter, or white fibrinous casts of the bronchi, which are ejected during violent paroxysms of cough. These concrete masses vary from mere fragments to large pieces of from *one* to *four* inches in length, and may be either tubular or solid, their ejection being preceded and often accompanied by spitting of fluid blood. These casts consist of concentric laminæ, found at different periods in successive layers, and consist of amorphous granular matter intermixed with mucus-corpuscles, compound granular cells, oil-globules, and ovoid cells containing dark coloring matter, such as exists in ordinary bronchial mucus (FULLER).

**Sputa Typical of Acute Phthisis** consists—(1.) Of frothy mucus, often speckled with blood; and when the tubercle softens, the sputa become muco-purulent or purulent; (2.) The sputa may be scanty, consisting of little more than frothy mucus; (3.) Expectoration, at first scanty, thin, colorless, and transparent, somewhat resembling saliva or gum-water, of a grayish color, and more or less frothy. After a time the thin colorless sputa lose some of their transparency, and are seen to contain specks of opaque matter, which gradually subside and form a deposit resembling the sediment in barley-water; or they remain suspended by the more ropy part of the secretion, and float in the transparent mucous fluid in the form of striæ. Gradually becoming less aerated, they become more glairy and more tenacious, lose their pearly-gray color, and are seen to be mixed with specks or streaks of an opaque white, or buff color, and not unfrequently with specks or streaks of blood. (4.) As the malady progresses, the characters of the sputa change again. They become opaque, of a whitish or yellowish hue, and are coughed up in more distinct and homogeneous masses. Sometimes they form rugged pellets of a yellowish-white color, resembling boiled rice, which sink or partially float in a colorless semi-transparent, ropy, non-aerated mucous fluid; or, ac-

accompanied by little mucous fluid, the sputa form large masses of a buff or yellowish-green color, flocculent in appearance, but perfectly smooth in outline, which do not coalesce, but remain distinct and separate from each other if expectorated into a vessel of water.

All these forms of sputa occasionally occur in *chronic bronchitis* as well as in *phthisis* (FULLER).

**Cough.**—The severity, the frequency, or paroxysmal nature of the cough must be ascertained; also the circumstances which excite it most; and whether it is attended with pain, or followed by expectoration or vomiting. The ease or difficulty of the expectoration must be noted, and whether it is accompanied or not by pain. The quantity of the sputa ought to be measured in the day and night, the form of the masses spat up, their transparency or opacity, color and viscosity, tenacity or adhesive property. The special characters of the sputa ought in every case to be closely observed, noting particularly its thin, serous, or frothy character; whether it contains any membranous or concrete exudation-masses or blood; and it should be examined microscopically.

**Pain.**—The exact locality of pain in the chest should be ascertained, its severity, and the direction it tends to take. What particular circumstances aggravate it, and the effects of breathing, coughing, pressure, and postures should be ascertained.

**Palpitation.**—When *palpitation* occurs, its severity ought to be estimated by laying the hand over the region of the heart of the patient. It is desirable to ascertain its constancy; the circumstances which aggravate its existence or produce it—such as the influence of exertion going up a hill or up stairs, and the influence of mental emotion (*What to Observe*, pp. 39–44).

In the following table the more prominent characters of the palpitation which depends upon organic disease of the heart are contrasted with those of palpitation arising independent of disease of this organ (BELLINGHAM, *op. cit.*, p. 172):

**PALPITATION DEPENDING UPON ORGANIC DISEASE OF THE HEART.**

1. More common in the male than the female.
2. Palpitation usually comes on slowly and gradually.
3. Palpitation constant, though more marked at one period than at another.
4. Impulse usually stronger than natural: sometimes remarkably increased, heaving, and prolonged; at others irregular and unequal.
5. Percussion elicits a dull sound over an increased surface, and the degree of dulness is greater than natural.
6. Palpitation often accompanied by the auscultatory signs of diseased valves.

**PALPITATION INDEPENDENT OF ORGANIC DISEASE OF THE HEART.**

1. More common in the female than the male.
2. Palpitation usually sets in suddenly.
3. Palpitation not constant, having perfect intermissions.
4. Impulse neither heaving nor prolonged; often abrupt, knocking, and circumscribed, and accompanied by a fluttering sensation in the præcordial region or epigastrium.
5. Extent of surface in the region of the heart, which yields naturally a dull sound on percussion, not increased.
6. Auscultatory signs of diseased valves absent; bruit de soufflet often present in the large arteries, and a continuous murmur in the veins.

## PALPITATION DEPENDING UPON ORGANIC DISEASE OF THE HEART.

7. Rhythm of the heart regular, irregular, or intermittent; its action not necessarily quickened.

8. Palpitation often not much complained of by the patient, occasionally attended by severe pain, extending to the left shoulder and arm.

9. Lips and cheeks often livid; countenance congested; anasarca of lower extremities common.

10. Palpitation increased by exercise, by stimulants, and tonics, &c.; relieved by rest, and frequently, also, by local or general bleeding, and an antiphlogistic regimen.

## PALPITATION INDEPENDENT OF ORGANIC DISEASE OF THE HEART.

7. Rhythm of heart usually regular, sometimes intermittent; its action generally more rapid than natural.

8. Palpitation often much complained of by the patient; readily induced by mental emotion; and frequently accompanied by pain in the left side.

9. Lips and cheeks never livid; countenance often chlorotic; anasarca absent, except in extreme cases.

10. Palpitation increased by sedentary occupations; by local and general bleeding, &c.; relieved by moderate exercise, and by stimulants or tonics, particularly the preparations of iron.

**Expression of the Countenance in Thoracic Disease.**—The countenance is often expressive of heart disease. In acute inflammatory affections of the lining or investing membrane it acquires an anxious and depressed character. An elevation and depression of the *alæ nasi* or *nares* are commonly observed with the respiratory acts; and the occurrence, in children, of these phenomena indicates a greater amount of disease than the general symptoms would lead us to suspect. In chronic cases, when the circulation is impeded, the expression of the countenance becomes almost pathognomonic. The venous system becomes congested, the face becomes bloated and dusky, the eyelids puffed, the eyes staring, the conjunctiva suffused, the lips and cheeks purple, respiration laborious, the air-passages loaded with mucus, and the jugular veins distended or pulsatile.

## CHAPTER IX.

DISEASES DURING WHICH LESIONS TEND TO BE LOCALIZED WITHIN OR ABOUT THE THORAX, ITS VISCERA, AND VESSELS THEREWITH CONNECTED.

## SECTION I.—DISEASES OF THE WALLS OF THE THORAX.

IN connection with thoracic pathology there are especially three affections which derive their interest, or rather importance, from the possibility of their being taken as evidence of pulmonary or cardiac disease. These are,—*pleurodynia* or *intercostal rheumatism*, *intercostal neuralgia*, and *thoracic myalgia*.

## PLEURODYNIA—Syn., INTERCOSTAL RHEUMATISM.

**Definition.**—*Pain more or less acute, sometimes of agonizing severity, due to Rheumatism of the walls of the chest, affecting especially their*



*muscular and fibrous textures. The pain is most common on the left side, in the infra-axillary and infra-mammary regions. It is increased by deep inspiration, by coughing, by movements of the trunk and even of the arm, by decumbency on the affected side, and by pressure both on the ribs and in the intercostal spaces (WALSHE).*

**Pathology and Diagnosis.**—The constitutional diathesis associated with rheumatism coexists with this affection; and when the pain coexists with acute articular rheumatism, the affection is associated with fever. It simulates the congestive or dry stage of pleurisy, and if febrile phenomena coexist, the diagnosis becomes extremely difficult; and therefore a positive opinion should be withheld until, a certain number of hours having elapsed, the presence or absence of the characteristic friction-sound shall have established the nature of the case. If the pain extends very low down, it may simulate *peritonitis*; but the absence of rigors, of vomiting, of febrile phenomena, of the anxious expression of countenance characteristic of that disease, will always distinguish pleurodynia from peritonitis (WALSHE).

**Treatment.**—Friction with anodyne and stimulant liniments; the warm, vapor, or hot-air baths; rest; with colchicum in small, and bicarbonate of potash in free and oft-repeated doses, are the means which the pathology of the disease suggests as likely to effect a cure (WALSHE).

#### INTERCOSTAL NEURALGIA.

**Definition.**—*Pains intercostal, occurring paroxysmally, following the course of the affected nerves, usually involving the nerves of the thorax from the sixth to the ninth; more common on the left than the right side. The pain sometimes passes directly backwards from the edge of the sternum to the vertebral groove, and is frequently accompanied with pain in the branches of the brachio-cephalic plexus, sometimes in the gastric filaments of the vagus. In the intervals of the sharper pangs, numbness, coldness, and formication are occasionally felt (WALSHE).*

**Pathology and Diagnosis.**—This form of neuralgia is usually associated with hysteria and anæmia, especially in women weakened by *menorrhagia* or *leucorrhœa* (see Neuralgia, p. 519). It is frequently associated with phthisis. Three tender points in the course of the affected nerves indicate the true nature of the disease. These painful points are to be detected by pressure—one in the vertebral groove, another about the axillary region, a third in front towards the terminal ramusculi of the nerves. There is sometimes general cutaneous hyperæsthesia in the affected region; and gradual but firm pressure over a hard surface usually gives relief. Months may elapse before a cure is effected; and the anæmic form is the most easily got rid of.

**Treatment.**—If local tenderness is extreme, leeches may afford relief, followed by a repetition of small blisters. A minor amount of pain, recurring from time to time, is best relieved by the endermic use of *morphia*, or by the inunction of ointments containing

*belladonna* or *aconite*, or their *alkaloids*. *Quinine*, *arsenic*, and *iron* may be advisable in some cases from time to time (WALSHE).

#### THORACIC MYALGIA.

**Definition.**—*A painful affection, having its seat in the fleshy parts of the muscles of the chest-walls, especially both pectorals, accompanied with exaggeration of the "muscular sense." It may be wholly subjective, or it may be a genuine hyperæsthesia excited by pressure and manipulation of various kinds* (WALSHE).

**Treatment.**—Rest, where the myalgia arises from over-work; while in the idiopathic variety, tonics, antispasmodics, and local anodyne applications, followed by stimulating douches, will generally effect a cure.

### SECTION II.—DISEASES OF THE HEART AND ITS MEMBRANES.

**Of Organic Diseases of the Heart.**—Our knowledge of the anatomy as well as the pathology of the heart and large bloodvessels may be said to begin with Harvey; but the subject can hardly be said to have taken a scientific form till the beginning of the present century, when the work of Corvisart appeared, followed by those of Burns in England, of Testa in Italy, of Kreysig in Germany, and by the works of Bertin, and more especially of Laennec, Bouillaud, Senac, and Collin in France. A large school has since been formed in Europe by the labors of these eminent pathologists.

Nineteen years ago Dr. Williams and Dr. Hope, in their respective treatises, showed how it was possible to make an accurate and minute diagnosis in almost every case of cardiac diseases; and the subsequent labors of Stokes, Graves, Bellingham, Forbes, Walshe, Davis, Sibson, Latham, Fuller, and Gairdner, in this country, have brought the pathology of diseases of the heart and lungs to their present advanced state of perfection.

The lesions of the heart are comprehended under diseases of the *Pericardium*, *Endocardium*, *Muscular Structure of the Heart* and of the *Bloodvessels*.

#### PERICARDITIS.

LATIN Eq., *Pericarditis*; FRENCH Eq., *Péricardite*; GERMAN Eq., *Pericarditis*—Syn., *Entzündung des Herzbeutels*; ITALIAN Eq., *Pericarditide*.

**Definition.**—*An inflammation of the fibro-serous membrane containing the heart, and investing it on its external aspect.*

**Pathology and Morbid Anatomy.**—The pericardium, like other serous membranes, is liable to inflammation, tending to the effusion of a serous fluid, diffused over a large extent of surface, and which sometimes becomes purulent—*suppurative pericarditis*. The morbid action may be of such a kind that the serous fluid coagulates upon the opposed surfaces, becomes vascular, and glues them together. The different forms of morbid action may coexist, so that

different parts of the pericardial surfaces may present very different appearances when examined after death. The inflammation may be acute or chronic, and attended with various productive results of the inflammation.

If the patient dies from acute pericarditis, the inflamed portion is of a bright rose color. This redness is, in the first instance, caused by the increased vascularity of the subjacent areolar and fibrous tissue; but as the disease advances, red blood penetrates the serous membrane, and punctuates the exudation with a number of dots, which become confluent, and form patches that extend till perhaps the whole membrane is one bright scarlet color. Besides being red, the membrane is thickened, first from coagulable lymph upon the surface, and then from the incorporation of interstitial growth with the subserous tissue. It is now opaque, white, thickened, and readily torn from its attachments; but the new membrane forms so commonly a covering to the heart that the pericardial sac has to be forcibly opened up, so as to expose the organ within.

**Dropsy of the Pericardium.**—The inflammation may terminate by resolution; but more commonly serous effusion results, besides the solid lymph, the quantity of serum effused varying from a few ounces to a few pints. The solid lymph is then often found in shreds. Louis has given one case in which it amounted to four pounds, and Corvisart another in which the quantity was still more considerable. Walshe records sixty ounces—and when such great effusions occur, the diaphragm is depressed below the end of the sternum, by the great amount of fluid contained in the sac of the pericardium.

The adhesive results of inflammation often coexist with the preceding inflammation, and lymph is generally formed in much greater quantity than from any other serous membrane. The lymph may be only in such quantity as to render the serum turbid, or so extremely loose in its texture as to float in it. More commonly, however, it is disposed as a membrane, often covering both surfaces of the pericardium, and especially that covering the heart, and measuring from two to several lines in thickness. This mass, when considerable, presents a remarkably irregular appearance, which has been compared to the stomach of a calf, to a portion of a honeycomb, or to two opposed surfaces united by grease and then forcibly separated. If the patient dies in the acute stage, this membrane is found only slightly coherent, and very rarely exhibits any trace of organization.

**Suppurative Pericarditis.**—When acute inflammation results in an accumulation of pus in the pericardium (which it does slowly), it is generally associated with a constitutional cause, and the pus may be of a laudable healthy character, though sometimes of a greenish hue. The quantity may be only a few ounces, or so abundant as to fill the pericardium.

**Hemorrhagic Pericarditis.**—In cases where there is a disposition to purpura or scurvy, or during the progress of some malignant febrile disease, it occasionally happens that blood as well as serum

or pus is effused within the sac. The source of the blood is from the new-formed vessels, which, being yet tender, give way; or it may be from the highly congested vessels of the serous membrane, and the softened tissues which prevail in scorbutus. The lymph of such cases is of a spongy red color and shaggy appearance, exactly similar to that which extends from the large intestine into the small gut in cases of scorbutic dysentery.

**Tubercular Pericarditis.**—In the scrofulous cachexia, where *tubercle* of the lungs exist, pericarditis with minute tubercle on the pericardium may be also found. It is a common form of pericarditis in the army.

**Adherent Pericardium.**—*In the chronic forms of pericarditis* all these morbid states may be observed; and when lymph has been effused, it is then commonly found organized, so that the pericardium is often partially or universally adherent all over the heart. In some instances the lymph effused, instead of forming adhesions, becomes converted into cartilaginous and even osseous patches, which are readily detached from the surface of the heart by the scalpel.

*The acute forms of pericarditis* generally involve the muscular walls of the heart to a greater or less extent. On cutting through them the muscular substance is seen for a greater or less depth of a deeper color than usual, and the cohesion of the tissue is also impaired, the finger readily passing through it.

**General Symptoms.**—The symptoms of pericarditis vary much as regards their expression. In some instances they are most insidious in their approach; in others they appear to be violent and unmistakable from the commencement. In this disease rheumatism has been found to assume a new and formidable aspect; and rheumatic pericarditis is generally attended with more violent symptoms than non-rheumatic pericarditis. The most marked characteristic of rheumatic pericarditis is pain more or less severe in the præcordial region; and from this point it radiates over the whole of the sternum, sometimes extending to the brachial plexus and down the left arm. This pain is accompanied by disturbance of the heart's action, a sensation of constriction over the whole chest, by urgent distress, and by an incapacity to take a long breath, or to cough. From these causes the patient is restless and anxious, and this anxious expression of the countenance is often peculiar and striking from the first. When acute pericarditis is not the result of rheumatism, the patient may suffer no pain, and the symptoms are often most obscure, general as well as physical. The countenance is pallid, and assumes an aspect of distress, and there is an incapacity or unwillingness on the part of the patient to lie on his left side. The pain in the region of the heart is increased by pressure upwards against the diaphragm; his pulse, varying from 90 to 110, full and strong, and often intermittent, or otherwise irregular; and this state of things having lasted from three to four days to a week, the patient may die suddenly.

Before death, and often throughout the more severe periods of the disease, there is delirium of a peculiar kind—sometimes quiet, but often wild and furious. This delirium is peculiar, and has been noticed particularly by Drs. Watson, Burrows, and others.

Even when the disease is most unmixed, it has been mistaken for a continued fever, or for pleurisy; and being usually one of the first complications of an otherwise fatal disorder, its mortality is thus far in excess of rheumatic pericarditis, which proves fatal in about one out of every six cases (FULLER).

Pure idiopathic pericarditis is rarely witnessed; and it very rarely occurs as a severe or clinically important form of disease.

Dr. Stokes, in respect of morbid anatomy, arranges cases of pericarditis into three classes. In the first class are to be placed those in which there is a slight though general effusion of coagulable lymph. In the second, those in which there is superadded the secretion of serum in abundance, causing distension of the pericardial sac. In the third class are to be placed those cases in which signs of muscular excitement, if not of myocarditis, are added to the preceding conditions. These three classes are thus contrasted, in respect of their symptoms or diagnosis, in the following tabular arrangement, given by Dr. F. Sibson:

FIRST FORM.	SECOND FORM.	THIRD FORM.
Absence of pain or local suffering frequent. No sign of muscular excitement, nor any special character of pulse. No increase of dulness over the heart.	The local and general symptoms more decided, though often very trifling. Irregular action of the heart and pulse, often very manifest in the advanced periods. Remarkable increase of dulness over the heart.	Local distress, often extreme, even at the outset. Tumultuous action of the heart. Irregularity of pulse. Dyspnœa, orthopnœa, œdematous swellings, syncope, death.

As the disease passes from the first to the last of these forms, there is a progressive increase in the violence of the inflammation, denoted in the second form by the occurrence of excessive serous effusion, and in the last by the altered and impaired condition of the muscular substance of the heart itself. Death tends to occur by syncope, induced by paralysis of the left ventricle. Rokitansky thus describes the influence of pericarditis on the heart-tissue: Its muscular substance is paralyzed, being of a dirty brown or yellow color, flabby, and easily torn—a condition which speedily leads to passive dilatation of the heart, general cachexia, and dropsy.

The first stage of pericarditis, before exudation, is not discoverable by physical signs (STOKES). This period rarely lasts longer than thirty-six hours. To Dr. Stokes, in 1833, the science of Medicine owes the description of the most characteristic physical sign of pericarditis. He established that in pericarditis a double *frottement* or friction-sound exists. Others also about the same time had noticed such a sound, and had correctly interpreted its meaning. Bouillaud and Collin, on the Continent, and Drs. Watson, Latham, and Mayne, in this country, had all, independently of each other, perceived and appreciated the symptom—a circumstance which, as Dr. Watson remarks justly, gives to the symptom a greater amount of importance. This sound closely resembles a rasping murmur. It has been named a “*to and fro*” sound by Dr. Watson. It is apt to disappear gradually from below upwards with the increase of



effusion, and to return with its decrease. It may disappear from the apex to the base with the progressive formation of firm adhesions. It is usually limited to the region of the heart, but changes its character and its seat from day to day. It is sometimes remarkably modified by local bleeding, passing from a loud rough sound to a soft bellows murmur—most rough and intense during inspiration. The hand applied over the cardiac region will sometimes detect a rubbing sensation, which ceases with the cessation of pericarditis.

In diagnosis, however, it is necessary to bear in mind that *friction-sound is not necessarily present in pericarditis*. During the progress of a case, friction-sound may be absent or it may be present for long periods—its presence or its absence bearing no appreciable relation to the intensity of the disease. The amount of fluid effusions has much to do with this. A really considerable effusion of fluid generally at first muffles, then renders barely audible, and finally removes the sound, the friction-sound becoming indistinct as the heart's sounds are gradually extinguished. Like the heart's sounds, the friction-sound continues audible longest, and is recovered soonest towards the base. When the lymph is rapidly condensed into firm granulations, and the parts of those granulations most exposed to attrition have become polished and rubbed away, so that the points gradually receding from each other present fewer and fewer points of contact, then the friction-sound may subside, although no further effusion of fluid takes place (W. T. GAIRDNER).

Again, *the presence of friction-sound is not necessarily a proof of the existence of pericarditis*. There are permanent exocardial murmurs, probably associated with the well-known "*milk spots*" on the anterior aspect of the heart, as pointed out by Professor Gairdner. When it is remembered how frequently slight, short, and ill-defined murmurs, especially with the first sound of the heart, may be discovered, in persons otherwise healthy, about the left border of the sternum, at the level of the third and fourth intercostal spaces, or lower, the existence of "*milk spots*" may be their cause. *It is only when the murmur arises for the first time under observation, or when it accurately coincides with the development of symptoms, or where it corroborates and explains the symptoms, and the other physical signs already existing, in such a manner as to leave no doubt of its nature, that we are justified in assuming that a friction-murmur over the heart is pathognomonic of acute pericarditis.*

The *white* or *milk spot* is so often found in hearts which in other respects are perfectly healthy, that many pathologists doubt their morbid nature (BAILLIE, SEMMERING, HODGKIN, J. REID). The anterior surface of the right ventricle is their most frequent seat. Occasionally these spots are observed upon the surface of the left ventricle, or upon the auricle, or upon the prominences of the coronary vessels. Their size varies from a fourpenny piece to a crown or larger. They are more common in adult than in early life; but they have been observed in the infant under three months old. They increase after the age of eighteen, apparently progressively with age. About 33 per cent. post-mortem examinations from the

ages of eighteen to thirty-nine show such *white spots*; and about 71 per cent. from ages between forty and eighty. Baillie, Laennec, Louis, and Todd, state that these opaque patches can easily be dissected or peeled off from the visceral layer of the pericardium, leaving the membrane entire. Corvisart, on the other hand, says they are seated on the under surface of the membrane. There seems, however, to be two forms of the white spot—namely, (1.) A superficial, which may be peeled off; and (2.) A deeper spot, which cannot be so detached (BIZOT, PAGET, KING, HODGKIN).

A great difference of opinion prevails as to the cause of these spots. Some may, no doubt, be due to previous pericarditis; but the weight of evidence seems to be in favor of attrition being their cause (HODGKIN, KING, JENNER, WILKS). The circumstances which seem to favor the development of this white spot are those which would increase the rubbing of the part against the pericardium applied to the anterior wall of the thorax. Those circumstances are—(1.) Dilated heart; (2.) Impeded action of the lungs, (*a.*) from those diseases which, leading to augmented volume of the lungs, tend to press the heart forwards; and (*b.*) from continuous pressure upon the chest in an antero-posterior direction, commenced at an early age, before the epiphysis of the ribs and the pieces of the sternum are fully grown and united—as in young soldiers who, during great exertions, carry a loaded pack and wear cross-belts.

These pericardial or exocardial friction-sounds or murmurs may be, and are often mistaken for endocardial murmurs (TAYLOR, STOKES, GRAVES, SKODA, SIBSON), and the distinction between exocardial and endocardial murmurs is not always easy, nor is it to be effected by the ear alone. The following distinguishing signs are condensed from Dr. Sibson's interesting review of the works of Stokes and Bellingham on diseases of the heart (*Medico-Chirurgical Review* for 1854).

The exocardial may be distinguished from the endocardial sounds by the nature and nearness of the exocardial sound—by its existence with diastole as well as systole—its limitation to the region of the heart—its non-existence over the great vessels—its variations over different parts of the heart—its rapid and frequent change in character, or its disappearance from day to day—its want of correspondence with the rhythm of the heart, while it seems to follow upon its movements (SKODA), or to precede and follow the impulse (WUNDERLICH)—its coexistence with tactile vibration, and where there is much effusion, with an extensive cone-shaped region of cardiac dulness. The apex of this dull region points to the top of the sternum, its broad base extending downwards to the right, and far to the left of the epigastrium. In addition to these signs, which, when they exist, serve to establish the existence of pericarditis, and separate pericardial from endocardial murmurs, we have other very characteristic signs by which to distinguish every case of pericarditis, especially in the earlier stages. By the aid of pressure, at first demonstrated by Dr. F. Sibson, applied gently over the region of the heart, we have a test decisive as to the cause of these sounds, when we are in doubt as to whether it is endocardial or exocardial.

If the noise is that of a valve-murmur, pressure from without does not increase or modify it, except in some anæmic persons, over the aorta. If, on the other hand, it is that of a friction-murmur, soft and bellows-like, of exocardial origin, pressure intensifies the noise, and converts the sound into a rustle or rub. By pressing gently on the costal cartilage or end of the sternum with the stethoscope, the intermediate fluid is displaced, and the roughened surfaces are brought into contact. This method of diagnosis is most valuable, especially in the early stages, when it is of real importance to arrive at a correct diagnosis (*Provincial Med. Trans.*, vol. xii, p. 540).

“A really refined and intellectual diagnosis,” and not one founded on the mere *aural* recognition of acoustic characters, is necessary to guard against serious mistakes. In the majority of cases where the friction-sound of pericarditis is recognized, it is known to be such by the circumstances in which it occurs rather than by the mere character of the sound itself (STOKES, WALSH, FULLER, GAIRDNER). Increased extent of dulness in percussion, and marked prominence over the cardiac region, are also two characteristic signs.

When extensive effusion takes place, the heart is pushed upwards to the fourth, third, or second intercostal spaces; consequently the seat of the heart's impulse, of the rubbing sounds, and of tactile vibrations, all are correspondingly raised (SIBSON, LATHAM, WALSH).

A valuable distinctive sign of pericardial effusion, when contrasted with pleuritic effusion, is, that when the left side is dull in front and resonant behind, it is a pericardial, and not a pleuritic effusion (STOKES). Any large increase of fluid at once betrays itself, especially in the young, by the protrusion of the left cartilages and ribs, the widening of their interspaces, prominence of the ensiform cartilage, and, in some extreme cases, by an epigastric fulness or even tumor. When the fluid increases, the pulse becomes feebler, and more disposed to falter and to flutter. It becomes irregular and excited; and often the patient is so fixed in one position, that he fears to move, lest he may aggravate by exertion the dyspnoea and action of the heart from which he suffers so intensely. The jugular veins not unfrequently become distended, and this distension does not lessen during inspiration when the effusion is great. A significant sign is thus furnished of the greatness of the obstruction which exists to the thoracic circulation. Edema and great coldness of the extremities are also apt to supervene with such a state of things. When, however, the products of inflammation become solid, and little serum remains, the pericardium, by the opposed serous surfaces, becomes attached to the heart throughout, and the pulse then resumes its force and regularity; and, if the patient survives, this adhesion remains for life.

The occurrence of acute pericarditis apart from vital phenomena or symptoms is not now believed in, and among the most characteristic general symptoms is the occurrence of a dry short cough, not explained by any morbid state of the lung.

**Prognosis.**—While the consequence of pericarditis “is sometimes, though rarely, the speedy extinction of life, in nineteen cases out of twenty the disorder proves fatal at a remote period, destroying

the subject of it more slowly, indeed, but almost as surely." Such is the opinion of Dr. Watson; and again he writes, "I am certain it is a fertile, but often unsuspected source of chronic disease of the muscular substance of the heart, and of its consequences, asthma, dropsy, sudden death." Sometimes death occurs suddenly when least expected; and the mode of death tends to be by asthenia. A fatal termination may occur, although rarely, in less than forty-eight or thirty hours. In ordinary cases which progress favorably, the disease generally begins to yield in the course of a week or ten days, and sometimes sooner under active treatment.

It is not now, however, generally believed that when the disease terminates by adhesion, dangerous or fatal lesions of the heart tend to supervene, as Dr. Hope believed. It is consistent with the experience of Drs. Stokes, Sibson, Wood, of Philadelphia, W. T. Gairdner, Smith, and other observers, that hypertrophy and dilatation of the heart do not commonly follow on pericardiac adhesion. Dr. Watson, however, with many others, regards adhesion of the pericardium as a suspicious prognostic of future evil—"that other structural changes will soon or slowly develop themselves; and first render life burdensome and full of suffering, and then consign the patient to an earlier grave than might else have awaited him."

Among the patients of a general hospital, from 6 to 8 per cent. die of pericarditis, while a proportion, varying from 3 to 8 per cent., have had the disease at a previous period and recovered from it (W. T. GAIRDNER).

**Causes.**—Pericarditis is generally the local manifestation of a disease which pervades the system. There is scarcely a form of constitutional malady which may not be accompanied by *pericarditis*; but those with which it is most apt to be associated are *rheumatic fever*, *Bright's disease*, *pyæmia*, *pneumonia*, *tubercular vomica*, *pleurisy*, *small-pox*, *scarlatina*, *scurvy*. These diseases are mentioned in the order of the frequency with which they give rise to pericarditis, as consistent with the observations of Drs. Taylor, T. K. Chambers, A. W. Barclay, and Dr. Fuller. In cases of *acute rheumatism*, pericarditis occurs once in every 5.97 cases (FULLER).

The muscular substance of the heart sometimes suffers in cases of pyæmia consequent on the vitiated state of the arterial blood, and pericarditis has been known to arise in this way (Kirkes, *Med. Times and Gazette*, October 25, 1862, p. 432). Such cases have usually been described as cases of carditis, or acute inflammation of the muscular substance of the heart depositing pus. Dr. Kirkes, however, believes that the muscular tissue of the heart is not primarily at fault, but, in common with other parts of the body, it is by accident the seat of secondary formations in consequence of contaminated blood; and the attendant pericarditis is an accident of the proximity of some of the deposits to the surface, and it may be of their bursting into the cavity, just as an ulcer in the wall of the intestines may burst into the *peritoneum* and cause *peritonitis*. It has also occurred by extension of other diseases from neighboring organs—*e. g.*, cancer of œsophagus, and the like (J. W. BEGBIE). (For more details regarding pericarditis, the student is recom-



mended to study the excellent treatises of Dr. Fuller *On Rheumatism* and *On Diseases of the Chest*; also Walshe and Stokes *On Diseases of the Heart*).

**Treatment.**—The results of recorded treatment are unsatisfactory in the highest degree. Dr. Watson thus writes, that “in a large proportion of the cases, whether they be treated well or ill, or not treated at all, the patients will seem to recover.” Although blood-letting and calomel have hitherto been regarded as two most efficient agents in accomplishing the indications of cure, yet Taylor’s cases show that pericarditis may come on during salivation. Dr. Parkes informs me that he has seen this occur two or three times. It is, then, very doubtful if calomel ever does any good in pericarditis, notwithstanding that its use, so as to affect the gums, is advised by Watson, Graves, Stokes, and Fuller. Professor W. T. Gairdner, Drs. John Taylor, of Huddersfield (*Med. Times and Gazette*, 1849), and J. Risdon Bennett, of London (*Lancet*, December 6, 1851), are all unfavorable to the use of mercury. In all the *constitutional diseases* in which pericarditis occurs as a complication, mercury is certainly contraindicated. In rheumatism and in Bright’s disease, which furnish by far the larger number of cases of pericarditis, mercury is decidedly objectionable; and it is now well known to be productive of most injurious consequences in Bright’s disease. Its use tends to weaken the heart and the system at large, and thus proves a source of additional irritation to the cardiac tissue, by undue reaction on the system.

Dr. Fuller, however, gives his opinion very strongly in favor of mercury as a remedy in the acute and sthenic form of pericarditis, as it presents itself in persons of a strong and healthy constitution. “No such case,” he says, “can be treated safely without its administration.” Such idiopathic cases are indeed rare, the causes which induce pericarditis being for the most part constitutional.

“A single bleeding from the arm,” writes Dr. Stokes, “appears on the whole justifiable, but its repetition will be a matter for careful consideration.” It is a remedy which has been clearly shown by the late Dr. Taylor to shorten the duration of pericarditis, and to do so the more effectually the earlier it is performed (*Medical Times and Gazette*, 1851). The treatment by bleeding may also be further carried out by leeches over the præcordial region, or by cupping there. In cases of rheumatism, Drs. Stokes and Gairdner hold that “it cannot be too strongly impressed upon the mind that, valuable as the discovery of the signs of an inflamed pericardium may be, it is not for these alone that he is to look, but rather for the indications of excitement of the heart, whether attended or not by the signs of exocardial or endocardial disease.” These indications may demand local depletion, “even though no friction-sound or valvular murmur whatever be present.” Dr. Watson gives a most judicious opinion when he says,—“I think there is a peculiar risk in frequently bleeding to syncope in this affection.” Blood may be drawn from the arm till some effect upon the pulse is produced; but Dr. Watson gives the preference to repeated relays of leeches over the præcordial region, or to free cupping. Dr.



W. T. Gairdner advises that when the pain is very marked, where it has strongly the characters of angina, leeches are to be applied in moderate numbers. From *four* to *six* leeches so applied, followed by fomentations, very commonly relieves the pain, and rapid improvement follows.

To judge as to the repetition of bloodletting, the force of the heart must be observed, as indicated by the pulse at the wrist, by the actual strength of its impulse, and by the character especially of the first sound. If the impulse continues vigorous, and the first sound is undiminished, bloodletting may be repeated by the further *local* abstraction of blood; but if the impulse has manifestly declined in force, while the first sound is lessened, great caution is required in the further abstraction of blood. Fomentations, sometimes plain and sometimes medicated with opium, friction with camphorated and ioduretted liniments, and, in obstinate cases, the use of blisters, are the chief local remedies to be relied on besides leeches.

In the second stage of the disease, when liquid effusion distends the pericardium, some reliance may be put in treatment by a blister of a large size over the præcordia.

“Stimulants,” writes Dr. Stokes, “are often imperatively called for. The weakened heart requires to be supported and invigorated. This may be effected by the use of wine, and by the use of opium.” If depletion has been excessive—if signs of muscular weakness supervene—if there be evidence that the heart, previous to the attack, was in a weakened state—if a tendency to collapse or to a typhoid state manifests itself—“we must give wine, quite irrespective of the physical conditions of the heart” (STOKES).

Opium, in doses of one grain (*i. e.*, stimulant doses) every third hour, seems “to expend itself solely on the disease,” and its beneficial effects are seen to result when it does not produce contraction of the pupil, nor headache, hot skin, furred tongue, nor constipation (CORRIGAN, STOKES, GRAVES, SIBSON). Battley’s solution is perhaps the most useful preparation.

There are two more important circumstances to be attended to, as stated by Dr. Sibson—namely, that our treatment of pericarditis must depend upon the stage of the disease in which it is first discovered to exist; and it is important to know that pericarditis from acute rheumatism calls for a totally different line of treatment from that associated with Bright’s disease, or diffused inflammation of a low type.

In the rheumatic form, colchicum, in the form of a draught, and the administration of alkaline remedies, are indicated by the constitutional state.

*Paracentesis* is recommended by Dr. Sibson in all those cases in which the effusion is so great as to cause alarming distress, orthopnoea, obstruction to the venous circulation, and serious interference with the heart’s action. In such cases a fine exploring trocar and canula is to be introduced (not plunged), so as to make a valvular opening below the heart, either to the left of the xiphoid cartilage, or through the fifth intercostal space, close to its anterior extremity,

and the fluid drawn off by means of a syringe (Sibson in *Med.-Chir. Review*, July, 1854).

The general principles of safe treatment are summed up by Dr. W. T. Gairdner as follows: (1.) To make large allowance for the insignificant and spontaneously healing class of cases revealed more by physical signs than by symptoms, and to regard them as demanding little active treatment; (2.) To consider rheumatic pericarditis in general as a disease susceptible, to a great extent, of cure under palliative local remedies and fitting constitutional treatment; (3.) To hold the general treatment as subordinate to the constitutional treatment of the disease with which the pericarditis is associated.

#### DISEASES OF THE ENDOCARDIUM—ENDOCARDITIS.

LATIN Eq., *Endocarditis*; FRENCH Eq., *Endocardite*; GERMAN Eq., *Endocarditis*; ITALIAN Eq., *Endocarditide*.

**Definition.**—*An inflammation of the lining serous membrane, or endocardium, covering the valves and lining the chambers of the heart.*

**Pathology and Morbid Anatomy.**—By a species of preference the coverings of the orifices and valvular structures of the heart are by far the most frequent seat of lesion in the internal inflammation of that organ. The frequency with which these parts suffer may in some measure be explained, if their minute anatomical and histological relations are connected with the morbid states of similar tissue. The peculiarity of the minute structure of these parts, as influencing the arrangement assumed in the first instance by morbid deposits, seems to have been first definitely stated and illustrated by Dr. Watson (Lect. lxi, p. 275, 3d edit.). There is found to exist inclosed between the reduplications of the endocardium a quantity of fibrous tissue. An increase in its amount takes place at the centre of each aortic valve, forming the *corpora Arantii*, and at each of the extremities or angles of the segments. The minute exudations which are formed as the result of the inflammatory process in the endocardium may be seen to arrange themselves in double festoons, suspended as if from the *corpus Arantii*, often in a row, like a string of beads, along the line of union of the thick portion of the valve, with the inner convex margin of its two thinner crescentic portions. The repeated attrition of the opposed surfaces of the valves pushes aside the exudation as fast as it is deposited, and while yet plastic, from the thin crescentic portions of each valve, and so heaps it up along those boundary lines of contact; “just as a thin layer of butter on a board would be displaced, and heaped up in a little curvilinear ridge, by the pressure of one’s thumb.”

While, doubtless, the structure of the serous membrane is the same throughout the heart, yet its mixture with white fibrous and elastic tissue, in great abundance at the valvular reduplications, renders it prone to disease, especially in those constitutional states of the system in which the fibrous textures are more particularly involved, as in rheumatism. These are the parts also on which the great tear and wear on the action of the heart is expended, and

thus they are probably the first to suffer, owing to the mutual friction of the valvular edges upon each other.

Diffuse inflammation of the endocardial membrane has been known to follow the application of a ligature round an artery. The morbid appearances of its inflammation are similar to those in other serous textures—namely, a silvery opacity, and more or less thickening of its tissue. Inflammatory lymph is often found strongly adherent to the valves, as already described, and forming fringe-like or fibrinous warty growths, or excrescences, as they are termed. By its agency the segments or lappets of the valves become variously distorted in shape, or soldered together, and insufficient to perform their functions. An orifice, naturally large, may thus be reduced to the condition of a mere slit, or to the diameter of a goosequill. One segment of the aortic valve may, for example, be turned up and bound to the aorta, or it may be turned down and bound to the inner surface of the heart, or it may be curled up like a shell. The lappets of the mitral valve may be similarly altered.

The prolonged existence of the inflammatory state ultimately thickens and hardens, by interstitial deposit, the tissue inclosed between the folds of the serous membrane constituting the valves, so that their action is much impaired. These changes may be limited to the fibrous zone which forms the base of the valves, surrounding the aortic orifice with a sort of collar, contracting its diameter, as well as impeding the play of the valves. In other cases the thickening may affect the free edge, or the central portion of the valve. The most remarkable circumstance, however, connected with chronic adhesive inflammation of the left side of the heart, is the excessive tendency which the valves have beyond all other serous tissues to become cartilaginous or ossified. This transformation commences in the substance of the serous tissue, but more commonly in the tissue connecting the duplicature of the valvular fold. This ossific deposition is not necessarily preceded by a cartilaginous formation, but is most frequently an original abnormal secretion, often containing a good deal of earthy matter. In all the instances in which I have examined such deposits, they did not exhibit the histological appearances of true bone-tissue. It is deposited in various forms: sometimes in layers, at others in points, and at others in large masses, in knobs or pyramids, occasionally acquiring a size as large as a pigeon's egg. Sometimes the tendons, or the *chordæ tendineæ* attached to the mitral valve, participate in these indurations, and Corvisart met with one entirely ossified. The irritation of these deposits often leads to their destruction, and the whole exudation softening and breaking down, may mingle with the current of the blood, and produce results, to be noticed, of a most serious description.

Dr. Latham and Dr. Hope were of opinion that endocarditis is more frequent than pericarditis. Dr. Stokes, Dr. Sibson, and others entertain a different opinion, consistent with the evidence of post-mortem inquiries, as recorded by Drs. T. K. Chambers, A. W. Barclay, and Taylor.

The tendency of endocarditis is—

1. To produce those affections of the heart which are also described respectively as “*valvular disease of the heart*”—“*hypertrophy*,” and “*dilatation*”—morbid conditions more or less simple or combined.

2. Associated as it often is with pericarditis, and acknowledging rheumatism as a most frequent exciting cause, we have the *muscular* substance of the heart itself sometimes affected, constituting what Dr. Watson terms “*rheumatic carditis*.”

**Symptoms and Results of Endocarditis.**—A more extensive, forcible, and abrupt impulse of the heart than natural, combined with endocardial murmurs, of a soft low pitch, tone, and blowing sound, combined with a febrile state of the system, and cardiac uneasiness, suggest the probability of endocarditis. A careful study of the development, order of occurrence, and combination of the general symptoms and physical signs, can alone convert that probability into a certainty.

1. **General Symptoms.**—The patient is observed to prefer to lie on his back (dorsal decubitus), and he may perhaps incline to toss about with his arms. Pyrexia may prevail of a specific kind, as when rheumatism, Bright’s disease, or typhus fever is present, or it may be idiopathic inflammatory fever, associated with the endocarditis. So long as the cardiac orifices are not seriously obstructed, and no obstruction exists in the lungs from pneumonia or bronchitis, no special sensation of dyspnoea is complained of. The pulse ranges in frequency between 80 and 120, and it has been stated by Dr. Taylor even to lose in frequency at the outset of the affection. More or less headache may prevail.

2. **Local Symptoms.**—Discomfort and uneasiness at the heart are most common symptoms, and more or less palpitation may be present.

The extent and power of the impulse of the heart ought now to be examined carefully and repeatedly, and the conditions which tend to subdue or to aggravate these phenomena ought, if possible, to be ascertained. The areas of the heart’s dulness, both superficial and deep-seated, undergo increase (BOUILLAUD, WALSHE).

The murmurs which accompany endocarditis purely acute are thus arranged in the order of their frequency by Dr. Walshe: (1.) Aortic obstructive; (2.) Mitral regurgitant; (3.) Aortic regurgitant; (4.) Aortic obstructive and mitral regurgitant together. (The student is referred back to page 577, *et seq.*, for the account of the sites of maximum intensity where these murmurs may be listened to.)

The comparative frequency of aortic and mitral valve disease has been determined with more accuracy by the results of the combined observations of Drs. Barclay, Chambers, and Ormerod, as tabulated by Dr. Sibson. From these records it is seen that the *mitral valves* are more subject to disease than the *aortic*; that the disease is often limited to one set of valves, but that it is more often common to both valves than limited to either. When associated with acute rheumatism, the disease affects both valves in the greater number

of cases; and the *mitral* more frequently than the *aortic*. In the young, who are subject to acute rheumatism, disease of the *mitral* valve, and in the old, who are subject to atheroma, disease of the *aortic* valve predominates. In the more severe cases in which the valve disease is itself the cause of death, the *mitral* valve disease is shown to be the most prone to go on to a fatal issue (*Med.-Chir. Review*, Oct., 1854, p. 431).

As there is no difference in character between the murmur of endocarditis and that which attends established valvular disease, it is necessary, in order to appreciate the existence of endocarditis more certainly, that the *murmur should be developed under observation at the early period of an acute attack* (WALSHE); and if a *mitral* or *aortic* murmur supervene while a case of acute rheumatism is being watched, especially if there be congestion and an expression of anxiety in the face, with distress in the region of the heart, *not caused by pericarditis*, there is a strong probability of endocarditis (SIBSON). But the symptoms are often exceedingly insidious in their origin and progress, and the disease is rarely simple, being generally combined with pericarditis; and, moreover, as the *general* constitutional symptoms of these two diseases do not differ, the detection of endocarditis, *per se*, is one of the most difficult in practice. Like pericarditis, it is often latent, as in rheumatic fever, and the practitioner is often surprised by his patient showing symptoms of valvular disease after an apparently perfect recovery from fever (STOKES). A murmur, *per se*, is no sufficient evidence of endocarditis. Stokes, Sibson, and Graves have each of them recorded cases where *mitral*, and still more often *aortic* murmurs have been generated, when no valvular disease existed,—in cases of fever especially, in a case of fatty degeneration of the heart, and in a case of pericardial adhesion. These murmurs have also been recorded to exist during life, in cases in which no trace of valve disease was observed after death, by Drs. Barclay, Markham, Chambers, and W. T. Gairdner (Sibson, in *Med.-Chir. Review*, Oct., 1854).

As in pericarditis, it is important to recognize the *friction-sound* pathognomonic of its existence, apart from any endocardial murmur with which it might be confounded; so in endocarditis it is, if possible, still more important to detect endocardial murmurs when masked by pericarditis, for the grazing sounds of the latter disease may altogether mask those of the valve murmurs. The principles on which the diagnosis is to be effected are involved in the facts that friction-sounds of pericarditis are limited to the heart's region (STOKES); while, as Dr. Walshe so clearly describes, the sounds of the heart, and the murmurs which attend the lesions of its valves, are propagated in certain determinate directions; and while they are heard in maximum intensity at certain points, more or less defined, they may be detected by following the line of propagation at points beyond the mere limits or region of the heart itself. Upon these grounds data are furnished by which to distinguish the murmurs of endocarditis. (See *ante*, p. 577, *et seq.*)

For this purpose the murmurs of endocarditis must be looked for in suspicious cases from day to day *beyond the region of the heart*;



and if a systolic mitral murmur is heard extending an inch and a half beyond the nipple, it is most probably due to mitral regurgitation (SIBSON). The detection of an aortic murmur with pericarditis is much more difficult, because the *friction-sound*, *frottement*, or “to and fro” sound, often mounts to the top of the sternum. The aortic murmurs are therefore to be listened to in the line of the natural propagations of the aortic sounds; and if an aortic murmur exists, it can only be distinguished in the neck, the best point for examination being just above the sternum, a little to the right, over the *innominata*. If, after listening to the first sound, the second sound be observed to follow clearly and distinctly, the chances are that there is no affection of the aortic valves, even if there be a loud systolic murmur. If, however, the second sound be indistinct, inaudible, or prolonged, or be replaced by a diastolic murmur, aortic endocarditis may be suspected or detected (SIBSON). (The student is requested to contrast p. 577, *et seq.*, with these statements.)

As far as the immediate practical value of the information is concerned, it seems to be really unimportant where the exact seat of the murmur is. It is of no practical importance, for example, in the first instance, whether the murmur proceed from a “mitral, a tricuspid, or a semilunar valve, or whether it may be due to a contraction, or a dilatation, an ossification, a permanent patency, or a warty excrescence.” The practical points to be determined, in the first instance, reduce themselves to two—namely, *First*, Do the murmurs proceed from an organic cause? *Second*, What is the vital and physical condition of the texture of the heart itself with which they are associated?

For subsequent practical purposes the limits of inquiry may also be very much circumscribed—namely, to the recognition of the occurrence of contraction or of dilatation of the orifice, because both of these conditions are attended with a permanently open state.

While, therefore, the occurrence of murmurs, and their nature, and the circumstances under which they are developed, are of the utmost importance to establish the existence of an endocarditis, the condition of the muscular substance of the heart must be the great guide in prognosis and treatment. The vital and mechanical state of the heart's cavities must be ascertained. The action of the heart must be carefully noticed at different times, as to whether its force and vigor is above or below the natural standard—whether it is liable to excitement from slight causes—and whether it tends to regular or irregular action, as regards rhythm or frequency of revolutions. The duration and origin of the disease must be ascertained; and how far the brain, lungs, or liver suffer from the mechanical or vital effects of the lesion (STOKES).

The symptoms, therefore, of acute endocarditis being detected, the immediate treatment of the disease must be proceeded with; and the physician requires also to look before him in anticipation of the results which are likely to ensue if valvular lesion is established, such as hypertrophy and dilatation (which see).

Another class of symptoms and results is apt to be associated

with endocarditis and valvular disease—as when products of inflammation are apt to poison or spoil the blood. Rigors, heat of skin, profuse perspiration recurring irregularly, dull, earthy-yellow discoloration of the skin (not of the conjunctivæ), diarrhœa more or less bilious, pinched, anxious countenance, intense prostration, and muttering delirium, are the symptoms which Dr. Walshe describes as announcing this untoward occurrence. Secondary deposits in the lungs, the liver, or the brain, are the records of its morbid anatomy. But, again, these secondary deposits may not be the direct result of existing endocarditis. Virchow, Kirkes, Simpson, and Rokitansky have shown how the fibrinous coagula, which have become permanently attached to the valves, constituting the vegetations upon them, may become worn away superficially, and taken into the blood in fine particles, thus leading to secondary coagula in the capillaries of the spleen and kidneys, to obliteration of these vessels, and in the capillaries of the brain leading to softening, and sometimes to sudden death. The great tendency to the formation of these coagula on the valves, in rheumatic pericarditic attacks, must be specially remembered when it is determined to abstract blood, as the slowness of the heart's action which may ensue greatly favors the tendency to coagulation of the blood and to the deposition of fibrinous deposits on the valves.

**Causes of Endocarditis.**—The inner membrane of the heart (exposed as it is to the action of many morbid poisons, and also to many substances which may be taken up by the absorbents and introduced into the circulation) is not found so frequently diseased as we might expect. Of all substances, however, alcohol has the most striking effects on this tissue. It is not only proved to be absorbed and actually to circulate in the blood, but there are few drunkards the inner membrane of whose heart and large vessels is not more or less diseased; so that alcohol probably acts as a specific poison on the endocardium. The morbid conditions associated with Bright's disease, with rheumatism and gout, and with syphilis, appear to act especially upon this tissue, and many who suffer from these diseases often ultimately die of some form of endocarditis often extending along the lining membrane of the aorta.

**Prognosis.**—So far as the endocarditis is concerned, the immediate prognosis is very similar to that stated under pericarditis; but the future chances of life being prolonged depend upon the lesions which remain permanent. If valvular lesion is fully established, and remains persistent, hypertrophy is certain to follow, and the danger will be the greater in proportion as dilatation of the cavities of the heart predominates over hypertrophy. “Under all circumstances,” writes Dr. Walshe, “dilatation is a most serious disease; and the danger increases directly as the excess of the capacity of the cavities over the thickness of their walls; directly, too, as the softness and flabbiness of the heart's tissue; directly, too, as the general deficiency of tone in the system and impoverishment of the blood. Once dropsy has supervened, life can with difficulty be prolonged by art beyond twelve or eighteen months. Dilatation of indubitable existence is not removable by *treatment*” (Walshe *On*

*Diseases of the Heart and Lungs*, p. 487). If neither aortic lesions, hypertrophy, nor dilatation results, the tissue of the heart itself may be so impaired as to lead to softening, as in typhoid and typhus fevers, scurvy, or purpura. Fatty degeneration of the cardiac tissue is also a result to be apprehended.

The valuable medical reports of St. George's Hospital, London, prepared by Drs. A. W. Barclay and Rogers, contain the following statistics relative to the percentage of mortality *among hospital patients from diseases of the heart* during a period of six years—namely, from *pericarditis*, 84; from *endocarditis*, 9.19; from *hypertrophy*, 60.5; from *dilatation*, 52.1; from *valvular disease*, 24.5.

**Treatment.**—What has been written relative to the treatment of *pericarditis* applies equally to acute *endocarditis*; but the management of cases in which the patients suffer from the valvular lesions and their immediate consequences demands the adoption of various lines of treatment. From whatever cause, it is one of the most intractable diseases with which we are acquainted.

When endocarditis seems lapsing into the chronic stage, Dr. Walshe recommends the use of *iodide of potassium* and *liquor potassæ*, combined with bitter tonics.

#### (CHRONIC) VALVE DISEASE.

**LATIN** Eq., *Morbus valvarum*; **FRENCH** Eq., *Maladies des valvules*; **GERMAN** Eq., *Klappenkrankheiten*; **ITALIAN** Eq., *Malattie delle valvole*.

**Definition.**—*Lesions of the valves of the heart, or of its orifices, coming on for the most part insidiously, and which, persisting, induce obstruction or regurgitation, tending to hypertrophy and dilatation of the heart, with congestion of the pulmonary and systemic capillaries, œdema, anasarca, and dropsy.*

**Pathology.**—The term “chronic valvular disease” employed here is used in order to distinguish the chronic lesions of the valves of the heart from “chronic endocarditis;” for it has been already shown that the valves and orifices of the heart are often damaged by this affection. But chronic valvular disease does not always owe its origin to endocarditis. Such chronic valvular disease is often very insidiously established as a local expression of chronic Bright's disease, or chronic rheumatism, and in gout, as well as in such forms of constitutional derangement of the system as are associated with imperfect nutrition of the body. A very slow deposition of fibrine on the substance of the valves, or degeneration of their structures, is the usual lesion they present. The valves ultimately become thickened, opaque, and puckered, and may be rigid by the presence of atheromatous or calcareous matter. Yielding under pressure, they are apt to become thinner than natural, or they become perforated or cribriform—a condition which may also be referable to congenital deficiency. Sometimes they rupture, and present all the phenomena of ulceration, or their surfaces are irregular and their edges beaded by the deposit of fibrine from the blood.

One segment of a valve may be found adherent to another, and in each of these cases the disease may be of such a nature as to obstruct the onward flow of blood, or to permit regurgitation through the diseased valves, when an obstructive or regurgitant murmur will be the result (see *ante*, under "Cardiac Murmurs"). The orifices of the heart may be the parts diseased, while the valves are sound. In such cases the orifices are usually roughened by calcareous or atheromatous matter, or they may be so much dilated that the valves are insufficient to shut them. The lesions to which valve disease may be referred are of the following varieties: (a.) *Vegetation*; (b.) *Fibroid thickening*; (c.) *Atheromatous and calcareous degeneration*; (d.) *Aneurism*; (e.) *Laceration*; (f.) *Simple dilatation of orifice*; (g.) *Malformations*.

The exact lesion which causes the morbid state of the valves or orifices must generally, however, remain a matter of conjecture; and it is not of so much practical importance to determine the lesion, as it is to determine whether it is of such a nature as to cause obstruction of the flow of blood, or to permit of its regurgitation. One or other of these conditions is indicated by a persistent endocardial murmur; and the persistence of such murmur assuredly points to cardiac hypertrophy and dilatation. Nevertheless, disease at the various orifices operates very differently the one from the other in many particulars, a *résumé* of which is here given from the admirable treatise of Dr. Fuller.

*Aortic obstruction*, one of the most common forms of chronic valvular disease, has little effect in producing engorgement of the pulmonary capillaries, or general systemic congestion and dropsy; but when it induces dilatation of the left ventricle, and so causes the *mitral valve* to become so inefficient as to permit of regurgitation, then it is that pulmonary systemic obstruction begins to appear. Aortic obstruction is the least rapidly fatal form of chronic valvular disease. The character of the pulse is not materially altered; but when the action of the heart is forcible, and the obstruction is rough, excessive eddyng of the blood may be produced, causing a thrill at the base of the heart, and in the track of the aorta and its branches.

The peculiarity seen in the pulse-tracing from the sphygmograph consists in the obliquity of the line of ascent, which marks the greater duration of the ventricular systole, and the gradual entry of the blood into the vessels. In the following two tracings from Dr. Foster these characters are shown; but as the second was taken from an old woman of over eighty, the modifications of senile change are superadded.

FIG. 27.



FIG. 28.





*Mitral obstruction* is comparatively rare. It necessarily induces dilatation of the left auricle. The action of the heart becomes rapid and tumultuous, the lungs become congested, and the right ventricle and pulmonary artery eventually become dilated and hypertrophied. This form of chronic valvular disease induces severe cough, dyspnoea, general distress; and proves rapidly fatal, with congestion and oedema of the lungs, and not unfrequently with pulmonary apoplexy. In mitral obstruction the pulse in the earlier and less developed stages is regular and of low tension; is easily depressed and altered in form by pressure on the spring of the sphygmograph; and the line of ascent is oblique rather than vertical.

*Obstruction of the pulmonary orifice* is a lesion still more rare. It leads to hypertrophy and dilatation of the right ventricle, and ultimately to regurgitation through the tricuspid orifice, with turgescence and pulsation in the large veins of the neck.

*Tricuspid orifice obstruction* is very rare; and leads to hypertrophy and dilatation of the right auricle, with excessive congestion of the venous system, unaccompanied by any visible pulsation in the neck.

*Aortic regurgitation*, from incompetency of the sigmoid valves, is one of the most common forms of chronic valvular diseases. It induces hypertrophy of the left ventricle, but does not produce embarrassment of the pulmonary circulation till dilatation of the left ventricle has reached a point at which the *mitral valve* becomes incompetent, and permits regurgitation. It is accompanied by a peculiar and very characteristic pulse-beat. The prolonged swell imparted to the blood at each systole is not sustained. The waves of blood are short and abrupt; the pulse jerks and leaps, and gives a sensation as if successive balls of blood were being shot suddenly under the finger. These peculiarities are consequences of the regurgitation of blood into the ventricle during its diastole. The traces of aortic patency given by the sphygmograph are marked by abnormally great amplitude. The vertical line of ascent marks the sudden ventricular contraction; and is suddenly terminated by a sharp-pointed process (Fig. 29, Dr. FOSTER). The summit of the pulsation-trace is in many cases very short; but in others it presents a horizontal or curved line, especially if any constriction of the aortic orifice exists, or other cause producing delay of the passage of the blood into the vessels. The second tracing (Fig. 30, Dr. FOSTER) was

FIG. 29

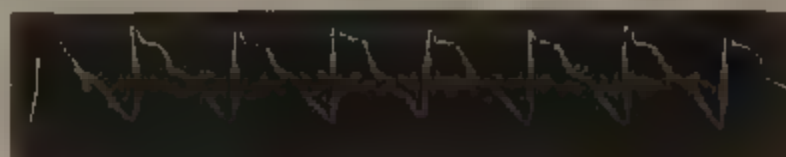
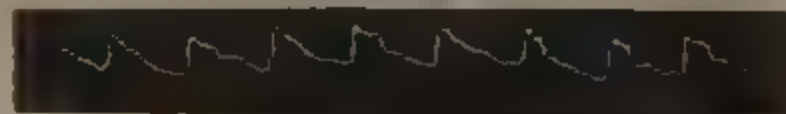


FIG. 30



taken in a case of aortic patency, complicated with senile change in the vessels.



Dr. Sanderson gives a somewhat different explanation of the same lesion and pulse-tracing (*Brit. Med. Journal*, July 20, 1867, p. 40). "Fig. 81 represents the pulse of a middle-aged man, who had acute rheumatism eight years ago, and several times since. When the tracing was taken, he was suffering from extreme orthopnea and præcordial pain. The impulse of the heart was to the left of the mammary line, and occupied a space as large as the palm of one's hand. A loud diastolic *bruit* was heard at the fourth cartilage, and a systolic bellows-sound over the aorta. Posteriorly, there was dulness at both bases, and abundant subcrepitant *râles* in inspiration. There could, therefore, be no doubt as to the nature of the case, which soon terminated fatally. After death, it was found that the aortic valve was so altered that the most copious regurgitation of blood must have taken place during each diastolic period. In this case the pulse exhibits characters which Dr. Sanderson believes are not met with excepting in connection with aortic incompetence. These do not consist, as is often supposed, in the unusual verticality of the expansion; for, as has been already seen, this peculiarity may be produced by merely functional disorder. The distinctive peculiarity consists in the collapse. The tracing shows that the artery becomes completely emptied during the interval between each beat and its successor; so that the diastolic expansion is no longer indicated. The explanation is simple. Immediately after the heart has ceased to contract, the blood injected into the aorta rushes back into the relaxed ventricle; so that, although the arterial equilibrium is for a moment disturbed, it is almost immediately re-established, the excess of pressure in the great arteries being at once relieved. In other words, the elastic force, which is naturally expended in producing what is called the diastolic expansion, is wasted in regurgitation."

FIG. 81.\*



The distinctive features of aortic regurgitation are to be found in the line of descent. There are often one or more extra secondary waves. The first secondary wave occurs earlier than in the normal trace, and the notch which precedes it is often very much exaggerated. But the main characteristic is the suddenness of the fall of the line of descent, and the comparatively small size of the second secondary wave or true dicrotism (Dr. FOSTER).

Mitral regurgitation is the most common of all forms of chronic valvular disease, producing hypertrophy of the left ventricle and dilatation of the left auricle, leading to systolic auricular impulse at the second intercostal space, by admitting of the transmission of the impulse from the ventricle. When excessive, it causes a vibration, a thrill, or a purring

FIG. 82.†



\* Pulse of aortic regurgitation.—H. R., aged 36.

† Dicrotic feeble pulse of mitral regurgitation.

tremor, perceptible on the chest-walls in the region of the heart, but which is not transmitted to any extent along the aorta or great vessels. The pulse-tracing is seen to differ from the normal dotted line principally in its great frequency, and in the depth and amplitude of the diastolic notch. This pulse closely resembles the undulating pulse of typhus. In the one disease the contractile force is *weakened*, in the other it is *ruined*. The effect is the same: the systole is ineffectual (SANDERSON).

When the regurgitation is also combined with obstruction, from contraction of the auriculo-ventricular orifice, the left auricle usually becomes hypertrophied as well as dilated; and this additional lesion always diminishes the irregularity of the pulse characteristic of pure mitral regurgitation; and the tracing from the sphygmograph is as follows (Fig. 33, B. FOSTER):

FIG 33 \*

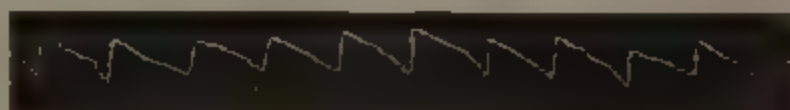
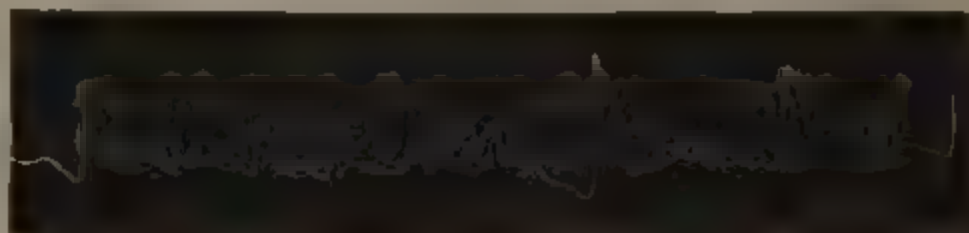


Fig. 34 shows a pulse often seen in rheumatic valvular disease, with large mitral regurgitation. The heart acts very irregularly. At times the ventricle contracts effectually; but at other times the systolic expansion of the arteries is imperfect and abbreviated, in consequence of which the mean arterial tension declines. Whenever this is the case, the pulse assumes a form which is as distinctly

FIG 34.†



dierotic as that of typhus; so much so, indeed, that its double character can be readily recognized by the finger (Dr. SANDERSON).

*Mitral regurgitation* primarily interferes with the circulation through the lungs, producing cough, dyspnoea, and other symptoms of pulmonary congestion; and pulmonary apoplexy is common. The pulse is characteristic. It is irregular in rhythm, and unequal in force and fulness.

*Regurgitation through the pulmonary orifice* is so rare that its effects have scarcely been verified by clinical observation.

*Regurgitation through the tricuspid orifice*, although rare as a primary disease, yet is not uncommon as a consequence of dilatation of the right ventricle. The right ventricle then becomes hyper-

\* Irregular pulse of pure mitral regurgitation (FOSTER).

† Pulse of mitral valve regurgitation (SANDERSON).

trophied, the right auricle dilated, the *venæ cavæ* distended, and there is a strong tendency to congestion of the systemic and cerebral capillary circulation.

**Symptoms.**—The symptoms produced by chronic valvular disease mainly depend on the impediment offered to the pulmonic and systemic capillary circulation. In the more advanced stages all forms of valvular mischief result in a certain amount of capillary engorgement; and therefore all forms of chronic valvular disease have many symptoms in common. Oppression at the chest, breathlessness, speedy exhaustion on exertion, a general sense of lassitude, headache, restless and disturbed sleep, with frequent starting and frightful dreams, cough, palpitation, dropsy, occasional pain in the region of the heart, and sometimes severe *angina*, are amongst the earlier phenomena. The cough is due to pulmonary engorgement, and varies with the amount of *mitral* obstruction or regurgitation. On the other hand, the headache, restlessness, disturbed sleep, and dropsy, are proportioned to the amount of systemic capillary congestion, and vary with the extent of tricuspid obstruction or regurgitation. Albuminuria is not an uncommon occurrence.

The *dyspnœa* of cardiac disease is peculiar and characteristic, as Dr. Hyde Salter has ably shown: It is rather a breathlessness than a difficulty of breathing. It has a panting, gasping character. Oppression, rather than tightness, is complained of, and there is a strangling, choking throat feeling about it. The breathing is always accelerated. The *dyspnœa* is extremely intolerant of movement, or of any exertion whatever, and is often the only circumstance under which *dyspnœa* is felt. As long as the patient remains at rest there may not be the slightest appearance of *dyspnœa*, but the moment any exertion is made the breath is gone. The *dyspnœa* of heart disease is also intolerant of the recumbent posture—hence the name "*orthopnœa*," which signifies "upright breathing"—the patient being compelled to sit erect in order to breathe. In extremely severe cases the patient may not lie down for many days and nights; and should he momentarily fall off into a doze, he is instantly awoke by a sense of impending suffocation, and is in a death-struggle for breath. No suffering can compare with this, and it is not wonderful that the sufferer longs for the sleep of death. "It were indeed a sad story," writes Dr. Ormerod in his admirable Gulstonian lectures, "to tell how patients with disease of the heart die—the tragedies, so to say, of the medical wards of our large hospitals. . . . How some, wrung with pain, have struggled in the week-long agony of death. How some have for days together fixed themselves in the most fantastic postures, the only way in which they could find relief: some leaning forward, resting their hands on a stool, to catch a few minutes' sleep; some on their hands and knees, till the approach of death, blunting their sensations, allowed them to lie down—a sure sign of coming dissolution."

The *dropsy* of cardiac disease is usually a late occurrence. It makes its appearance as *œdema* or *anasarca*, very partial and slight at first—a puffiness merely of the eyelids, or more frequently slight *œdema* of the ankles. By slow degrees it ascends towards the

trunk, and ultimately involves the upper extremities and the face; the scrotum in men and the labia in females becoming enormously swollen. Towards the close, effusion is apt to occur into one or both pleura, but ascites is not common (BELLINGHAM). The first appearance of the dropsy, as Dr. Latham observes, marks an eventful period in the progress of cardiac disease. It indicates that a new law takes effect in the circulation, and gains the mastery over the law of health, which has hitherto been able to retain the watery elements of the blood within the bloodvessels. Now, the serum escapes from the bloodvessels, and accumulates in the areolar tissue of the body.

The forms of valvular disease, in the order of the frequency in which dropsy is met with, are,—(1.) Considerable contraction of the left auriculo-ventricular orifice; (2.) Dilatation of the right auriculo-ventricular orifice, with hypertrophy and dilatation of the right ventricle; (3.) A state of the mitral valve and orifice permitting free regurgitation; (4.) Considerable contraction of the aortic orifice. As a general rule, it supervenes earlier the earlier that general venous congestion ensues. A varicose condition of the bloodvessels in the air-vesicles of the lungs is very soon established, which essentially aggravates the dyspnoea.

**Prognosis.**—It is most unfavorable in cases of mitral and tricuspid regurgitation, and least so in cases of aortic obstruction; and, generally, it may be said that the form of disease which is most rapidly fatal is that which is most rapidly productive of systemic or pulmonic capillary congestion. If the heart be healthy at the date of the occurrence of valvular disease, if the extent of the lesions be not excessive, if the blood be of a normal character, if the viscera be healthy and the secretions free, if the patient's mode of life be regular, temperate, and sedentary, life may be prolonged; but if the heart be hypertrophied and dilated, and if these conditions progress, and if the blood be spanæmic, and the lungs or liver unsound, if the secretions be irregular or defective, or if the patient leads a laborious life, the disease will run a comparatively rapid course, proving fatal probably within two or three years. In either case death is apt to occur suddenly from syncope.

**Treatment** is palliative; the object being to regulate and moderate the action of the heart, controlling the tendency to local congestion, and mitigating or removing the symptoms which result from the cardiac derangement. If the patient be plethoric, the heart's action tumultuous, and its impulse forcible, cupping between the shoulders may afford immediate relief to palpitation and oppression at the chest. Repeated or profuse venesection is dangerous, and is calculated to excite irritability of the heart, to impoverish the blood, and to induce dropsy. Repetitions of bloodletting ought therefore to be effected by the application of a few leeches to the *præcordial* region, and very general relief will be obtained by purgatives, aided by dry cupping, mustard poultices, and turpentine fomentations. When, however, active congestion of the lungs exist, venesection, cupping, blisters, and sinapisms are required.

The action of the heart is markedly influenced by the internal

administration of *veratrum viride*. The dose of *the tincture* is from *five to twenty minims*, which should be gradually increased from the smaller to the greater dose till some obvious effects are produced. If the pulse is reduced, or nausea occurs, no increase of the dose should be made; and if vomiting occur, it should be suspended; and when resumed, the dose should be diminished. *Veratria* is to be given in doses of from *one-sixteenth* of a grain to *half a grain*. When the pulse is sufficiently reduced, the doses should be diminished one-half. *Morphine* or *laudanum*, with brandy, is an antidote for an overdose of this *veratria*, which is an exceedingly powerful remedy as a cardiac sedative, and requires to be used with great caution, the patient being constantly watched. Its depressing effects on the circulation and nervous system are often very striking—a pulse of 75 or 80 being subdued in the course of a few hours to 35 or 40 (*Record of Pharmacy and Therapeutics*, No. v, p. 35, J. C. Braithwaite). *Aconite*, *hyoscyamus* and *digitalis* are also remedies which exert a sedative influence over the heart.

Sleeplessness being one of the most distressing symptoms, opium in some form might be considered advantageous; but, as Dr. Hyde Salter justly observes, “To give sedatives in such a case would be the refinement of cruelty. What keeps this poor man awake is not a want of tendency to sleep, but a condition that makes sleep impossible. Relieve him of his orthopnoea and he would be asleep in ten seconds, and so dead asleep that it would take a great deal to rouse him, like a half-asphyxiated child on whom tracheotomy had just been performed. His great struggle, as it is, is the struggle between sleep and life; with opium thrown into the scale of sleep, the struggle for life would only be so much the harder. In one way, and only in one way, would opium give him ease: the narcotic of opium, added to the narcotic of the carbonic acid already circulating in his veins, might accelerate by some hours, or even days, the final coma, and make him sooner sleep the sleep of death. But the euthanasia that is purchased by anticipating the natural process of death comes very near to homicide, and is an alternative that few would adopt” (*Brit. Med. Journal*, Feb. 8, 1862).

Careful regulation of the patient's mode of life is above all things necessary in chronic valvular disease. Excitement of all kinds must be avoided; and the diet should be light, nutritious, and of moderate quantity.

When anasarca supervenes, the hydragogue cathartics are required to insure copious watery discharges from the bowels. *Gamboge*, *elaterium*, *podophyllin* and *compound jalap powder* should be given on alternate days. Stimulants also may be required, the most suitable being Hollands gin, or whiskey.



## MYOCARDITIS.

LATIN Eq., *Myocarditis*; FRENCH Eq., *Myocardite*; GERMAN Eq., *Myocarditis*; ITALIAN Eq., *Miocarditide*.

**Definition.**—*An inflammation of the muscular structure of the heart, extremely rare as an idiopathic disease.*

**Pathology.**—Cases of inflammation of the muscular substance of the heart from acute rheumatism or pyæmia are not rare; but the cases recorded of other forms of inflammation are too few to enable any satisfactory generalization of their phenomena to be given. Myocarditis is generally a concomitant state, associated with *endocarditis* and with *pericarditis*, and its effects are especially obvious in the strata of fibres nearest the inflamed membranes. As an idiopathic disease, nothing clinically is known concerning it. Its results may be seen after death in the following lesions:

(a.) *Suppuration and Abscess.*—M. Simonet has recorded a case of *suppuration* of the heart, in which the disease appeared to result from rheumatism. The patient, a woman, was brought to the hospital laboring under most tumultuous action of the heart, with a pulse irregular and contracted, her breathing oppressed, and her extremities cold. She was bled, but died in a few hours in a fit of syncope. Several purulent collections were found in the substance of the heart, and especially in the interventricular partition. The internal surfaces of the cavities were red in several places: the muscular structure was of a yellowish hue, softened, and easily torn with the least effort.

Dr. Graves was once consulted by a gentleman, fifty-five years of age, complaining of palpitation, dyspnœa, and finally anasarca. He suffered from severe pain and oppression at the region of the heart. Hypertrophy and dilatation of the ventricles were detected. The patient died suddenly a few weeks afterwards; and besides the hypertrophy and dilatation, an *abscess* was found in the walls of the heart, which contained about two ounces of pus.

Another case in proof of suppurative inflammation taking place in the heart is one that was examined by the late Mr. Stanley. In this instance the vessels were loaded with venous blood, and the muscular fibres were of a very dark color, of a very soft and loose texture, and easily torn by the fingers. On a section of the ventricles, numerous collections of dark-colored pus were seen among the muscular fasciculi. Some of these were seated near to the cavity of the ventricle, while others were more superficial, and had detached the pericardium from the heart. The muscular parietes were softened, and loaded with dark blood.

Suppuration of the muscular substance of the heart and of the coronary artery, in a case of pyæmia, is recorded in the sixth volume of the *Pathological Society's Transactions*, p. 151.

(b.) *Ulceration* of the heart has been occasionally seen, from an abscess in the walls of the heart having opened either into one of its cavities or into that of the pericardium. It has also resulted

from the softening of a cancerous tumor, or from a suppurating tubercle. Cloquet has given the case of a man, aged seventy-nine, subject to frequent syncope, who died suddenly, in whose heart there was an ulceration of the left auricle, through which about two pints of blood had escaped into the pericardium.

(c.) *Rupture and Ramollissement* of the walls of the heart have been occasionally met with. In this affection the heart is flaccid, so that if an incision is made into the ventricles the walls collapse. Its substance tears with great facility. This disease is almost always accompanied by some change in its color, which is sometimes deeper than natural, and at others, according to Laennec, of a yellowish tint, like that of an autumnal leaf—an appearance which does not necessarily occupy the whole thickness of the muscular substance, but often merely the central layers. This degenerescence is sometimes general, but often partial, affecting only the walls of one ventricle of the interventricular partition, or the walls of one auricle. It is from this cause, perhaps, rather than from any other, that patients sometimes die from rupture of the heart.

Examples of rupture of the right side of the heart are more rare than those of the left; or, according to Bouillaud, there are six ruptures of the left side to four of the right side. Rupture of the auricles is perhaps as frequent as that of the ventricles; or, out of the ten cases mentioned, four were ruptures of the right auricle and two of the left auricle. The extent of the rupture, when it takes place in the ventricle, is various. In one case the ventricle was ruptured from its apex to its base, along the sulcus which separates the two ventricles; in another the rupture was from ten to twelve lines; in a third, the base of the ventricles was severed from the aorta, and one of the aortic valves split transversely. It is remarkable, however, that the rupture has seldom been found at the apex, where the walls of the heart have least force and consistency. The number of the ruptures is as various as their seat; thus, out of forty-eight cases collected by Ollivier, eight were multiple. Again, in two cases related by Rostan, there were two ruptures in each case towards the apex of the left ventricle. Morgagni gives one case, and Portal another, in which there were three ruptures in the left ventricle; and Andral met with a third, in which there were five ruptures; but of these, three were superficial, only two opening into the cavity of the left ventricle.

Corvisart is the first who has given examples of another kind of rupture of the heart, and it is that of the *carneæ columnæ*, or tendons of the valves. It is probable that rupture of these parts is owing more frequently to ramollissement, or to induration, than to any other cause. Laennec, however, mentions a case in which it appeared to result from ulceration. In three cases related by Corvisart the rupture followed some violent exertion; and Bertin saw a case in which one of these tendons was ruptured in consequence of a violent fit of coughing. The first symptom in all these cases has been a sudden sense of suffocation, and the patient has in general suddenly died, although in some instances he has survived a few days.

(d.) *Induration* of the walls of the heart is also an occasional le-

sion of this organ. Bouillaud has collected a series of cases in which this change of structure has been observed. In one, the walls of the heart were almost tendinous. In another, the *carneæ columnæ* of the ventricle were so increased in density as to split. In a third, the walls of the right ventricle seemed to be undergoing a cartilaginous transformation; and Broussais has seen them as hard as a cocoanut. Chronic congestion of the heart leads to this condition; and then to defective contractility, leading to dilatation (Jenner, *Med.-Chir. Trans.*, vol. xliii). The more usual mode of induration is ossification—a change which usually begins in the coronary arteries, and frequently stops there; but in some rare cases this ossification extends, so that the walls of the auricles, of the ventricles, or of both, and sometimes also of the cardiac partition, become converted into bone. There are specimens in the museum of St. Thomas's Hospital, and many other hospital museums, which make it remarkable how life could have been continued, looking to the unyielding nature and great extent of the ossification of the walls of the heart and great vessels.

#### HYPERTROPHY.

LATIN Eq., *Hypertrophia*; FRENCH Eq., *Hypertrophie*; GERMAN Eq., *Hypertrophie*; ITALIAN Eq., *Ipertrofia*.

**Definition.**—*An abnormal increase of the muscular substance of the heart.*

**Pathology.**—Although occasionally an idiopathic disease, it is more commonly a secondary affection caused by disease of the valves. The hypertrophy may be general or partial—that is, may affect the whole heart or one side of the heart, or one ventricle, or one auricle, or the ventricle of one side and the auricle of the other, or both ventricles or both auricles, or, indeed, every possible combination of the four cavities. The auricles, however, are much less frequently affected than the ventricles.

The natural thickness of the walls of the left ventricle is in the adult about six and a half lines; but Laennec has seen them, in cases of hypertrophy, to measure an inch and a half, or eighteen lines, in thickness at the base, or triple the healthy standard. This thickness generally diminishes towards the apex, which latter is often natural: but in other cases even the apex is thickened, and instead of two lines it may measure four. The *carneæ columnæ*, and likewise the cardiac partition, are proportionably hypertrophied in these cases.

In hypertrophy of the right ventricle the walls are more uniformly thickened than in hypertrophy of the left ventricle: still, however, the increased thickness is always more marked about the tricuspid valves, and at the origin of the pulmonary artery. The greatest thickness observed has been seldom more than four or five lines, which, taking the natural thickness at two and three-quarter lines, is scarcely a twofold increase. In malformations of the heart, however, it has been found much greater; and both Bertin and

Louis have each seen a case in which the foramen ovale was open, and in which the thickness varied from twelve to sixteen lines. Besides an increase of thickness, the walls of the right ventricle, when hypertrophied, acquire a greater firmness, so that on cutting through the walls they do not collapse.

Hypertrophy of the heart seldom takes place without an alteration in the size and form of the chambers. These may, indeed, be natural, but more commonly they are increased; so that, supposing the chamber of the natural size to hold two ounces, when thus diseased it will often contain the larger portion of a pint. This state of parts has been termed *eccentric* hypertrophy; and admitting the normal heart to weigh from eight to eleven ounces, according to age, the weight in hypertrophy is often double or triple that amount; and Bouillaud speaks of eighteen, twenty, and twenty-two ounces being not uncommon. On the contrary, hypertrophy sometimes takes place *concentrically*, or at the expense of the cavity of the heart, and from this cause the ventricle has been found so reduced in size as to be not larger than an unshelled almond.

An analysis of ninety-six cases, collected from various authors, and tabulated by Dr. Sibson (*Med.-Chir. Review*, October, 1854, pp. 434, 435), shows that by far the larger proportion of cases of valve lesion tend to thicken the walls and enlarge the cavities of the heart; that aortic regurgitation with narrowing of the aperture, and still more without such narrowing, induces active dilatation of the left ventricle, followed consecutively by enlargement of the left auricle and the right ventricle and auricle; that disease of the pulmonic valves causes dilatation of the right cavities; that mitral narrowing with regurgitation leads to enlargement of the left auricle, followed in succession by dilatation of the pulmonary veins, congestion in the lungs, enlargement of the right ventricle and auricle, distension of the venæ cavæ, engorgement of the liver, congestion in the systemic capillaries, and at length, and in nearly one half of the cases, enlargement of the left ventricle itself; that combined disease of the aortic and mitral orifices causes enlargement of the left ventricle, and to a less, but nearly to the same extent, of all the other cavities. Thus there is established a great pathological fact—originally stated by Senac, and confirmed by Morgagni—that *dilatation*, with *hypertrophy*, acknowledge as their cause a force acting *a tergo*, attempting to overcome an obstacle in advance. But there are also other morbid states which, upon the same principle, tend to these results, and are independent of valve disease—namely, *bronchitis*, *emphysema*, and any lung disease in which there is an obstacle to the flow of blood through the lungs. Disease also of the arterial trunks, such as *atheroma*, or morbid states of the blood like *anæmia*, dilatation or narrowing of vessels, whether of the lungs or of the system, may induce dilatation of the right and left cavities of the heart (ROKITANSKY). The retrograde influence of the systemic capillaries, as shown by Dr. Sibson, tends to exercise a similar influence, as shown by the effect of sudden fright and despair, causing rupture of the left ventricle, or by the influence of Bright's disease altering the quality of the blood, tending thereby



to retard its progress through the systemic capillaries. Thus hypertrophy and dilatation ensue from increased resistance to the exit of blood from the cavity of the heart, and from the necessarily increased efforts to expel it, and to propel it onwards. Increased arterial resistance may be connected with a variety of causes. Thus, it may arise either from a contracted state of the capillaries, from diminished elasticity of the arteries, or from narrowing of the aortic valve. Figs. 35 and 36 serve to illustrate these points. An example of the most simple case of resistance to the contraction of the left ventricle—namely, that which occurs in aortic stenosis—is given in Fig. 35. This is the pulse-tracing of a patient in whom the heart, with the adherent pericardium, weighed thirty ounces. The left ventricle was both thickened and dilated, and the aortic

FIG. 35.\*



valve so deformed and beset with vegetations, that the orifice would scarcely admit the tip of the index finger. The other valves were healthy. All the other morbid appearances found were distinctly referable to the cardiac lesion as their cause. The tracing scarcely needs explanation. The second event, which, in the pulse of aortic regurgitation, and indeed in all atonic pulses, is either entirely suppressed or difficult to distinguish, is here extremely well-marked; and the form of that part of the pulse-curve which represents it shows that the systolic expansion of the artery is of nearly equal duration with the diastolic interval (SANDERSON).

The patient whose pulse is represented in Fig. 36 was complaining, at the time when the observation was made, of dyspnoea on the slightest exertion, and precordial pain. He was subject to nocturnal paroxysms of cardiac distress, which, he stated, always came

FIG. 36†



on with pain at the scrobiculus, followed by palpitation and nausea, with violent respiratory efforts. On examining his chest, Dr. Sanderson found that the cardiac dulness extended from the sternum to the mammary line, and that the precordial impulse was diffused and expansive. The systolic sound was prolonged, but no abnormal murmur could be made out. The man died several months afterwards, when it was found

that the heart weighed twenty ounces, and that the left ventricle

\* G. M., aged forty. Pulse of aortic obstruction, with hypertrophy of the left ventricle (SANDERSON)

† W. S., aged fifty-nine. Hypertrophy of the left ventricle without valvular disease.



was both hypertrophied and dilated, *without valvular disease*. (See under "Bronchitis" how collapse of the lung may also produce dilatation without valvular disease.)

In detecting enlargement of the heart and thickening of its walls, the size and force of the heart, ascertained by the hand and by auscultation, furnish the principal data. It is by the extent and power of the impulse that the heart's muscular condition is ascertained; and so long as the muscular condition is sound, the valve disease has but little influence on health (SIBSON and STOKES). As a rule, however, the persistence of valve disease implies an enlarged heart, with an impulse increased in extent and in power. But there are also cases to be guarded from mistake—namely, those where a murmur exists with a preternaturally strong, troublesome, quick and smart impulse, *but limited within a diminished cardiac region*. In such cases such a murmur is of anæmic origin, and the heart is usually lessened rather than enlarged.

**Hypertrophy and Dilatation.**—Hypertrophy of the heart's substance usually occurs under three forms—namely, (1.) *Simple hypertrophy*, when the cavities retain their relative capacities to each other, although the walls are increased in thickness; (2.) *Eccentric hypertrophy*, when the cavities enlarge or dilate, and the muscular walls thicken—commonly known as *hypertrophy with dilatation*; (3.) When the walls thicken at the expense of the capacity of the cavities—known as *concentric hypertrophy*. The existence of this last form of hypertrophy is now disbelieved, except as a congenital malformation. Cruveilhier, and more recently Dr. Budd, have proved that no such form of hypertrophy exists as a result of disease.

Dilatation of the heart implies that the capacity of its cavities is increased disproportionately to the thickness of their walls. It occurs in three forms—namely, (1.) *When dilatation predominates over hypertrophy*; (2.) *Simple dilatation, where the thickness of the walls is normal*; (3.) *Dilatation where the walls of the heart are attenuated, or thinner than normal*.

Practically, therefore, the physician has principally to deal with the diagnosis and treatment of—(1.) *Simple hypertrophy*; (2.) *Hypertrophy with dilatation of one or more of the cavities*; and (3.) *Simple dilatation with or without attenuation of the walls of the cavities*.

The following table (compiled from Dr. Walshe's treatise) exhibits sufficiently the main points in the symptoms of these forms of disease for the purposes of comparative diagnosis :

TABLE CONTRASTING THE MAIN SYMPTOMS OF THE FORMS OF HYPERTROPHY AND DILATATION.

A.—GENERAL PHYSICAL SIGNS.

Simple Hypertrophy.	Symptoms superadded to constitute Hypertrophy with Dilatation.	General Simple Dilatation.
<p>Arching of the præcordial region, with widening and bulging of the left intercostal spaces from the third to the seventh.</p> <p>Impulse increased in extent to the left of the sternum.</p> <p>Maximum impression felt below and about the left nipple.</p> <p>Heart's impulse slow and heaving, as if pressing steadily against an obstacle—in rhythm regular, in force unequal.</p> <p>Superficial and deep-seated dulness augmented in area, but its shape retaining the triangular form.</p> <p><i>First sound</i> is dull, muffled, prolonged, and weakened, almost to extinction, directly over the ventricle.</p> <p><i>Second sound</i> full and clanging, <i>post-systolic silence</i> shortened.</p> <p>Mitral regurgitant murmur, as a clinical fact, exists at one time and disappears at another.</p>	<p>Extent of visible impulse much greater ; pulse may be felt in the back, and its character is less heaving, sharper, and more knocking, than in simple hypertrophy.</p> <p>Point of the apex beat carried downwards and outwards beyond the line of the nipple, so as to reach the seventh interspace.</p> <p>Force increased, so as to shake the head or trunk of the patient, or the bed on which he lies.</p> <p>The dulness tends to assume a square form in place of a triangular one, and may reach from the second interspace to the eighth rib, and from an inch and a half to the right of the sternum to three inches, or even more, outside the vertical line of the nipple. It may be detected in the back.</p> <p>Sounds gain greatly in loudness and extent of transmission, especially if the valves are not thickened.</p> <p>In consequence of the altered direction of the orifice of the aorta to the cavity of the ventricle, a systolic basic murmur may be generated.</p>	<p>No prominence of cardiac region.</p> <p>Apex beat indistinctly visible or actually invisible, the rounded-off form of the heart destroying the apex form.</p> <p>Impulse conveys an undulatory sensation to the hand, and feeble in proportion to the purity of the dilatation.</p> <p>Force of successive beats is unequal.</p> <p>Rhythm irregular, to a slight or to the very highest degree.</p> <p>The hand applied to the region where the impulse is felt does not feel the beat always strike at the same place.</p> <p>Intensity of superficial percussion dulness not increased : and in cases of attenuated resistance is less marked than in health. General areas of dulness widened.</p> <p><i>First sound</i> short, abrupt, and unnaturally clear at the apex and base, appears near the surface, and its maximum point of expression is slightly lowered.</p> <p><i>Second sound</i> not specially affected. Upon the quality of the heart's texture, as regards softness or flabbiness, will depend the extent to which the sounds are transmitted. Intracardiac murmur always regurgitant.</p>

## B.—GENERAL FUNCTIONAL SYMPTOMS.

Simple Hypertrophy.	Symptoms superadded to constitute Hypertrophy with Dilatation.	General Simple Dilatation.
Strength unimpaired. Power of walking or of ascending a hill diminished, on account of dyspnoea induced by the effort. Face florid.	Strength tends to become impaired.  Purpleness and lividity great in proportion to the valvular or pulmonary obstruction.	Strength fails, and the patient is habitually irritable and melancholy.  Lividity and mottling of the face prevails, and of the lower extremities generally, with chilliness of surface. Softly pitting anasarca spreads from the feet to the abdomen, external genitals, thorax, face, and neck. Ascites follows anasarca.
Constipation habitual.		Bowels habitually constipated or alternately relaxed—discharges dark.
Dyspnoea occasional. [Occasionally hæmoptysis.]*	Paroxysmal attacks of dyspnoea.	Dyspnoea, sometimes called cardiac asthma, becomes complete and habitual, with asthmatic paroxysms, in which the cough is dry, harassing, and convulsive. Expectoration serous, sometimes streaked with blood.
Radial pulse full, strong, firm, tense, resisting, and prolonged, without jerk or thrill.	Fulness of pulse continues, but strength and power of resistance lost.	Pulse small and feeble, and abnormally late in time after the ventricular systole. It may be regular, or narrow, feeble, fluttering, and irregular.
Pain rare.	Pain not uncommon.	Palpitation and cardiac uneasiness most distressing.
Rarely, and never rapidly, the direct cause of death.	Indirectly, and more or less rapidly, leads to a fatal issue.	Faintness occurs from time to time, and may lapse into syncope and sudden death.

[**Prognosis.**—Although cardiac hypertrophy, when it has reached a high degree, does not appear to be susceptible of cure, still much may be done to alleviate its symptoms, and, perhaps, arrest further growth. Nor does it seem to necessarily involve the occurrence of secondary and fatal affections. Dr. Da Costa, writing in reference to his army experience, says: I did not see a death from it, directly or indirectly. Yet I think I have seen enlarged hearts decrease. I found the complaint amenable to treatment to a very high degree, and excellent health was enjoyed by those who, after the force of the action was reduced by treatment, lived regularly and did no laborious work (*l. c.* p. 375).]

**Treatment.**—The symptoms of simple hypertrophy may, in the majority of cases, be greatly mitigated by such means as tend to tranquillize the action of the heart. This end may be best accomplished by occasional very moderate cuppings or leeching over the

\* [Dr. Da Costa has noticed that spitting of blood, so rare in cases of cardiac hypertrophy in civil life, is not infrequent amongst soldiers with that disease. In most it makes its first appearance after excessive exercise, as a forced march, or after battle. It may, or may not, be associated with a scorbutic taint.—*U. S. San. Com. Med. Mem.*, p. 872.]

præcordial region. No known drug possesses the power of controlling the growth of the heart. Saline and aloetic purgatives aid the calmative influence of the local abstraction of blood. Diuretics are useful independently of the existence of dropsy; and direct sedatives of the heart's action are indicated throughout, such as *hydrocyanic acid*, *acetate of lead*, *digitalis*, and *belladonna*; but of all medicines of this class Dr. Walshe considers *aconite* the best, in the form of the alcoholic extract, given in doses of *one-eighth of a grain*. In repeating the doses, the effects must be watched, while they relieve the painful sensations and disquietude about the heart.

[Dr. Da Costa says: Nothing answered as well, as universally well, as *aconite*. But I had to learn, to be successful with it, it must be given in a very different manner from what it has hitherto been and still generally is. It must be persevered in for months; my general plan was to administer one or two drops of a good tincture twice or three times daily, and to go on without increasing or decreasing the dose until the impulse of the heart had become decidedly softer; at the same time, usually, the pulse was lessened in frequency. Then the medicine was kept up in varying doses, always watching whether or not it produced the desired effect, or acted too much. And what, fearing its activity as a sedative, I commenced hesitatingly and doubtingly, I soon was taught to use fearlessly and sanguinely. I am certain, that in a large number of cases, thus employed, the remedy prevented the further growth of the heart. I think that in some it lessened the already existing bulk of the organ (*L. c.*, p. 378). The writer can add his confirmatory testimony to *aconite* used in this manner in simple cardiac hypertrophy, along with proper means to lessen the work, and distention, of the heart.]

If anæmia prevails, animal food should be permitted; and the more soluble and less astringent preparations of iron should also be given. Fluids must be taken in small quantities; and alcoholic fluids of all kinds are to be entirely avoided. Months and even years of treatment may be required to produce any impression on the disease.

Like *hypertrophy*, *dilatation* of the heart is not removable by treatment, but judiciously directed remedial measures may render the condition bearable, and even for a time unappreciated by the patient. To improve the tone of the muscle and strengthen the action of the heart, without exciting its irritability, are the objects to be aimed at in the management of the case. The groundwork of medicinal remedies consists in the administration of general tonics in the form of *bitters*, *mineral acids*, and preparations of *iron*. *Belladonna* may be employed to tranquillize undue excitement with greater safety than any other sedative remedy. Sedatives, as a rule, are unsafe, and require the utmost caution in their use. Due action from the bowels must be daily obtained, to accomplish which the aloetic medicines are the best, aided by the gentle action of an occasional mercurial aperient.

The diet should be nourishing without being exciting; and may include animal food, with a moderate allowance of light beer or wine in small quantities to dinner.

When dropsy appears, diuretics yield most relief in the form of *acetate, nitrate, iodide, and bitartrate of potass, nitric ether, compound tincture of iodine, the infusion and spirits of juniper, or gin*, may all be employed in successive changes, and variously combined. Occasional small doses of blue pill and squill, at bedtime, will facilitate their action generally; and so will cupping over the region of the kidneys, if symptoms of congestion of these organs prevail.

Hydragogue cathartics also aid the diuretics in subduing the dropsical effusions, in the form of *elaterium, gamboge, bitartrate of potass, and the compound jalap powder*.

Dr. Walshe, whose mode of treatment has been here described shortly, prescribes the following formula for the administration of *elaterium*:

R. Extract. Elaterii, gr.  $\frac{1}{4}$ —gr.  $\frac{1}{2}$ ; Extract. Creasotonis, gr. j; Extract. Hyoscyam, gr. ij; *misce, fiat pilula*.

#### (CARDIAC) ATROPHY.

LATIN Eq., *Atrophia*; FRENCH Eq., *Atrophie*; GERMAN Eq., *Atrophie*; ITALIAN Eq., *Atrofia*.

**Definition.**—*An abnormal wasting and loss of the muscular substance of the heart.*

**Pathology.**—Care must be taken not to confound this condition with rigid contraction of the substance of the ventricle diminishing its cavity; and, except as a congenital malformation, its idiopathic occurrence is not believed in.

The walls of the heart may be atrophied instead of being hypertrophied, so that this organ has been found to weigh in one case only four ounces two scruples, instead of nine and a half ounces, while the thickness of its parietes was reduced to little more than a thin membrane. This atrophy may be general or partial. In some cases the atrophy takes place without any notable alteration of the capacity of the chambers of the heart. This is termed *simple atrophy of the heart*. More commonly, however, when the walls are thinned, the chambers of the heart are enlarged, and this is termed *eccentric atrophy*. Again, the whole heart may be atrophied and reduced in size, as is often seen in phthisis. Thus, Bouillaud gives the case of a woman, aged sixty-one, whose heart was no bigger than that of a child twelve years old; and Burns gives the case of an adult whose heart did not exceed that of a new-born infant. This form has been termed *concentric atrophy*.

*Dilatation* of the cavities of the heart may exist both when the substance of the heart is hypertrophied and atrophied; but it may also exist when the walls of the heart are of their natural thickness. In any case the dilatation may be partial or general. Partial dilatation of the heart sometimes presents many curious phenomena: thus, the walls of the right ventricle have been seen divided into two distinct parts; or, as Laennec has described it, into a sort of hour-glass contraction.



In other cases this partial dilatation is perfectly aneurismal. Corvisart gives the case of a young negro who died suffocated, and in whom the superior and lateral part of the left ventricle was surmounted by a tumor almost as big as the heart itself. The inner surface of this tumor contained many concentric layers of lymph, exactly similar to those of an aneurismal sac. The cavity of this tumor communicated, by means of a small opening, with that of the ventricle. Laennec mentions two cases in which a tumor of a globular form, and the size of a duck's egg, was situated at the point of the left ventricle, and communicated with the ventricle by an opening an inch in diameter. In these cases the left side of the walls of the sac presented a continuation of the muscular fibres of the heart, while on the right side they appeared formed by the two pericardia. Laennec thinks that these aneurismal tumors are formed by ulceration of the internal walls of the ventricle, as in false aneurism of the arteries; others, that it is owing to a separation of the muscular fibres and the protrusion of the inner pericardium.

*Hydatids or Echinococcus cysts* have been found in the walls of the heart, beneath the inner membrane. Dupuytren found hydatids in the thickness of the right auricle, forming a tumor projecting into the cavity as large as the heart itself. Morgagni found in an old man—who had in no degree suffered from palpitation, syncope, or irregularity of pulse, but had died of acute disease—a cyst the size of a cherry in the walls of the left ventricle. Such parasitic cysts abound in sheep and oxen.

**Symptoms of Myocarditis, Hypertrophy, and Atrophy.**—Few authors have met with a case of myocarditis, unless complicated with pericarditis, and no distinction has hitherto been observed between the symptoms of these two diseases. Corvisart says it is impossible to distinguish between these affections. M. Laennec affords us no assistance in this dilemma, for he considered that no incontestable example of carditis existed; while Bouillaud says he knows of no symptom which is especially characteristic of carditis (FULLER).

The symptoms of *ramollissement* of the heart are,—a feebler impulse, a slower beat, and greater dulness of the sounds of the heart. Patients suffering from this affection are usually hypochondriacal, liable to palpitation on the least exertion, and often die from the ventricle rupturing.

The symptoms of *induration* of the heart are,—a stronger impulse and louder sound than usual. This class of patients is greatly subject to *angina pectoris*.

The symptoms of *hypertrophy* of the heart are local and general. The local symptoms are,—a more powerful impulsion, a wider range of action, and some change in the sounds of the heart. There is also a greater extent of dulness of sound in the cardiac region, and sometimes a bulging out of the left side.

The increased impulsion in hypertrophy of the heart is in proportion to the greater thickening of the walls. Thus, in slight cases, it is only sensible to the hand, while in others the heart “knocks against the ribs,” and even raises the head of the auscultator. This greater impulse not only often causes a vibration of the *præcordial*

region, but even shakes the whole of the chest. Besides being sensible to the touch, the abnormal action of the heart in these cases is often sensible to sight, each contraction agitating the patient's dress, and sometimes even moving the bed-clothes. The point of the heart deviates more to the left, and its motions may be sometimes traced from the second or third rib as low as the sixth or seventh intercostal space.

The increased thickness of the walls of the heart is evidently unfavorable to the transmission of sound; and in simple hypertrophy without enlargement of the cavity, the natural sounds will be duller than in the normal state; and if the hypertrophy be concentric, or with smaller cavities, the natural sounds will be scarcely heard. When, however, the cavities are enlarged, as in eccentric hypertrophy, the sounds are often clear, full, and even much louder than natural.

In hypertrophy of the left ventricle the impulse is stronger immediately under the inferior portion of the sternum than between the fifth and sixth ribs. Lavoisi laid it down as a sign of hypertrophy of the right ventricle that there is swelling of the jugular veins, which pulsate synchronously with the carotids. Corvisart repudiated this symptom; but Laennec found it in every case of hypertrophy of the right ventricle. In general, this pulsation is limited to the inferior parts of the jugular veins, but in other instances it has been seen to extend to the superficial veins of the arm. He regards this symptom, therefore, as one of the best diagnostic signs of hypertrophy of the right ventricle.

In estimating the general symptoms of hypertrophy of the heart, our knowledge of the influence of the left ventricle over the arteries would lead the inference, *a priori*, to softening of the brain; that one of the effects would be a disposition to congestion and to hemorrhage; and that apoplexy, hæmoptysis, and hemorrhage from the bowels would often result. It appears, according to Bouillaud, that out of fifty-four cases of hypertrophy of the heart, eleven, or one-fifth, were attacked by cerebral hemorrhage, or ramollissement of the central ganglia of the brain. Many suffer from pulmonary hemorrhage, while a few suffer from hemorrhage from the bowels. Indeed, on opening bodies that have died of this disease of the heart, the abdominal viscera and mesenteric veins are found loaded with blood. The conjunction of hypertrophied heart is very common in Bright's disease. Besides these concomitants, a pouchy or otherwise diseased state of the aorta often coexists with hypertrophied heart—the diseased aorta being caused by the abnormal power of the heart; or the hypertrophy of the heart results from a supplemental force being necessary to compensate the functional incapacity of a diseased aorta. Many persons affected with hypertrophy of the heart suffer severely from *angina pectoris*, with palpitation.

The symptoms of *atrophy* of the heart are also local and general. The local symptoms are,—a feeble impulse of the heart, while its sounds are louder, clearer, and more distinct than in health, the intensity of sound being greater in proportion to the atrophied state of the walls, combined with increase of size of the chambers of

the heart. The general symptoms are,—slowness of the pulse, occasional palpitation, difficulty of breathing, and tendency to dropsy.

**Diagnosis.**—The differential diagnostic symptoms of myocarditis, of ramollissement, and of induration of the heart, are imperfectly known. On the contrary, the symptoms of hypertrophy of the heart, and of enlargement of its chambers, are so striking that it is impossible to mistake them; but it should be remembered that they are often latent, unless aroused by some mental emotion or sharp exercise—a fact necessary to be remembered in the examination of recruits, or of lives for insurance offices.

**Prognosis.**—It is probable that diffuse inflammation of the substance of the heart may exist, and may be recovered from; but if any morbid product forms in its substance, the prognosis is eventually an unfavorable one. Ramollissement, as well as induration of the heart, from the tendency to rupture in the one case and to ossification in the other, if they can be determined to exist, are always of grave prognosis, although the patient, perhaps, may survive many years. Hypertrophy, atrophy, or dilatation of the heart, are perhaps compatible with health till dropsy or hemorrhage takes place, when the conjoint diseases are either most difficult of cure or fatal.

#### DEGENERATION OF THE MINUTE TISSUE OF THE HEART.

**Definition.**—*A change in the muscular substance of the heart, which results in the elements of the muscular fibres being replaced by molecular particles, chiefly fatty or fibrinous (fatty or fibroid degeneration). The change tends to sudden death by rupture of the heart, or by syncope.*

**Pathology and Symptoms of Cardiac Degeneration.**—Microscopic observation has revealed certain remarkable changes, of the nature of degeneration (see vol. i, p. 118), in the heart, as well as in other organs, of slow and insidious development, most difficult to detect during life, hitherto unassailed by any remedy, productive of most fatal consequences, and the immediate cause of the sudden and unexpected demise of many distinguished men. It has especially cut off the hard-working men of the intellectual class—*e. g.*, the Rev. Dr. Chalmers, and Dr. Abercrombie, of Edinburgh; Dr. Pereira, of London; and very recently Sir Cresswell Cresswell and Sir Frederick Slade. Improved means of diagnosis have taught us to anticipate such a termination to many cardiac affections with which we are every day familiar; and in convalescence from severe injury, or under chloroform, sudden death is extremely apt to supervene by syncope, in cases where the heart has undergone the degenerations about to be described.

At least two varieties of fatty disease of the heart have been recognized. In the one form the fat, composed of oil in nucleated cells—the ordinary fat-cells—grows on the surface of the organ between its muscular fasciculi and the reflected pericardium, especially at the junction of the auricles and ventricles, along the trunk of the coronary veins, at the edges of the ventricles, at the apex, and at the origin of the aorta and the pulmonary artery. The right

ventricle is often almost entirely covered with fat. This form of fat accumulation is not degeneration; but it so gradually encroaches on and insinuates itself between the muscular fibres, that it conceals, impoverishes, and ultimately causes them to waste, so that the muscular walls become thin, especially towards the apex and over the walls of the right ventricle. In these parts the fibrous structure almost disappears, and the *columnæ carneæ* appear to spring altogether from the *endo-pericardium*. In this form of fatty heart the muscular fibres may remain healthy, although they sometimes eventually degenerate.

In the other form of fatty heart a degeneration of the fibre ensues. Its muscular element disappears, and its place is taken by fat in a molecular form, and minute oil-globules ultimately come to fill the sheaths which previously contained muscular fibre. But it is now observed that various forms of degeneration are capable of microscopic demonstration in the minute fibrillæ of the heart's substance, the result of decay or disintegration.

The recognition of fatty degeneration in the minute tissue of voluntary muscle led to the observation of analogous changes in the tissue of the heart by Corvisart and Laennec. In Scotland the younger Duncan, Cheyne, and Adams were amongst the earliest observers; but the subject has been mainly elucidated by the writings of Smith, Stokes, Andral, Rokitansky, Hasse, Paget, Ormerod, Quain, Begbie the elder, and Handfield Jones. Considerable variety of opinion prevails amongst these observers regarding the nature and the sequence of phenomena associated with this degeneration: and especially as to the symptoms diagnostic of fatty degeneration of the heart, by which its existence may be inferred during life.

I have made an analysis of twenty-nine cases of sudden death associated with such a lesion, the histories of which are scattered throughout the first ten years' "Records of the Transactions of the Pathological Society of London" (*Med.-Chir. Review*, 1858, p. 429). The results of this analysis are incorporated in the following account, illustrative of the pathology of this formidable disease:

*Age.*—Of the twenty-nine cases sixteen were females and thirteen were males, in all ranks and social conditions of life. The average age of the females was *forty-six* years, and the average age of the males was *fifty-two*. The youngest patient was a male infant six months old; while the oldest male and the oldest female appear each to have been *seventy-six* years of age. The youngest female was *ten* years of age; and the youngest male (exclusive of the infant) was a boy eleven years of age.

In the female a tendency to the degeneration seems to have been observed at a much earlier age than in the male; but between the ages of *fifty* and *eighty*, in both sexes, the greatest number of cases have occurred. The Rev. Dr. Chalmers's age was *sixty-eight*; that of Dr. Abercrombie was *sixty-five*; that of Sir Cresswell Cresswell was *seventy*. Among eighty-three cases collected by Dr. Quain, death occurred in fourteen between the ages of *fifty* and *sixty*; in eighteen between *sixty* and *seventy*; and in fourteen it occurred between *seventy* and *eighty*.

*The general health and condition of the patients previous to their fatal illness* has been variously described. In eight of the cases recorded in the Society's *Transactions* the previous health is described as delicate, weak, nervous, or reduced by previous illness, such as miscarriage, menorrhagia, hemorrhoids. The patients who are thus described were of short stature, thin, and spare make. Eight other cases are described as strong, stout, fat, muscular, or hearty. Of these some were of sedentary habits, unaccustomed to active employment, but temperate in habit. Others had suffered from slight attacks, such as of bronchitis, "liver complaints," "spasmodic pains of the stomach." Some are described as anæmic and pale, although at the same time stout and well-grown.

A third class of cases had suffered more or less severely from previous attacks of acute diseases, although they had recovered from them to some extent. One had suffered from occasional headache, with seizures of an apoplectic-like nature after fifty-five years of age. A similar case is related by Dr. Fuller (*Diseases of the Chest*, p. 602). Another had been thirty years in India, exposed to malaria, and had suffered from frequent attacks of intermittent fever. A boy, aged ten, had fever at the age of four years, afterwards chorea, and subsequently scarlatina and rheumatism five months before death. Mental anxiety and domestic distress, inducing great irritability and nervousness, were the antecedents of two other fatal cases; and five other cases are described as having to all appearance enjoyed excellent health.

Dr. Begbie, senior, in a most interesting communication to the Medico-Chirurgical Society of Edinburgh (January 15, 1851), relates some passages in the lives and deaths of the Rev. Dr. Chalmers, and of Dr. Abercrombie, which bear on the previous general health of these two distinguished men. Of the Rev. Dr. Chalmers he relates, that "to a mind of the highest order, and of wondrous energy, he united a hale and vigorous, a manly and robust frame. He spared no exertion, either mental or physical, in carrying out the great object of his life. He was hardly ever incapacitated by infirmity or loss of health in prosecuting his enterprise; and from early manhood to green old age, even up to his latest hour, he toiled and spent his energies and strength."

These things Dr. Begbie mentions, "to show that the fatal disease which lurked within, which was progressive in its nature, and probably of long standing, could neither have produced serious uneasiness nor proclaimed its presence by any unequivocal signs." But it is related that, thirteen years before his death, Dr. Chalmers had a sudden seizure on the street, of what proved to be a serious and alarming illness. He lost the power of the arm and leg of the right side, and experienced diminished sensation on that side. His face was pale, the skin cool, the pulse soft and frequent. After a few weeks of rest and quiet he completely recovered, and returned as vigorously as ever to his professional duties; but with accumulating years there came a disposition to obesity; and with the silver gray on the massive forehead came also the pallid and somewhat sickly look of fading health. He was sometimes sick at the



stomach from some trivial ailment arising from indigestion; but he was never faint, nor ever swooned away.

Of Dr. Abercrombie it is related that he enjoyed, during a long series of years, uninterrupted health; but three years before his death he was suddenly seized with loss of power and impaired sensation over the left side, but without the loss of consciousness or any affection of speech. He experienced great anxiety, and complained of præcordial uneasiness and slight headache. He sighed frequently, and had a cold skin and pallid countenance. The pulse was frequent and small at first; but after a while it subsided in frequency and rose in strength. Dr. Abercrombie's own impression was that his illness was paralysis, connected with cerebral disease. It never occurred to himself or his medical advisers to connect the symptoms with deranged circulation arising from a damaged heart; and although he complained of præcordial uneasiness, which never amounted to actual pain, and of something more than uneasiness in the left arm and shoulder, and at the base of the scapula, yet the circulation was regular, though feeble—a feebleness which might have been accounted for by the active depleting measures he himself employed, and the scanty fare to which he subjected himself. Although he recovered from this attack he continued pallid; and just before his death he was observed to be breathless on ascending a stair.

In the cases detailed in the *Transactions of the Pathological Society*, cardiac symptoms occurred sometimes suddenly after exposure to cold, with pain in the chest, shortness of breath, and palpitation. In some of the cases associated with *angina pectoris* the pain was sometimes excessive, shooting down the left side and arm, especially after any excitement. In other cases there were cardiac symptoms, consisting of a dull pain in the region of the heart or ensiform cartilage before death, associated with dyspnœa. In some *angina pectoris* prevailed for many years. The cardiac affection in several instances betrayed itself by cough, dyspnœa, and general debility, a sense of oppression at the chest, and desire to draw a deep breath, the breathing being sometimes embarrassed and aggravated by exertion. In seventeen out of twenty-nine cases death was sudden and unexpected, and cardiac disease had never been observed, and only in one case suspected to be present, by the medical attendant.

In most instances the attacks of giddiness and apparent coma are due to syncope, causing a deficiency in the supply of blood to the brain, consequent on feebleness of the heart's action. Unlike what happens in cases of apoplexy connected with hypertrophy of the left ventricle and excitement of the arterial circulation, the patient when attacked is pale, has a feeble pulse, and presents more or less lividity of the lips—symptoms which may serve as indications of the true nature of the disorder (FULLER).

*The condition of the pulse* having been observed after the cessation of an acute attack of disease, in twelve cases out of twenty-nine, which afterwards terminated fatally, it was noted as “irregular and unequal;” as “feeble, rapid, and irregular;” as “feeble and intermitting, or occasionally so every eight or ten beats;” as “large,

jerking, and compressible, one hundred and six per minute—afterwards a hundred, but small, jerking, and regular to the last;” as “small and feeble, but no irregularity or intermission.” In one of the cases the pulse was observed for two years and a half previous to death to be irregular and uncertain in its action—a condition which disappeared during an attack of bronchitis, but which reappeared as the patient recovered from the bronchitic attack—varying from *seventy* to *eighty* in a minute. In other cases, where its condition had been long noted, it is stated to have been weak, irregular, and intermitting, numbering at first *eighty*, becoming weaker, often intermitting, and more slow (*sixty* to *seventy*) shortly before death, or small and feeble. The slowest pulse recorded is *fifty-five*. In one case of death under the influence of chloroform, the pulse at first was *ninety-four*, regular, and of average firmness: subsequently, under its influence, it was accelerated, and suddenly began to get smaller, weaker, and imperceptible. In extreme cases the pulse may fall as low as twenty-six or thirty in a minute, owing to the failure of certain systoles of the heart to communicate a pulsation to the radial artery, the heart itself beating at the rate of fifty-six or sixty in a minute (FULLER, C. J. B. WILLIAMS, GAIRDNER).

*The sounds of the heart* have been observed to be modified. In one case “a loud bellows murmur” was heard all over the chest; death followed after complete destruction of the mitral valve by rupture. In another case there was extensive dulness over the heart, and a loud bellows murmur with the first sound; and in this case the edges of the mitral valve were fringed with bead-like vegetations. In a third case a *diastolic bruit* existed in the region of the aortic valves, the heart’s action ultimately becoming tumultuous, and the sounds obscured, attended with a rolling action four days before death. In this case a band of lymph was found extending across the aortic orifice, with irregular vegetation over the sigmoid valves, while at the opening of the coronary artery a false aneurism opened into the muscular substance of the left ventricle. In a fourth case the systole was attended with a loud and prolonged *bruit*, loudest over the mitral valve, followed by a distinct natural second sound, to which succeeded a remarkably prolonged interval of rest, and the rhythm was frequently irregular. Associated with these sounds was some thickening of the aortic valves, and especially a peculiar degeneration into fibrinous matter of the muscular substance towards the base of the heart. In a fifth case the area of the *præcordial* dulness was less than natural, and there was a systolic endocardial grating murmur, with feeble impulse, and the mitral valve thickened.

In four only out of the twenty-nine cases were the cardiac sounds so modified that (in the absence of valvular disease) the cause of the modification was ascribed to the morbid condition of the muscular tissue of the heart. In one of these cases the impulse and sounds of the heart were observed to have been feeble six weeks before death. In the second case the impulse was noted to have been feeble and of limited extent, the second sound being very in-

distinct, but no murmur. In the third case the heart's action was feeble and irregular, the sounds being weak; and in a fourth case the sounds were muffled, but no *bruit* existed.

*Other symptoms* associated with this remarkable degenerescence and formidable disease were,—in some cases severe vomitings and faintings, or a peculiar sinking and sense of faintness, with profuse perspirations; paleness and lividity, with urgent dyspnoea for some weeks before the fatal result; sudden aggravation of cardiac symptoms, of cough, or of general debility, for some days before death. In one case there was evidence of scurvy or of purpura; and thirteen days before death this patient became unsteady in gait, had impaired vision, with a vacant countenance, dilated and sluggish pupils, headache, and pains round the orbits, with spectral illusions, partial blindness, and hemorrhage from the nose and bowels (Bristowe, *Path. Society's Trans.*, vol. v, p. 93). In another case the general symptoms consisted of slight dyspnoea, with evidence of acute rheumatism; and four days before death dyspnoea became urgent. In a fourth case, for four years previous to death, sudden and severe attacks of shortness of breath were observed on exertion. These attacks were relieved by *ether* and *ammonia*, but they were always followed by faintness and exhaustion, with coldness and lividity of the face and extremities (Quain, *Path. Society's Trans.*, vol. iii, p. 82). In the fifth case, what is termed "biliousness" was troublesome, with a sense of heaviness and oppression referred to the sternum. Two months before death there was general *malaise*, numbness of the fingers of the left hand, with tingling or uneasy sensations over the surface generally. A sixth case suffered from attacks of headache at intervals of a few months, associated with flashes of light before the eyes, and darkness of half the objects seen. Such attacks lasted about a week, attended with loss of speech, impairment of memory, and feebleness. In three cases, attacks of *angina pectoris* are recorded. In one of these, six weeks before death, the attacks became so violent that the patient lived in constant dread of pain, which was of a most excruciating kind, accompanied by a feeling of suffocation. Three days before death he attempted suicide by cutting his throat in one of these paroxysms; but the wound was very superficial, and healed. A convulsive fit continued for ten minutes two hours before death.

*The modes of death* may be described as follows:

In eleven out of twenty-nine cases the death was absolutely *sudden* or *instantaneous*: two were comparatively sudden in bed, the patients having been previously up at the night chair; three patients were found lying dead; and in three a death-struggle existed of from three to five minutes' duration; in one case four paroxysms of rigor, nausea, and "spasm of the stomach," with small and contracted pulse, occurred at intervals within twenty-four hours, and at last suddenly proved fatal; another patient died in a paroxysm of *angina pectoris*; another under the influence of chloroform.

Of the cases of absolutely sudden or instantaneous death, nine died from rupture or laceration of some part of the texture of the

heart. Such was the mode of death of Dr. Abercrombie: a rent half an inch in length existed in the posterior aspect of the left ventricle, from which the pericardium was suddenly filled with blood. Such cases die of broken hearts—literally, not figuratively. Their cords are either torn asunder, or the fibres and minute vessels of the substance of the heart are lacerated. In one instance there was sudden rupture of the mitral valve. In five, the substance of the left ventricle was ruptured, two at the upper and posterior part, three across or along the anterior wall, and more or less close to the septum. In one there was rupture of the septum itself, penetrating the right ventricle.

Death by syncope is another usual mode of death in such cases. Thus died Dr. Chalmers, the eminent Scotch divine. It is a mode of death apt to occur in cases of convalescence from injury; and the case of Dr. Pereira, who died while convalescing from rupture of the *tendo Achillis*, and of Sir Cresswell Cresswell, who died while recovering from an injured patella, might suggest the necessity of a cardiac examination in similar cases, and the maintenance of a sufficiently stimulating diet, and wine, if previously taken, with a careful avoidance of such circumstances as may tend to syncope, in handling the injured parts of men otherwise healthy, but advanced in years.

**Morbid Anatomy and Nature of the Change observed in the Minute Tissue of the Heart.**—A considerable variety of description under this head has been given, which may be classified as follows:

1. *Cases in which there is an interstitial deposit, exudation, or growth of material, thickening or indurating the substance of the heart, and in which the new material and the sarcal particles within the muscular sarcolemma alike undergo degeneration* (Risdon Bennett, Bird, Ogle, *Path. Society's Trans.*, vol. iii, pp. 273, 276, 281). In such cases there was evidence of previous or of existing pericarditis, and sometimes of rheumatism; the heart was hypertrophic, increased in weight, and the fibrillæ exhibited undoubted evidence of extreme brittleness (Barlow, *Path. Society's Trans.*, vol. iv, p. 71; Bristowe, Peacock, Cholmeley, *l. c.*, vol. v, pp. 84 and 102; vol. vi, pp. 147 and 148). A section showed the muscular substance of the walls of the ventricle to be increased in thickness, encroached upon, and in part replaced by some adventitious product. Towards the base, the muscular substance gradually disappeared at the expense of its outer part, so that at the distance of three-quarters of an inch from the aortic valves, and from that point upwards, it was entirely wanting, and was replaced by a firm, dense, slightly translucent fibrous tissue, which extended some little way upwards on to the aorta, and downwards on the exterior of the muscle, gradually losing itself in the substance of the pericardium.

Several masses of deposit existed in the muscular substance, of an irregular form, opaque, yellowish, and somewhat firm. This deposit consisted of cell-elements, fatty particles, and degenerate muscular fibres. The cells were spherical, and varied in size from  $\frac{1}{80}$ th to  $\frac{1}{20}$ th of an inch in diameter, having very thin walls, easily broken down, and containing a few granules. Some larger

cells showed nuclei in their interior; and they were arranged in lines, so as to give the appearance of fibres. This deposit was considered of a doubtful nature as to its being malignant, tuberculous, concrete pus, or fibrinous deposit, such as is sometimes seen in the spleen. This latter view was adopted by Dr. R. Bennett, Mr. Simon, and Dr. Quain. The deposit in the heart resembled a similar lesion figured by the late Sir Robert Carswell in the fourth fasciculus of his "*Morbid Anatomy*," plate 3 (*Path. Society's Trans.*, vol. iii, p. 273). In some cases the deposit or altered part appears to be circumscribed by a boundary line, where blood-globules are abundantly present; and had the process of softening continued with surrounding vascular activity, "a circumscribed abscess must have been the result; if absorption had taken place, a fibrous degeneration would have been left" (QUAIN, *l. c.*, p. 281). The simultaneous occurrence and progress of both these forms of morbid process has also been observed (OGLE, *l. c.*, p. 282). In one case, where the heart weighed *twenty* ounces, the lesion appeared to consist of lymph amongst the muscular tissue, which lymph becoming converted into more condensed fibroid material, compressed, and ultimately replaced, the muscular substance (BRISTOWE, *l. c.*, vol. vi, p. 150).

Dr. Henry Kennedy, of Dublin, has observed that *enlarged heart* forms an important element in the natural history of the affection (RANKING, *Abstract*, vol. xxx, p. 93).

2. *Cases in which there is a very moderate amount of degeneration of tissue, or alteration in the bulk of the heart, but in which the functional disturbance is serious in the extreme, and the case rapidly fatal in its issue* (WILLIAMS, *l. c.*, vol. ii, p. 186).

3. *Cases in which, amongst apparently healthy tissue, a portion of the muscular substance has undergone degeneration*—the evidence of degeneration consisting of "the disappearance of the cross-marking of the muscular fibrillæ," and "the fibres being filled with oleo-albuminous or fatty granules." The coronary artery in such cases is found ossified or obstructed, going to the seat of degeneration (QUAIN, *l. c.*, vol. ii, p. 188; and vol. iii, pp. 262, 270, 273). In the case of the Rev. Dr. Chalmers, Dr. J. H. Bennett, Professor of Physiology, of Edinburgh University, made the post-mortem examination, and reported that the substance of the heart throughout consisted of fatty granules. The muscular fasciculi could scarcely be seen, although here and there traces of the longitudinal fibrillæ could be observed. No transverse striæ were anywhere visible. The heart was flabby and unusually soft. The coronary artery was loaded with calcareous deposit, much contracted, and in one place obliterated, presenting considerable resistance to the knife.

4. *Cases in which the death could not be ascribed to fatty degeneration of the heart alone, but where other lesions existed*—such, for instance, as fatty degeneration of the cerebral arteries, resulting in death by apoplexy; or in cases in which apoplectic seizures occur—a lesion we owe so much to Mr. Paget for elucidating; or in which, according to Dr. Quain, obstruction to the flow of blood from the head leads to congestion of the brain and hemorrhages. The



source of the obstruction Dr. Quain believes to be due to the want of power of the right ventricle (on account of degeneration) to maintain the circulation through the lungs; the blood, therefore, accumulating in the brain, tends to death by apoplexy, for the most part meningeal (*l. c.*, p. 190.)

5. *Cases in which, in addition to the degeneration of the heart's fibres, they are also encroached upon and rendered powerless by the growth of fat intruding upon them, and covering them up.*

Atrophy, wasting, and disappearance of the proper muscular tissue are the immediate results of this encroachment, terminating in a thinning of the muscular parietes of the organ (*l. c.*, vol. i, p. 192). Along with this especially atrophic result, some of the fibres of the heart which remain undergo the fatty degeneration within the sarcolemma; but this homogeneous sheath seems to remain intact, while the nuclei within or upon it disappear, or break up into streaks of oil dots. Thus, true "sarcous elements" ultimately come to be replaced by minute opaque molecules (the nature of which has not been in all cases determined), or actually by small drops of oil.

6. *There are cases of this degeneration which result in the "cardial apoplexy" described by Cruveilhier, where hemorrhagic spots, or extravasations of various sizes, occur in the substance of the muscular tissue; the surrounding tissue is found in the state of fatty degeneration, and the coronary arteries leading to the degenerate tissue and hemorrhagic spot are ossified or obstructed* (QUAIN, *l. c.*, vol. i, p. 192; vol. ii, p. 190). A condition somewhat of this kind seems to have occurred in the case of Dr. Abercrombie. The late Mr. John Goodsir (Professor of Anatomy in Edinburgh University), who made the post-mortem examination, reported that irregular ecchymosed spots were situated near the rupture which proved fatal. These spots consisted of effused blood; but their connection with ruptured vessels could not be distinctly made out. The serous membrane over them was quite entire. Both coronary arteries were much dilated as they passed from the aorta; and throughout their course they contained in their walls much atheromatous matter. The heart was slightly enlarged and dilated, rather loaded with fat, and remarkably soft, as if from interrupted nutrition. Mr. Goodsir examined the tissue near the rupture with the microscope, and found that the muscular fibres had undergone the fatty degeneration. The granules were arranged in transverse rows, and some of the fibres were nearly empty. To the naked eye the muscular tissue of such hearts presents a pale, mottled, or dirty yellow appearance. It has none of the flesh-red hue of health. The heart is soft and flabby to the touch; and its texture at the degenerate parts so friable, and sometimes brittle, that it yields to the slightest pressure, and may be torn without difficulty.

The elasticity of the muscular substance is so completely lost that, when cut across, the walls at once collapse.

**Diagnosis.**—The diagnosis of a degenerate heart is by no means easy; and a diagnostic value having been attached to the existence of fatty atrophy of the cornea—the "*arcus senilis*"—in connection

with this disease, it is necessary to notice the circumstance only to mention that, as a sign of fatty heart, it is by no means to be depended upon in every case.

**Treatment.**—Iron in its various forms, quinine, and mineral acids are the medicinal agents suggested by the nature of this disease. Freedom from anxiety, thorough repose of mind, entire avoidance of fatigue, gentle and regular exercise in the open air, careful attention to the state of the skin, a generous and stimulating diet at regular intervals, in moderate and equable amount at each meal, are the main hygienic indications calculated to impart tone to the system, improve the condition of the blood, and so induce a more healthy nutrition of the heart (FULLER). A salt-water sponging bath should be used daily. Most druggists supply the saline materials for such baths. In cases where digestion is sufficient, cod-liver oil, cream, and milk may be given with great advantage (TANNER). The bowels should be so regulated as to render straining at stool unnecessary.

Living as we do in a tumult of incessant excitement, hurry, and competition, "the struggle for existence" is maintained till the heart fails almost unperceived. The Physician can but indicate the fact; and in the life that we now live, the disease, if it is not more frequent, is certainly obtruding itself more on public notice than hitherto.

### ANGINA PECTORIS.

**LATIN EQ.,** *Angina pectoris*; **FRENCH EQ.,** *Angine de poitrine*; **GERMAN EQ.,** *Angina pectoris*; **ITALIAN EQ.,** *Angina pectoris*.

**Definition.**—*Pain or spasm of a weakened heart (CHEVERS), referred to the lower part of the sternum, or to the præcordial region, extending through the chest to the left scapula, and up the sternum to the root of the neck. The pain is characterized by its suddenness, its severity, and by a sense of constriction or of burning. It compels the patient, if walking, instantly to stop,—almost prevents inspiration. The pain is felt likewise in the left shoulder, whence it sometimes reaches to the elbow, rarely to the hand, often with a sensation of numbness in the parts. A tendency to syncope exists, associated with intense anxiety, and a sensation of approaching dissolution.*

**Pathology.**—This disease had attracted little attention till Dr Heberden, in 1772, drew the attention of the profession to it by two papers published in the second and third volumes of the *Transactions of the London College of Physicians*. He connected it with disease of the heart; and it has ever since been treated of in conjunction with diseases of this organ. It has subsequently been studied by Drs. Black, Parry, and Jenner, and by many Continental physicians; and Dr. Parry's work—*An Inquiry into the Symptoms and Causes of the Syncope Anginosa, commonly called Angina Pectoris*—will well repay perusal, though published so long ago as 1799.

The immediate cause of the paroxysm, as shown by the most weighty testimony, appears to be a sudden impediment to the coronary

circulation of the heart, and particularly to the return of the blood by the coronary veins. These results are in general due to a temporarily over-distended state of the chambers of the heart, and an inability in them to empty themselves, whether owing to weakness of the muscular parietes of the left ventricle or to other causes. If the cavity of the left ventricle is considerably dilated, or its walls attenuated or softened, or otherwise degenerate, the contractile power of its muscular tissue will be impaired in proportion. If the circulation under these circumstances happen to be suddenly hurried, or the heart's action suddenly disturbed, the cavity of the left ventricle may become so much distended as to render it incapable of contracting efficiently upon its contents, which would be quickly followed by distension of the auricle on that side, and, if relief were not soon experienced, by distension of the right side of the heart (BELLINGHAM).

The organic lesions of the heart most apt to be attended by *angina* are conditions of the aortic valves which permit of free regurgitation, with a rigid, dilated state of the ascending portion of the arch of the aorta, which permits the blood from the large vessels to regurgitate into it, combined with either of the following conditions of the left ventricle:—(1.) Dilatation of the cavity; (2.) Attenuation of the parietes; (3.) Softening or degeneration of the muscular tissue (BELLINGHAM).

Any one of those morbid conditions may be present, or two or more of them may be combined, and yet *angina* may not necessarily occur, so long as the circulation continues tranquil, and so long as the left ventricle is able to get rid of the blood which enters its cavity, and does not get over-distended. But if the heart's action becomes disturbed by some sudden mental emotion or other cause; or even without this occurring, if the stomach is loaded with indigestible food, and it and the intestines are distended with flatus, by which the cavity of the chest is encroached upon, and the heart's movements are impeded, a paroxysm of *angina* is the general result. John Hunter, who suffered greatly from this disease, used to affirm that his life was in the hands of any person or circumstance which acted powerfully on his mind; and, in fact, he ultimately died in St. George's Hospital, from strong but suppressed feelings on a point in which he was interested. Ascending a staircase or other acclivity, or indeed any active exertion, is a powerful exciting cause. In persons who have had previous attacks, the paroxysm is liable to supervene during sleep, as the result of a frightful dream disturbing the heart's action, or of considerable distension of the stomach by flatus impeding the movements of the heart.

The late Sir John Forbes, in an able article on this subject in the *Cyclopædia of Practical Medicine*, has shown that *plethora* becomes a very common complication of *angina pectoris*. The very existence of *angina* tends to produce *plethora* if it did not previously exist; a sedentary life and abandonment of all active bodily exertions are almost inevitable consequences of the disease.

*Angina pectoris* ought therefore to be regarded rather as a *symptom of organic disease of the heart* than as a distinct form of disease.

What *dyspnœa* is to the lungs, *angina* appears to be to the heart; so that Bellingham has termed it the "dyspnœa of the heart."

**Symptoms.**—The paroxysms of this disease generally supervene suddenly, and are characterized by a constrictive anxious pain, fixed most commonly on the left lower half of the sternum, and rarely extending above the fourth rib. Occasionally, however, it extends over the whole anterior portions of the chest, along the neck to the lower jaw, into the back and shoulder, down the arm to the elbow, and even to the hand and fingers. In this course it affects externally the superficial cervical plexus and its ramifications, as well as the anterior thoracic nerves, the cubital nerve, and its divisions. The pain is also sometimes sub-sternal, and then follows the course of the nervous plexus placed between the folds of the mediastinum, and also the branches of the eighth pair which go to the large arteries and surround the bronchial tubes. The pulse is sometimes rapid, sometimes hardly to be felt; and the breathing is sometimes accelerated, and at other times it is imperceptible. Mr. Hunter, when laboring under the paroxysm, could scarcely feel his pulse, and thought he should die unless he exerted his voluntary muscles to aid respiration. Many have died so—asphyxiated. Darwin has seen the action of the diaphragm, and consequently the phrenic nerves, affected; while Laennec mentions that the lumbar and sacral nerves also partake of the pain. Besides the parts which have been mentioned, the gastric system is also much affected, the patient perhaps being in an instant distended with wind, and only relieved by repeated eructations. The urine is sometimes suppressed during the paroxysm. In all cases where the patient is not broken down by disease, the mind is clear, but the face and extremities are cold and pale. At length the paroxysm subsides gradually, when much wind is discharged, accompanied by a copious and almost involuntary flow of pale, limpid urine, and the patient for the time recovers.

The duration of the paroxysm depends on the persistence of the impediment to the coronary circulation. Sometimes the pain only lasts a few minutes, while at other times it will continue for two or three hours, a whole day, or even longer. The interval of respite from pain is likewise very uncertain—from a few hours to a few days, or a few months. Each repetition, however, increases the tendency of the paroxysm to return, and increases its violence; and at length, perhaps, an aggravated attack occurs which puts a period to the patient's existence. So seems to have died the late John Leech.

**Prognosis.**—The danger is in proportion to the nature and degree of the organic lesion on which the disease depends. Sir John Forbes found that of sixty-four recorded cases of angina forty-nine died, almost all of them suddenly.

**Treatment.**—The indications for the treatment of *angina pectoris* are to be found in a study of the lesions on which the paroxysms are found mainly to depend. Medicine can do little more than mitigate the severity of an attack; and this is generally best done by diffusible stimulants, such as *brandy, ether, chloroform, ammonia,*

*chlorodyne*. [The inhalation of from five to ten drops of the *nitrite of amyl* has relieved the pain (BRUNTON).] Alcohol in small doses often repeated, *sesquicarbonate of ammonia* in doses of *three to five grains*, the *muriate of ammonia* in doses of from *ten to twenty grains*, have each purely powerful stimulant effects. Hot bottles and *sina-pisms* should be applied to the feet. The bowels may require to be rapidly and efficiently acted upon.

### CYANOSIS.

LATIN Eq., *Cyanosis*; FRENCH Eq., *Cyanose*; GERMAN Eq., *Cyanose*—Syn., *Blausucht*; ITALIAN Eq., *Cianosi*.

**Definition.**—*A peculiar blue condition of the skin symptomatic of various malformations or derangements of the heart and great vessels, so that a small portion only of the blood is subjected to aeration in the lungs.*

**Pathology.**—The blood corpuscles absorbing oxygen in the lungs convey it through the circulation to every part of the body, and thereby render the metamorphosis of tissue possible. If this absorption of oxygen in the lungs by the blood-corpuscles is for any reason stopped or diminished, *arterial* blood retains the properties of *venous* blood, and the condition termed *cyanosis* arises.

In its *minor degree* it is associated with various forms of cardiac and pulmonary derangement having the effect of obstructing the flow of blood in the veins of the lungs and of the system generally. The chief of these is constriction or partial obstruction of the pulmonary artery, combined with systemic venous engorgement. In cases of cyanosis the skin is usually thin, the capillaries abnormally large; hence, when obstruction to the pulmonary and systemic venous circulation causes imperfectly aerated blood to flow throughout the system—and still more so in cases when, in consequence of some congenital malformation, a small portion only of the blood is subjected to the aerating influence of respiration—a dark, dusky, more or less livid hue is imparted to the skin.

In its *more severe form* the condition is usually associated with such a malformation, disease, or injury of the heart or great vessels, as permits venous and arterial blood to mix, and after mixture to be so distributed to the systemic capillaries. A patulous condition of the *ductus arteriosus*, an open *foramen ovale*, a deficiency of part of the *septum* of the ventricles, a heart formed of one ventricle and one auricle only—the aorta and pulmonary artery rising from a common trunk—are amongst the usual conditions which lead to cyanosis.

It is usually, therefore, a congenital affection, and the physical signs vary with the precise condition of the heart and arteries to which the cyanotic discoloration is due. The action of the heart is usually more forcible than in health, and hypertrophy and dilatation of the right ventricle are almost always present. Deficiency of animal heat is also a constant phenomenon.

The causes of cyanosis are arranged by Vogel into two groups:



(1.) In the one group the supply of oxygen to the blood, and consequently the oxidation of the blood-corpuscles, is prevented by derangements of the respiration or circulation (*i. e.*, by causes which are external to the blood); (2.) In the other group the blood-corpuscles lose the property of absorbing oxygen. This occurs in some cases of *pyæmia* and the last stage of *pulmonary tuberculosis*. There seems to be a diminished capacity of the blood-corpuscles to redden themselves.

**Malformations of the Heart** may be here simply enumerated. They may be arranged under the following heads:

1. *Misplacements of the heart (Ectopia Cordis)* occurring congenitally, as in cases of transposition of the viscera. For an account of all that is known on this subject, the reader is referred to a paper by Professor Allen Thomson, in the *Glasgow Medical Journal* for July, 1853; and to the *Lancet* of August 8, 1863, where the condition is described by Professor W. C. Maclean as having been recognized during life. For an excellent report on cases of *ectopia cordis*, see *Pathological Society's Trans.*, vol. vi, p. 98.

2. *Congenital deficiency or absence of the pericardium*, in which the heart is, as it were, naked, and lying in one cavity with the lungs (*Path. Society's Trans.*, vol. iii, p. 60; vol. vi, p. 109).

3. *Arrest of development of the heart at an early period of fœtal life*: as in hearts with one auricle and one ventricle; with imperfect separation of the ventricles; cases of contraction or absence of the pulmonary artery, the aorta arising from the right ventricle, and the *septum* of the ventricles imperfect. In cases of imperfect *septa*, the aorta sometimes arises from the two ventricles. Sometimes the ventricular *septum* is wholly absent.

4. *Premature closure of fœtal passages—the foramen ovale and ductus arteriosus—malformations causing changes which ought not to ensue till after birth*. The consequences of such premature results are chiefly cyanosis, combined with imperfect dilatation of the branches of the pulmonary artery.

5. *Irregularity of the valves, and origins of vessels, which may not in the first instance interfere with the functions of the heart, but which are apt to lay the foundations of disease in after-life*. Examples are to be seen in cases where two aortic valves occur; where there is fusion or union of two of the valves; where there is transposition of the aorta and pulmonary artery, both auricles opening into the left ventricle (*l. c.*, vol. vi, p. 117). Excess of pulmonary valves (*l. c.*, vol. iii, p. 301; vol. iv, p. 102). (See *College of Physicians' Nomenclature*, p. 181.)

The **treatment** of malformations which are associated with cyanosis is mainly preventive of dyspnœa and palpitation, by the avoidance of fatigue and mental excitement, the maintenance of temperature, and especially by a nourishing diet and warm clothing.

## PALPITATION AND IRREGULARITY OF THE HEART.

LATIN Eq., *Palpitatio et tumultus cordis*; FRENCH Eq., *Palpitations*; GERMAN Eq., *Herzklopfen und Unregelmässigkeit in der Herzthätigkeit*; ITALIAN Eq., *Palpitazioni ed irregolarità del cuore*.

**Definition.**—*Disturbances of the action of the heart unconnected with organic mischief.*

**Pathology.**—The interest which attaches to this subject consists in the difficulty which sometimes exists in the recognition of *functional* as distinguished from *organic* disease; and the fact that great uneasiness and distress, both mental and bodily, result from the occurrence of functional disturbance, while its persistence is apt to induce organic disease. Death has resulted from simple functional disturbance (GRAVES).

The forms of functional disturbance which simulate organic disease of the heart are palpitation, fluttering, or a “rolling” action of the heart, sometimes associated with a valvular murmur and irregularity, feebleness, or altered rhythm of the heart’s action. The causes of such phenomena are common to adolescence and middle adult life, and are most frequently traceable to excessive mental exertion and sedentary occupation; great anxiety and strong mental emotion; nervous exhaustion from various causes—such as uterine irritation, excessive venery, masturbation; the influence of poisons on the heart’s action—such as that of tobacco, spirits; *gout*, *rheumatism*; derangements of the stomach and liver, characterized by the existence of flatulence and acidity (FULLER).

Exaggeration of the functional acts is characteristic, and the general symptoms indicate great distress, a sense of fulness and of deep oppression in the *præcordial* region, pain, breathlessness, and tendency to faintness. Frequent giddiness, with pain, heat of head, singing in the ears, flushing of the face, coldness of the extremities, are associated with forms of dyspepsia characterized by excessive flatulence, acid eructations, restlessness at night, depression of spirits, and mental anxiety as to the nature and probable issue of the disease; all contribute to induce and maintain the functional disturbance of the heart (FULLER).

The characteristics of palpitation, as due to functional and organic causes, have been already indicated (see pp. 628 and 629); and repeated examinations of the patient are necessary to arrive at a just conclusion.

Perversions of rhythm have sometimes been attributed to functional disturbance merely; but when altered cardiac rhythm is not merely of temporary duration, but is attended with giddiness, faintness, or actual syncope, it is more likely (in the absence of positive knowledge as to the state of the heart’s tissue) that such altered rhythm is due to organic or textural degeneration. In every case examined by Dr. Fuller he has found it to be so.

[**Irritable Heart.**—There is another form of functional disorder of the heart which was quite common amongst the soldiers during the late war,

and is occasionally met with in civil life. It has been called by Dr. Da Costa, the *Irritable Heart*, and by Dr. Henry Hartshorne, *Cardiac Muscular Exhaustion*. It was familiarly known as the "trotting heart." The symptoms are: great frequency of the heart's action, constantly recurring attacks of palpitation, and pain in the præcordial region. Though the palpitations happen chiefly during exercise, they may occur when the patient is at rest, and, in some cases, the seizures are at night, hindering sleep. During the fits of palpitation, dizziness and headache are complained of, and sometimes occur constantly. The pain may be dull and constant, or shooting and paroxysmal; its site is over the apex, and the skin over that point is generally quite sensitive. The very rapid action of the heart is associated with an extended, not forcible, but abrupt or jerky, impulse, of sometimes irregular rhythm, and with a short sharp first sound, and a very distinct second sound; occasionally the first sound can hardly be heard, resembling the cardiac condition met with at times in continued fevers (vol. i, p. 374). There are no cardiac or neck murmurs. The area of percussion-dulness is not increased. The pulse is usually compressible, and may, or may not, share the character of the impulse; it is influenced by position, being less by twenty beats when the patient is lying down. There is no constant increased frequency of respiration, though in some cases there is distressing breathlessness on exertion. The general health is often good.

The disorder is an obstinate one, and hinders exercise, and consequently unfits the soldier for active duty. Its cause is uncertain. In many instances it has followed forced marches; in others it has happened after fevers or camp diarrhœa. It did not appear to be necessarily connected with scurvy, or the abuse of tobacco, nor with anæmia, the aspect of the sufferers being often that of ruddy health.

The most successful treatment was keeping the heart quiet by occasional doses of digitalin, or veratrum viride, or atropia, and giving tonics (DA COSTA, *Medical Diagnosis*, 2d ed., 1866, p. 317. H. HARTSHORNE, *Am. Jour. of the Medical Sciences*, July, 1864).

**Disease of the Heart, how far a Disqualification for Military Service.**—"Heart disease" was one of the most common causes for application of discharge from the service on surgeon's certificate of disability; there is no doubt that it was often feigned, and that it gives large opportunity for the practices of the malingerer. By the United States Army Medical Regulations, "heart disease," to render a discharge necessary, must consist of valvular lesion. How far cardiac disorder is really a disqualifying element, and hinders the man doing duty, is a practical question which is constantly presenting itself to the military medical officer. Dr. Da Costa writes: "The mere fact of a disorder of the heart existing ought not to be a cause for discharge. If a nation engaged in war has a population flocking to its standard; if the ranks can be readily recruited with able-bodied men; if it does not care for the military efficiency of those who have been long enough in the army to attain military efficiency, unless this be combined with perfect physique,—then of course it had best at once restore to the field and workshops those whose frames are no longer free from disorder. But under other circumstances many cases, even of organic disease of the heart, may be retained for service in garrison and the like, and a skilful medical officer should be chosen to select them—the principle of selection involving the extent and nature of the complaint, and taking as a guide the coëxisting evidences of disturbed function, rather than the mere name, or label it bears" (*United States Sanitary Com. Med. Mem.*, p. 381-2).]

### SECTION III.—MORBID CONDITIONS OF THE GREAT BLOODVESSELS AND OF THE BLOOD.

#### AORTITIS.

**Definition.**—*Inflammation of the aorta characterized by pains, with local pulsation along the course of the aorta or under the sternum, associated with rigors and pyrexia, with apnœa and great palpitation of the heart.*

**Pathology and Symptoms.**—Mere redness of the artery, without any alteration of the inner membrane, may be simply cadaveric, owing to imbibition of blood-pigments. The post-mortem appearances of true aortitis are,—great vascularity, and especially of the *vasa vasorum*, a thickened and pulpy state of the inner and lining membrane, which, having lost its glistening aspect, has a villous, rugous, or granular aspect. Lymph may be seen on its surface, and slight excoriations or superficial ulcerations may be observed here and there. The elasticity of the vessel no longer exists, so that rupture takes place very readily. Local pulsation is extremely energetic; and as we know that in cases of inflammation of the carotid there is pain when the vessel is pressed upon, so pain in an inflamed aorta may be aggravated by motion. A murmur is appreciable—a loud, rough, systolic bruit, due to the passage of blood over the rugous and unpolished state of the inner membrane (GENDRIN, PARKES). In the case related by Dr. Parkes it was heard over the third dorsal vertebra, down into the lumbar region (*Med. Times*, Feb. 23, 1850).

**Causes.**—Rheumatism, gout, and syphilis seem to have a decided influence in the development of this disease.

**Treatment.**—Leeches should be applied freely over the course of the vessel; and large doses of opium, or of *ether* with *chloroform*, may be given to relieve pain and dyspnœa. Iodide of potassium, or colchicum, may also be indicated.

#### ANEURISM OF THE AORTA.

**Definition.**—*A circumscribed dilatation of some portion of the aorta, consequent on lesion of some of its coats.*

**Pathology and Symptoms.**—They most frequently arise from the ascending portions of the arch, and seem to spring most frequently from those parts of the vessel against which the current of the blood is most forcibly directed. The effects produced vary with the seat and the size of the tumor; and aneurism is generally one of the most distressing and puzzling of thoracic diseases.

Aneurisms opening upon mucous surfaces, especially upon the air-passages, are generally attended with small and irregularly-repeated hemorrhages. The persistence of these trifling amounts of blood in the expectoration justifies suspicion of aneurism, in the absence of any other circumstances to account for it. Tumors,

such as are caused by aneurism, often give rise to such symptoms as are suggestive of laryngeal disease; therefore, in all cases, the larynx ought to be examined on the one hand by the laryngoscope, and on the other, a physical examination of the chest should be made for the signs of an aneurism or tumor. The frightful and agonizing dyspnoea is generally due, in cases of aneurism, to implication of the recurrent laryngeal nerve, and not to ulceration or disease of the larynx.

In some cases of thoracic aneurism the dyspnoea may have a spasmodic asthmatic character, without any tendency to laryngeal spasm, associated with the persistence of small hemorrhagic expectorations. In such cases the aneurismal tumor presses upon the bronchi towards the roots of the lungs, evidence of valvular disease and of pneumonia being absent.

In another class of cases paroxysmal sufferings, in the form of *angina pectoris*, have their origin in the interference by an aneurism with the thoracic nerves, or with the venous circulation in the heart itself.

The variable character of the hemorrhage, and the remarkable intermissions of such hemorrhages from aneurismal sacs, are symptoms of thoracic aneurism which are now only beginning to be appreciated in their proper light, especially since the case of Mr. Liston drew the attention of the profession to them. Hemorrhage may occur in one of the three following forms: (1.) A frothy bronchitic sputum streaked with blood; (2.) A rusty sputum, very like pneumonia, but usually more abundant, more frothy, and less viscid; (3.) A deeply dyed purple or brownish-purple sputum, like the so-called "prune juice" expectoration characteristic of the third stage of pneumonia, and of certain forms of pulmonary hemorrhagic condensation from valvular disease of the heart; (4.) Any of the preceding forms of hemorrhage alternating with small discharges of pure, unmixed, but generally imperfectly coagulated blood. The bronchitic varieties of sputum, either stained or streaked in different proportions with blood, occur chiefly in tumors pressing on the trachea and larger bronchi, and not producing consolidation of any part of the lung; while the "prune juice" sputum occurs when the lung is directly involved in the tumor (W. T. Gairdner's *Clinical Medicine*, p. 454, *et seq.*)

Permanent contraction of the pupil on the affected side is in some cases another sign of aneurism, which Drs. Reid, Gairdner, Ogle, and others have clearly demonstrated.

The correlation of the symptoms, as possibly indicating thoracic aneurism, is the main point for attention; and in addition to those noticed, raucous voice and aphonia are found sometimes associated with thoracic aneurisms. Undue pulsations, dyspnoea at intervals, oppression at the præcordial region, with difficulty of swallowing solid food, are, in combination, significant of aneurism.

The sphygmograph is calculated to yield important aid to diagnosis (SANDERSON, ANSTIE, and FOSTER), by showing in the pulse-trace the modifications in the movement of the blood produced by the diseased condition. These modifications are intimately depend-



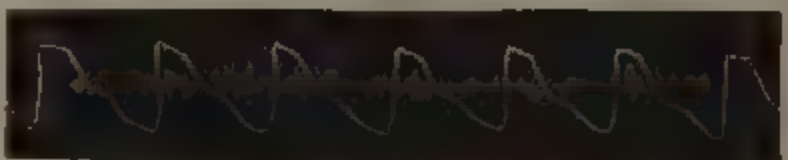
ent on the seat of the tumor, its size relatively to the vessel with which it is connected, and the elasticity of its walls (FOSTER).

The more important modifications may be considered: *first*, As to aneurism so situated on an artery that the pulse can be observed on the vessel below the tumor; and *secondly*, Aneurisms affecting the aorta. The beat of an artery, when carefully felt below an aneurismal tumor which implicates it, is found to present unusual characters: it is weakened, and generally retarded. The sphygmograph shows that modifications of the pulse occur both in its form and force. In the pulse-trace collected from an artery below an aneurism, the movement of the blood in the vessel approaches rather to that which is normally seen in the smaller arterial branches. The vertical line of ascent disappears; and often this line approaches in length that of descent. Thus we have a more feeble pulsation given to the finger; and, as the summit is slow to occur, there is a sensation of apparent retardation felt in addition. The following figures (37, 38) represent the modifying influence of an elastic aneurismal sac on the pulse-trace of the left radial artery

FIG 37 \*



FIG 38 †



(Dr. B. FOSTER). There was an aneurism of the left subclavian artery within the thorax. The right pulse shows the trace of another lesion under which the man labored—namely, insufficiency of the aortic valves. So characteristic of the conditions in this case were the above traces, that they alone enabled Dr. Foster to arrive at the diagnosis to which ordinary means had led those watching the case. At the time when the above traces were taken, the tumor was evidently large and very elastic—hence the great modifications in the pulse-form produced.

In *aneurisms of the aorta*, very much less striking indications are afforded by the pulse-trace. Nevertheless, Dr. Foster's experience leads him to believe that the position of a thoracic aneurism may be indicated by the pulse changes *e. g.*, when a small aneurism or even a considerable dilatation of the ascending aorta exists, the pulse of the right radial is generally reduced in size, when compared with the left (Dr. Foster's *MS. Notes*). The indications pointed out by Marey are,—(1.) Modifications in the force of the pulse; (2.) Modifications in the intensity of the diastolic; and (3.) The exis-

\* Left radial.

† Right radial.

tence of a constant difference in the pulse-form of the two radial arteries.

(1.) The force of the pulse, according to Marey, is seldom much diminished—a character of small value in diagnosis. The causes of this want of change in force reside probably (*a.*) In the small size of the tumor relatively to the volume of the aorta; (*b.*) In the fact that the sac is sometimes not placed in the direct route of the blood, but communicates with the vessels, and thus has a much less transforming effect upon the blood-movement than a tumor which must be traversed by the current; and (*c.*) In the thickness and slight elasticity of the wall of the sac often found in aneurisms in this situation. The force of the pulse, too, in these cases is altered very often, not in one artery alone, but in the vessels of both sides of the body; and, on the other hand, it must be remembered that the tumor, by compressing the orifice of one of the branches of the aorta, may cause a peculiarity in the radial of one side.

(2.) The modification in the dicrotism may exist in one or both radial pulses, and is occasionally a sign of much value.

In aneurisms of the descending thoracic aorta the dicrotism proper is often much increased in both pulses, but especially in the right, while the left radial is smaller than the right. This latter fact may be explained by the relation of the innominate and left subclavian to the blood current, the former of which receives the full force of the blood discharged from the ventricle; but the current has to deflect itself slightly to reach the left subclavian, and is partly drawn into the descending thoracic aorta where the aneurismal sac gapes to receive it. The dicrotism is increased in these cases by the contraction of the aneurismal sac (Dr. FOSTER).

(3.) The presence of a constant dissimilarity in the pulse-traces of the radial arteries is the most valuable sign in the diagnosis of aortic aneurisms. In many cases the finger can perceive a want of parallelism in the beats of the radials, but often this is too slight to be detected by the finger. It sometimes shows itself in the trace by a slight difference in the dicrotism only; at others, the difference in form is more evident. When the tumor is so situated that it can be handled, we can gain valuable evidence as to its nature by observing the changes in the tension of the arteries, produced by its alternate compression and relaxation (Dr. B. FOSTER).

**Causes.**—The causes of thoracic aneurism are exceedingly obscure; but there is good reason for believing that the morbid constitutions associated with *gout*, *rheumatism*, and *syphilis* are the circumstances under which aneurisms are most apt to be developed, the elasticity of the vessel being impaired by structural changes. With regard to the influence of syphilis, I may here observe that I have dissected, during the past four years (at Fort Pitt and at Netley Hospitals for invalids), twenty-six bodies of soldiers, in each of which a distinct history of syphilis was present, associated with unmistakable syphilitic lesions; and of these twenty-six cases, *seventeen* had the coats of the thoracic aorta impaired by characteristic changes—changes which are uncommon at an early period of life, and which I have every reason to believe are due to syphilis. The changes are obvious

from cicatricial-like loss of substance of the inner coats, small local dilatations of the artery, and in several cases aneurismal expansions, one as large as an orange, which proved fatal. A characteristic case of aneurism of the thoracic aorta resulting from syphilis is also recorded by Assistant-Surgeon Alfred Lewer, in the *Medical Report of the Army Medical Department* for 1862, p. 512. Hereditary transmission of aneurism has been noticed by Dr. Fuller.

**Treatment.**—Local bleeding is useful when there is pain and tenderness over the aneurismal sac; and general bloodletting may be useful if the circulation is excited, and the patient be of full habit, but not on the principle advocated by, or ascribed to, Valsalva. Of all remedies, *digitalis*, *aconite*, and *veratrin* are the most useful in tranquillizing the action of the heart. They tend to render the circulation slower, without deranging the action of the stomach. The deposition of fibrin from the blood is more prone to take place when the circulation is “slowed;” indeed, it is the principle of treatment in the cure of aneurisms by pressure. The current of blood is not stopped, but is simply rendered more slow, so as to have an amount of stagnation of blood in the sac favoring the separation of fibrin and its coagulation. A diminution of from *ten* to *fifteen* pulsations of the heart in the minute will thus greatly tend to the filling of the sac with coagula (FULLER).

[Dr. George W. Balfour, of Edinburgh, has collected fifteen cases, some of them occurring under his own observation, in which the treatment of aneurism of the aorta by iodide of potassium was persistently carried out, and in all, save one—and that a perfectly hopeless case—there was marked relief. In twelve there was undoubted diminution in the size of the sac, while in a few there was so complete a subsidence of the tumor and improvement in all the symptoms, as to amount to an apparently perfect cure. The dose varied from five grains to thirty, three times a day. The action of the remedy would seem in some degree proportionate to the quantity taken, because the relief of pain, which is one of the earliest symptoms of amendment, did not happen till a certain efficient dose had been given. It is therefore of consequence to attain a saturated dose as rapidly as possible, and it is perhaps better to begin with thirty-grain doses, intermitting them for a day or two, if the happening of symptoms calls for it. Some of the published cases seemed to show that a few weeks were sufficient to bring about the curative result, but Dr. Balfour’s experience is, that any considerable amendment can only be procured by keeping the patient for many months, perhaps twelve or more, persistently saturated with the drug. The strict enforcement of the recumbent position is insisted on as an adjuvant of paramount necessity (*Edin. Med. Jour.*, July, 1868).]

A most interesting paper has been published in the *Medical Report of the Army Medical Department* for 1862 (p. 472), from the pen of Joliffe Tufnell, Esq., advocating the treatment of aneurisms of the thoracic and abdominal aorta on the principle here enunciated, of “slowing” the circulation. His treatment consists of restricted diet and perfect rest in the horizontal position, for periods varying from eight to thirteen weeks, combined with the employment of

such remedies as may be necessary for special ends. The horizontal posture must be *strictly and absolutely maintained*, in a light and cheerful airy room, into which the sun shines, and from which the patient may be able to have as cheerful a view as possible out of the window.

The diet must be confined to *three* meals, served at regular intervals, and restricted to the following in kind and amount:

*Breakfast*—Two ounces of white bread and butter, with two ounces of milk or cocoa.

*Dinner*—Three ounces of broiled or boiled meat, with three ounces of potatoes or bread, and four ounces of water or light red wine.

*Supper*—Two ounces of bread and butter, and two ounces of milk or tea.

These diets should make in the aggregate *ten ounces of solid and eight ounces of fluid food in the twenty-four hours, and no more*. The object of the special diet is to maintain life on as little food as possible without inducing restlessness, as in some irritable constitutions; but if such restlessness should occur, a little more food may now and then be allowed. *Anodynes, aperients, narcotics, sedatives, and tonics* are also useful aids in the management of the case. Of anodynes, Mr. Tufnell regards *lactucarium* as the most valuable, given in the form of a pill, either by itself or combined with *humulin* and *hyoscyamus*. Mr. Tufnell's practical suggestions in detail will well repay a careful study, and merit publication in a more accessible form than in a "*blue-book*."

Tracheotomy may prolong life in some cases where *stridor* exists, if the laryngeal symptoms are the source of immediate danger (W. T. GAIRDNER).

## PYÆMIA.

LATIN Eq., *Pyæmia*; FRENCH Eq., *Pyohémie*; GERMAN Eq., *Pyæmie*; ITALIAN Eq., *Piemia*—Syn., *Piemasia*.

**Definition.**—*A febrile affection resulting in the formation of abscesses in the viscera and other parts, and usually associated with phlebitis.*

**Pathology.**—There are febrile conditions in which the blood is materially disturbed in various ways, and which tend to complex forms of lesions in many parts, and especially to multiple centres of inflammation, with a great tendency to suppuration. A number of morbid processes, having many elements in common, of great practical importance, of frequent occurrence, and dangerous to life, have been described of late under the very various names of—(1.) *Simple pyogenic fever* (JENNER); (2.) *Acute purulent diathesis* (TESSIER); (3.) *Septic poisoning of the blood* (the *septicæmia* of VOGEL); (4.) *Systemic infection* (the *ichoræmia* of VIRCHOW); (5.) *Putrid fever*; (6.) *Pyæmia*; (7.) *Phlebitis*.

The multiplicity of views and great uncertainty which still pertain to many points regarding the pathology of the phenomena comprehended under these various names, make it necessary to write with doubt and some hesitation; more especially as the de-



tails into which the topics may be carried which are embraced under this head are daily becoming more and more extensive. The aim of this account, therefore, is to put the student in possession of the more important bearings of the subject, in relation especially to pyæmia, phlebitis, and the phenomena of embolism.

The first two—namely, the *pyogenic fever* of Jenner, and the *purulent diathesis* of Tessier—are characterized by the occurrence of *multiple abscesses*. The remaining *five* of the morbid states which have been enumerated have been recently classed by Virchow, Bilroth, and others, under the common name of “*metastatical dyscrasiæ*.” In them there is evidence of the occurrence of secondary or metastatical inflammation—a tendency to multiple centres of inflammation, to “multiple abscesses,” and to suppuration in various parts of the body. Associated also with these lesions is the occasional formation of *clots, thrombi, plugs, or embolia*, the occurrence of *thrombosis*, or of *embolism*, or of *phlebitis*, *softening* of the minute tissue of visceral organs, such as the *brain*, the *lungs*, the *heart*, the *liver*, or the *kidney*, or of *gangrene* of the extremities.

**Pyogenic Fever.**—In the Gulstonian lectures delivered at the Royal College of Physicians in London, for 1853, Dr. William Jenner directed particular attention to some lesions which are apt to follow immediately after the termination of acute specific diseases, such as scarlet fever and the like. The simplest form of these lesions consists in the formation of several (or multiple) abscesses of small size in the subcutaneous connective tissue, especially of the scalp, chest, loins, legs or arms, accompanied by more or less febrile disturbance. Such multiple abscesses are (on circumstantial evidence only) presumed to owe their origin to a diseased condition of the blood—to represent what is popularly known as “the dregs of the fever”—as the media by which something unwholesome is ultimately evacuated—or as a *crisis* in which the specific affection terminates. Sometimes, on the other hand, these abscesses are accompanied by severe constitutional disturbance, and instead of being superficial, are deeply seated, either in the connective tissue or in the cavities of the joints, and in rare cases even in the serous cavities of the cranium, abdomen, or thorax. Cases of all these varying degrees of severity are observed, which seem to differ only in the more or less wide diffusion of the local affections. Such cases are found to be in close alliance with those diseases in which purulent discharges issue at the same time from the mucous passages, and to that chronic cachexia in which the least scratch or abrasion of the skin tends to “fester” and not to heal. This condition of the blood and system was first recognized and described by Tessier, in 1838, under the name of the “*purulent diathesis*,” or “a tendency to suppuration in the solids and coagulable fluids.”

In the cases which are recognizable as belonging to this class of diseases, the febrile disturbance seems to be established before any local lesion is set up; and the morbid condition of the blood thus came to be looked upon as “a primary substantive affection,” because it seemed to lead to a “fever” followed by these lesions.

Cases of this morbid condition are related by Dr. Jenner in the



*Medical Times and Gazette* for May 7, 1853; and they may be compared with a case of multiple abscesses in and about the joints, associated with rheumatism, and described by Dr. Bennett in his valuable *Principles and Practice of Medicine*, p. 803, under the name of *ichoræmia* or so-called *pyæmia*.

The subcutaneous tissue and the joints are the most frequent seats of these abscesses; much less frequently the lungs or other viscera, which in the following class of cases are the most frequent seat of the multiple abscesses. Here we have an important distinguishing character between the two classes of diseases—namely, that the abscesses of pyogenic fever seem to be developed out of a *constitutional* state of general ill-health, whereas those which are about to be considered may be regarded from the beginning as due to a blood-poison, or other causes of irritation set up from external sources. In the former case the cause of the disease seems to be *constitutional*; in the latter, *specific*. Hence the College of Physicians places pyæmia among the general diseases, Class A (see vol. i., pp. 176 and 178). Again, the class of lesions associated with pyogenic fevers is still further distinguished as of one kind by the following characters: (1.) The abscesses are not consequent upon the pre-existence of any abscess, ulcer, or clot, or plug in the bloodvessels; (2.) There is no evidence of the formation of pus or of fibrinous debris in the veins, nor of the passage of that fluid into the blood, and the establishment of (so-called) *pyæmia*; (3.) The symptoms, and still more the *situation*, of the disseminated abscesses differ in cases of *pyogenic* fever from those which occur in the so-called cases of *pyæmia* or *phlebitis*.

It is with typhus or typhoid fever only that pyogenic fever is apt to be confounded; but from this fever it is distinguished by the activity of the febrile symptoms at the outset, the early delirium, the absence of eruption, and the rapid formation of the numerous centres of suppurative action. The closest alliance, pathologically, of pyogenic fever seems to be with phlegmonous erysipelas.

**Metastatical Dyscrasie**, under the generic name of pyæmia, have attracted considerable notice from two points of view:

(1.) In connection with various conditions of the veins, to which the name of “phlebitis” has been given; (2.) In connection with various changes in the blood itself, and more especially of late years, as described by Virchow and Bennett, under the name of “leucocytosis” (*ante*, p. 102). Several scientific questions associated with each of these views are still the source of controversy.

*Pyæmia* literally means a condition in which—(1.) There are pus-cells in the blood; but the expression has now come to imply—2.) That the blood is altered throughout the whole system by the poisonous action of putrid animal substances, in the form of gases, fluids, or solid particles, which so disturb its relations with the living tissues as to induce coagulation of the fibrine of the blood in some part, during life, within the bloodvessels, associated with fever, and the formation of local abscesses in one or more of the viscera and other parts, and usually accompanied by *phlebitis*.

This latter signification of pyæmia has been in some measure

forced upon us, because it is found impracticable to say when any given specimen of blood is full of white blood-discs, or of pus-corpuscles. It was once supposed that pus could be absorbed *as pus*, and conveyed away in substance; and that two results might follow such an event: either—(1.) That the pus would be passed off by the urine or the fæces as an excretion, but still in the form of the original pus; or, (2.) That the phenomena of pyæmia would be the other alternative.

Now we know that neither of these results ever takes place. Pus, *as pus*, is never taken up or absorbed into the system. Its fluid part only may be absorbed by veins or lymphatics; but the solid portion remains as a thick, inspissated, or concrete mass; and thus the absorption or disappearance of an abscess sometimes gives rise to such “cheesy” products as are described by the name of “tubercle;” and which may subsequently induce ulceration, as a foreign body would. Again, pus may be completely absorbed; but only after the cells have been reduced to the state of milkiness by fatty degeneration, and become converted into an emulsive mass—a kind of milk, composed of water, albuminous matter, fat, mucus, cholesterine, sulphates, lactates, and the like, and in which also sugar may be present.

The composition of pus varies considerably; and the conditions under which it varies have not yet received the attention which its importance demands. It seems to vary with the locality whence it comes, and with the circumstances under which it is formed. The pus of a *pulmonary vomica* differs from that of a *psoas abscess*, and that again from the pus of a *mammary* or *hepatic abscess*; and so also the pus of *syphilis* differs from that of *small-pox*. The very large and variable amount of organic elements which pus contains renders it also extremely liable to change; and the products of decomposition of the elements of which pus is composed are extremely various and diffusible—*e. g.*, ammonia, gas products, and salts, leucine and tyrosine; the acids also of the butyric group, as well as formic acid. Another fact to bear in mind is the “spontaneity” with which they (and especially albumen) undergo the process of putrefaction, without any *apparent* co-operation of other matters, and solely by the influence of atmospheric agents; and as constant products of such putrefaction there are always to be found *carbonate* and *butyrate*, and *valerianate of ammonia*, *sulphide of ammonia*, *leucine*, and *tyrosine*. By putrefaction, and under the influence of impure air especially, pus may undergo an acid or alkaline fermentation. The former is rare; but when it does occur, there are developed volatile and fixed fatty acids, such as *butyric acid* and *marginic acid*.

Unhealthy pus more commonly tends to become alkaline without becoming acid. It evolves ammonia and hydrosulphate of ammonia. The corpuscles soften and run together, nuclei disappear, and the whole becomes a mass of granules, as is the case in hospital gangrene (as I have repeatedly observed), which are probably capable of reabsorption.

Pus essentially consisting, as it does, of cells in a pus-serum vary-

ing in size from  $\frac{1}{800}$ th to  $\frac{1}{100}$ th of an inch, has only one way in which its cells can find their way into the blood—namely, by the perforation of a bloodvessel by an ulcer or a puncture. An abscess close to a vessel may open into it; but the result is harmless if the pus is fresh and healthy. The passage of pus into lymphatic vessels is still more easy when such vessels run into open abscesses. But then lymphatics do not empty their lymph into bloodvessels (with one exception) till they have elaborated such lymph in the lymphatic glands; and from the nature of gland-structures it is known that no pus-corpuscle, as such, can pass a lymph-gland. Such glands not only *filter* mechanically, but are living absorbents of some constituents only with which they come in contact, and amongst others, no doubt, the constituents of the pus-fluid without the actual pus-corpuscles, whose debris would simply be retained within the glands. Irritation of these glands leads to proliferation of the gland-cells, and subsequently to the passage into the blood, by the left jugular vein, of cells which cannot be distinguished from pus—the colorless blood-cells. Under such circumstances nothing is easier than to demonstrate what seems to be pus. The presence of white or pus-like cells in the blood, by what we know of blood-formation, can be explained. We know that the blood gets these blood-cells—(1.) After every meal we take; (2.) By irritation of lymphatic and other glands from cachexia; (3.) With the advance of pregnancy and splenic enlargement. In scrofula, typhoid malaria, and cancer, they also abound.

The existence of pus in the blood cannot therefore be demonstrated as the term “pyæmia” has hitherto been understood to mean; and now, therefore, when the term is used, it is meant to imply that an unknown matter, derived apparently from the spontaneous decomposition of some kinds of purulent or albuminoid substances, has mingled with the blood—has poisoned that circulating fluid, or has so altered it that it tends to coagulate in the vessels during life, and has given rise to various secondary phenomena about to be noticed.

“Pyæmia,” as a specific original entity—as a result of absorption of pus, as such—is not now believed in; and any evidence of such an event daily becomes less and less obvious. *Pyæmia* is now rather to be regarded as a collective name for many very different disease-processes, just as the essential phenomena of Bright’s disease are brought about by several different morbid conditions of the kidney. It is especially necessary to distinguish between the following phenomena—namely, (1.) Leucocythæmia; (2.) Embolism, with resolution of clots, putrid decomposition, or gangrene; (3.) Absorption of putrid fluid, without embolism; (4.) Septic endosmosis of gases, independent of embolism.

To these phenomena (excluding those connected with leucocythæmia) Virchow has given the general term of “metastatical dyscrasie;” and amongst them *phlebitis* and *hospital gangrene* are too often fatal, as the result of operations under certain unhygienic conditions.

The phenomena presented by individual cases, capable of being

classed under the head of *metastatical dyscrasie*, are sometimes so extremely varied that names have been given to diseases as well as to lesions which do not always have the same limit or range of significance.

**Phlebitis.**—The first set of phenomena to be considered are those which accompany *phlebitis*. So long as pus-corpuscles, as such, were looked upon as the really noxious material, it was supposed that the tissue of a vein being inflamed, pus would be secreted from its inner wall, just as from a serous membrane. John Hunter has the credit of having suggested this; but he did so merely *as a query*, and subsequent writers adopted the suggestion without further examination and without evidence (see Arnott "On the Effects of Inflammation on Veins," in vol. xv, of *Med.-Chir. Trans.*) Veins are exceedingly slow to inflame. When they do, the inflammation-changes begin in the connective-tissue, towards the outer parts of the vessel, and this even when irritant bodies are introduced into the cavity of the vein itself (see Lee's "Essay," in vol. xxv, of *Med.-Chir. Trans.*) Mr. Lee introduced cotton-wool into a portion of vein emptied of blood. The lining membrane remained unchanged; and the lesions commenced outside the vessel. There the pus formed, and thence the inflammation spread by the connective tissue, and simply by continuity. The generation of secondary multiple abscesses is attended by quite a different process. It was first shown by Cruveilhier that in the (so-called) inflammation of veins a clot of fibrine forms, and is always present, independent of any lesion in the vascular wall; and so he passed to the extreme belief that all inflammation essentially consisted in coagulation of blood in capillary vessels. The first great fact has now been quite substantiated—namely, that long before any visible effects of inflammation occur in the lining membrane of a vein, a clot of fibrine is formed at the part, and in this clot fluid comes to be produced, in all external appearances resembling pus. It must also be remembered that blood, when arrested in the vessels or extravasated out of them, coagulates at the ordinary temperature of the body—98° Fahr. (*Phil. Trans.*, vol. lxxxvi)—a most valuable event in the cure of aneurisms. From this observation as a starting-point, Virchow has developed his very beautiful explanations of the various phenomena connected with phlebitis. Inflammation of veins as the cause of secondary inflammatory phenomena and of multiple abscesses is to be rejected. "Coagulation of the blood in the living vessels"—the formation of a clot or "thrombus" (*thrombosis*)—are the phenomena which attend the formation of multiple abscesses in the phlebitis of pyæmia. The impulsion or projection onwards of a coagulum-clot, or thrombus, or substance detached from the walls or valves of the vascular system, is described under the name of *embolism*.

The coagula may travel in particles or larger masses from the veins to the heart, or from the heart to the arterial peripheric vessels.

Thus, on the one hand, *deposition* of morbid substances in various parts of the body distant from the heart is accounted for; and, on

the other hand, results are explained on simple mechanical principles which hitherto have been obscure. Formerly many of the cases thus explicable would have been recorded as cases of "sudden death," or as "sudden retrocession of gout or rheumatism," or of "gout in the stomach," or "palsy of the heart."

All the cases, fully recorded, which illustrate the phenomena of so-called pyæmia or phlebitis, that I have ever examined, have shown that the affection essentially begins by a real coagulation of the blood at *some definite fixed point*; and this is the most obscure part, and the most difficult to discover, in the history of all the cases. But where this beginning is traceable, the history is exceedingly significant, as pointing to some sources of *local irritation*, which, by simple disturbance of the flow of blood, determines in some way its coagulation in the living vessels. The beautiful experiments of Professor Lister throw much light upon this subject ("Croonian Lectures," *Lancet*, Aug. 8, 1863). Some of the cases mentioned by M. Ribes, in 1825, occurring so far back as 1799, illustrate this point by morbid anatomy. In one case chilblains was the starting-point. Clots formed in the veins, and proceeded up even to the superior vena cava, into the right auricle and ventricle. In one of the most striking cases of this description recently recorded, "a venous clot of fibrine twenty inches long was found in the right auricle and ventricle" (Druitt, *Med. Times and Gazette*, July 19, 1862). It showed such marks on its surface as clearly demonstrated its formation in a vein; and other circumstances pointed to the chief vein of the thigh as the site of the primary formation of this *clot* or *thrombus*. Œdema of the limb prevailed, and the disappearance of the œdema was associated with those sudden cardiac and other symptoms which indicated the passage upwards of this *coagulum* or *thrombus* to the heart, where it was found coiled up in the right auricle and ventricle. In cases of fracture of bone, of amputations, of enlarged glands, of ulcer on the foot (imperfectly healed by scabbing), of open wounds, gunshot passing near vessels, and ulcers, we have great and many sources of irritation. The contiguity of these must be to more or less large veins, or to smaller vessels in or about these sources of irritation and disturbance.

"The immediate cause of pyæmia in any given case is, that some diseased part (which need not be an external wound) so affects the blood circulating through it, that this blood afterwards excites destructive suppuration in parts to which the circulation carries it—namely, commonly first in the lungs, or (in certain cases) liver and lungs, and later generally about the body" (Simon, *Public Health Report* for 1863, p. 60). In a case of which Mr. Simon was cognizant, and which is described by Mr. Bowman, fatal pyæmia in a young gentleman was apparently produced by an ulcerated state of the mitral valve of the heart. In another case Mr. Simon records fatal pyæmia produced by the penetration of pus from a small mesenteric abscess into the thoracic duct; and in the thirteenth volume of the *Pathological Society's Transactions* Dr. Bristow records several instructive cases, in which pyæmia complicated, *ab initio*, cases of *idiopathic necrosis unattended by external wound*. In no case, there-



fore, of swelled legs, enlarged glands, subacute inflammation, or hardening over the course of lymphatics or superficial veins, should the possibility of the mortal accident of *embolism* to the right side of the heart be overlooked; or the possible supervention of pyæmia.

**Hospital Gangrene.**—The influence of foul air, by inducing decomposition, the liberation of absorbable gases and other fluids, also influence the blood passing the part, and bring about not only coagulation in living vessels, but also poisoning of the blood itself, as in hospital gangrene—“*a sloughing phagedena occurring endemically in hospitals.*”

**Cases prone to Hospital Gangrene.**—The disease shows a preference for those cases where cancellous bone-structure has been injured, as in compound fracture, or in the surgical procedures of amputation and resection; and all the more, perhaps, in proportion as the injured bone is large. The other cases in which it is apt to occur are those in which large vein-trunks have been involved in traumatic inflammation (as in gunshot wounds). Although the exact local changes are not well understood, yet very often they seem part and parcel of a process not simply suppurative, but involving also much foulness of wound, putrefactive softening of fibrine and blood-clot, with the evolution of such products as have been already noticed, which, entering the circulation, establish the pyæmic state, and therewith usually more or less of putrid infection of the blood. Many phagedenic and gangrenous materials are inoculable from patient to patient (Thomson's *Lectures on Inflammation*, 1813, p. 484), and are so far *specific* diseases. All the forms of disease which come under the term *metastatical dyscrasiæ* having thus, in common, an intimate affinity with ordinary putrefactive processes, they ought provisionally to be regarded as general diseases, of the class A (see vol. i, p. 176), the respective *contagia* of which may arise in any putrefaction of wound-products; and when such diseases as erysipelas or pyæmia break out in an hospital, it shows that the ventilation is inadequate to remove the *traumatic* or other organic impurities generated in the wards. Thus there prevails an atmosphere which contains much decaying animal matter of the kind which wound-surfaces contain; and any *specific* change arising in this atmosphere, or on any wound-surface which exhales its excreta into the atmosphere, has peculiar chemical facilities for infecting other wound-surfaces within the range of atmospheric influence (see Simon's *Sixth Report on Public Health*, pp. 61, 62). Therefore, in ill-kept hospitals, wounds go on badly. They undergo certain characteristic morbid changes. Erysipelas frequently attacks them. They become to a large extent gangrenous and phagedenic, tending to putrefactions of effused or otherwise stagnant blood, to the re-opening of half-healed arteries and veins, to septic and suppurative infections of the system, generalized under the term *traumatic infection*, all of which are comprehended amongst the phenomena of *metastatical dyscrasiæ*.

Such, pathologically, are some of the causes which may determine the formation of clots either in large or in small vessels. But, after Cruveilhier's observations, the next link in the chain of evi-

dence regarding the nature of *metastatical dyscrasiæ* was established by an arduous worker (then in the Army Medical Department, and since a distinguished Professor in the Royal College of Surgeons of England)—namely, Mr. Gulliver, formerly surgeon of the Guards. He showed that the puriform mass in the interior of clots does not originate in the wall of the vessel or clot, but is produced by transformation of the central layers of the clot—a transformation which may be imitated, as he did, by a chemical process. Sir James McGrigor communicated Mr. Gulliver's observations to the *Medico-Chirurgical Transactions*. Mr. Gulliver examined the clots microscopically, and found that the fluid was not pus; and to show that his observation was intimately connected with the observation of Cruveilhier, I found (by mere accident) that a remark on this point, in Mr. Gulliver's handwriting, exists in one of Cruveilhier's drawings contained in the Library of the Army Medical Department now at Netley.

This is the point from which Virchow starts in his interesting account of this subject—namely, the character of the contents of these clots—a puriform, but not a purulent substance (as Gulliver first showed), composed of granules chiefly. The question, then, immediately suggests itself, "What becomes of them?"

The next set of phenomena characteristic of the phlebitis of pyæmia and the formation of multiple abscesses, are those connected with the softening, disintegration, and breaking up of the thrombi or clots. Virchow was the first to demonstrate the results that ensue. He showed (1847) the embolic characters of certain products previously thought to be inflammatory in their origin (*e. g.*, white fibrine-like masses in the spleen, &c.); and he arrived at the following conclusions: (1.) The occurrence of fibrinous plugs or clots in the pulmonary artery long before death is always secondary, where obstruction is independent of pneumonia, or other changes in the parenchyma. They are apt to arise in any part of the venous vascular system anterior to the lungs in the course of the circulation—*e. g.*, in the veins of the limbs, as in the case described by Druitt, already noticed; in the pelvic veins, as after the operation of ligature for internal piles, whence the clots are carried by the current of the blood to the right side of the heart, and thence into the pulmonary artery, and so to the lungs. Experiments on animals also supported his views. He introduced the pith of the elder tree, as well as animal substances, into the veins, and so produced violent pneumonias, commencing with inflammatory hyperæmia. These localized pneumonias extending, led to rapid deposition in the air-cells of fibrinous exudations, which became purulent, or the portion of lung gangrenous. As these changes advanced in the lung, pleurisy very soon was developed at the periphery—at first producing fibrinous coagulable exudation slowly over the affected portion of the lung; but rapidly, as it progressed towards the other side of the chest, inducing watery hemorrhagic exudation into both serous cavities. The pleura then became gangrenous, and finally gave way to pneumothorax. Such severe lesions may be completed in so short a time as five days. Previously, however, to these im-

portant demonstrations of Virchow, there had appeared the observations of Paget on the obstructions of the branches of the pulmonary artery, and the sudden mode of death to which they gave rise (*Med.-Chir. Trans.* for 1844); but the importance of Virchow's observations consisted in demonstrating the transportation of clots or plugs of fibrine or of blood from different parts of the venous vessels to the heart. Such plugs are known also to be sometimes arrested in the liver, giving rise to so-called metastatic abscess; and while those plugs which find their way to the lungs have their origin in any part of the venous periphery, those which find their way to the liver originate either in the *portal venous system* or in those veins round the *rectum, prostate, vagina, or uterus*, which (communicating alike with the *systemic veins* and with the *inferior mesenteric veins* of the *portal system*) may induce multiple centres of inflammation, and abscesses in the liver as well as in the lung (*Med. Times*, January 18, 1862). Simultaneously with Virchow's, or a little after his observations, we have the observations of the late Dr. Kirkes on "The Detachment of Fibrinous Deposits from the Walls of the Heart" (*Med.-Chir. Trans.*, vol. xxxv, for 1852). Two most instructive cases of this kind have been recently related by Dr. Goodfellow, in which extensive arterial obstruction, gangrene of the lower limbs, and death, followed the separation of cardiac vegetations (*Med.-Chir. Trans.*, vol. xlv). Thus it was gradually proved, that as clots occur in the veins, so they also may occur in the heart's cavities; and next in the history of this interesting subject the connection is established between *peripheral clots* and *cardiac clots*, and how far the softening of these clots or *thrombi* may give rise to the phenomena indiscriminately described as *pyæmia* or *phlebitis*.

Secondary disturbances—not so much by the softened mass as by the detachment of larger pieces, and just so large as to be arrested in vessels more or less remote from the seat of the original clot—are known to occur; and on the advance of clots and their debris from small into large vessels, bits break off and flow on into the stream of blood. Clots in peripheral veins, however small, are thus the sources of great danger. As a rule, they lead to secondary and multiple deposits and abscesses in the lungs; and it is chiefly differences in the size of the capillary vessels which determine their ultimate locality, where they act as any foreign body would. The debris of clots, and large cell-elements from clots, in the mesenteric veins, and from ulcers of intestines, passing through the liver capillaries and proceeding to the lungs, where they are arrested, illustrate this. The lungs have the smallest capillaries of all. They average from  $\frac{1}{100000}$ ths to  $\frac{1}{100000}$ ths of a line (scarcely sufficient to let pass a white cell of blood or of pus, which on an average measures  $\frac{1}{100000}$ ths of a line), whereas the liver capillaries have a much larger range—namely, from  $\frac{1}{100000}$ ths to  $\frac{1}{100000}$ ths of a line.

**Puerperal Fever.**—Virchow regards the metastatic abscesses in the lungs, one and all, as embolic, and founds much on the facts connected with puerperal fever—"a continued fever, communicable by

*contagion, occurring in connection with childbirth, and often associated with extensive local lesions, especially of the uterine system, peritonitis, effusions into serous and synovial cavities, phlebitis, and diffuse suppuration.*" He observed that cases of puerperal fever with multiple abscesses in the lungs had always thrombi or clots in the pelvic vessels; whereas in those cases where the lymphatics only were inflamed there were no multiple secondary abscesses.

The position of embolia in the pulmonary artery induces more or less instantaneous asphyxia. Paget's, Richardson's, and cases recorded by other observers, of more or less sudden death, illustrate this; so also do some cases of gangrene of the lung. If death does not at once occur, the clot in the vessels of the lung becomes broken up. Its fragments pass to the left side of the heart, if not expectorated, and clots are induced there. The phenomena of embolism from the left side of the heart are then apt to be expressed by gangrene of distant parts, as of the limbs, softening of brain, or loss of vision. Gangrene, or decomposition of puriform matter, occurs most actively in the lungs, because here the process is brought most intimately in contact with the outer air; and here, therefore, the influence of impure air becomes so much more injurious; but the cases of arterial embolism developed in this way (*i. e.*, primarily in the lungs) are not as yet known to be numerous.

*The sources of embolism* therefore imply some knowledge of the circumstances under which the blood spontaneously coagulates, or permits the separation of its fibrine to take place in the living vessels. In a pathological point of view the following events in their order may be regarded as the most important: (1.) Local disturbance to circulation; (2.) Stoppage or retardation of the bloodstream—*failure of heart's action*; compression or constriction of the bloodvessels: (3.) Cohesion of red or white blood-cells into a mass, as a result of diseased artery.

The sources of local disturbance are,—(1.) Irritation of the outer coats of the bloodvessels; (2.) Changes which take place in them; (3.) The formation of a clot; (4.) Changes take place in it, and it then furnishes ample materials for further mischief; or (5.) Organization of the clot takes place, when it is converted into connective tissue, and generally remains harmless.

*Arterial Emboli* are observed as a consequence of—(1.) Gangrenous phlebitis of the pulmonary veins or of pulmonary tissue (*rare*); (2.) Organic affections of the aortic or mitral valves on the left side of the heart, fibrinous concretions, and warty excrescences (*common*); Atheromatous disease of the inner membrane of the artery. A first obstruction is generally followed by others—multiplicity and complexity of lesions being one of the characters of the disease. The most frequent sites of embolic arterial thrombi are,—(1.) Arteries in the fissure of Sylvius; (2.) Internal carotids; (3.) Arteries of the lower and upper extremities; (4.) Splenic and renal arteries; (5.) External carotid and mesenteric arteries.

The symptoms in sequence characteristic of the lesion may be noted as follows:—(1.) Valvular disease; (2.) After exertion, there occurs (3.) Palpitation, with the cessation of which (4.) The pulse

disappears in the affected artery below the site of the plug—*e. g.*, in the *radial*—if the plug is arrested in the *brachial*, with pain in the hand of that side. These phenomena are all due to the sudden separation of fibrine from the warty growths on the valves. The particles are suddenly separated, and being carried along with the stream of blood, become fixed in the artery. Recovery may ensue for a time; but (5.) Repeated subsequent attacks occur, each associated with palpitation and irregular action of the heart; and with the cessation of the cardiac symptoms, sudden and simultaneous obstruction of peripheric arteries; (6.) At last gangrene ensues in some parts, and death results (MALMSTEN, of Sweden). In the brain such infarction leads to yellow softening. In the spleen and kidney the infarction consists of a conically-shaped mass of material exactly limited, of a color varying with the size of the lesion, and generally more dense than the surrounding parenchyma (*Med. Times and Gazette*, vol. xvi, p. 278). The obstruction is generally at the point of narrowing of arterial branches immediately after bifurcation (*splenic pencilli*). (7.) Strong pulsation of vessel on cardiac side of occlusion (TUFNELL).

*Venous Embolism* occurs—(1.) When coagula arrive in the right side of the heart. Here they are attended with symptoms of exhaustion, pulse small and intermitting, followed by collapse and powerlessness of muscle. The patient, although inhaling deeply, seems to suffer from apnoea; the veins become highly turgid; and sudden death occurs if large concretions separate and suddenly obstruct the pulmonary artery; as in the case recorded by Druitt, and noticed at p. 661. The symptoms were,—hurried and anxious breathing; pulse rapid and scarcely perceptible; features intensely pale, bluish, and distressed; the whole surface of the body cold, but drenched in perspiration; no pain, but great agitation and feebleness; the air entered the air-cells freely, but the beating of the heart was a confused and feeble “wobble”—its rhythm and force were gone. The intellect was clear.

The diagnosis of embolism generally may be summed up as follows:

I. *Phenomena of Pulmonary Arterial Embolism*: (A.) *Parenchymatous*.—(a.) Collapse of lung; (b.) Peripheral pleuritis of lung; (c.) Hemorrhagic effusion; (d.) Capillary bronchitis, with cough and expectoration if the embolism is capillary. (B.) *Functional*.—(e.) A craving for air (anxietas): although a deep breath may be drawn without pain, yet every movement of the body tends to increase the anxietas; (f.) Lowering of temperature; (g.) Systolic murmur; (h.) Increased impulse of the right side of the heart; (i.) Irregularity of rhythm; (k.) Undulation of veins in the neck; (l.) Cyanosis, vertigo, albuminuria, œdema of limbs. These phenomena are seldom all present, death being too sudden.

II. *Phenomena of Systemic Arterial Embolism*.—These have been already described on the previous page (Simpson, in *London and Edin. Monthly Med. Journal*, 1854, p. 281; *Med. Times*, Feb. 1, 1862, p. 112). The coagula, which exist without simultaneous lesions of the arterial wall, or adjoining capillary circulation, are never formed



on the spot, but have been separated from some distant point in the circulation, and have been carried by the current of the blood as far as they could go. Such are genuine emboli. They are always found in places where a large arterial trunk, by giving off branches, suddenly acquires a more constricted calibre. Virchow, Paget, Malmsten, and Kirkes have shown independently of each other that—(1.) Fibrinous concretions may separate from the valves of the heart during life; (2.) That they may cause obstructions of particular peripheric vessels; (3.) That by admixture with blood they may have a poisonous decomposing influence similar to typhus or pyæmic poisons. In the lungs, for example, apoplectic foci become developed. Infarctions occur, and their results, in broad masses of fibrine, remain. On the arterial side of the circulation metastatic fibrinous wedges occur in the vessels; petechial spots occur on the skin and on mucous membrane, pericardium, and peritoneum. Similar fibrinous deposits accumulate in the spleen.

III. *Phenomena of Venous Embolism*.—The coagula found in the veins are the products of local stasis, often caused by roughness of their inner surface, by alteration of structure and relaxation of elementary parts. When the coagulum adheres only to one wall, with the effect of narrowing the passage, such a thrombus is never of an embolic nature. It is a clot, plug, or coagulum of fibrine formed or forming there as a result of local causes of irritation (see previous description).

IV. *Phenomena of Pigmental Embolism*.—A peculiar form of embolism is associated with accumulations of pigment in the blood. It is primarily developed in the spleen, whence the thrombi are conveyed to the *vena portæ*, gradually increasing in circumference. Malarious fevers establish such pigments in the blood (PLANER of Vienna). In such cases sanguineous extravasations, in great numbers, are found in the brain; and abscesses in the liver: laceration of hyperæmic capillaries is also a result—*e. g.*, Malpighian glomeruli of kidneys. [See Malarial Toxæmia, vol. i, p. 462.]

[Mr. W. S. Savory has studied experimentally the “Local Effects of Blood-poisoning in relation to Embolism” (*St. Bartholomew’s Hospital Reports*, vol. i, 1865). He first injected disintegrated fibrine, oxide of zinc, &c., into the veins of dogs and rabbits, the animals being killed subsequently, and the several organs examined; in another series of experiments he injected carefully filtered fluid, rendered putrid by the maceration therein of flesh, or putrid liquor amnii; and in a third series, fresh pus. The results were substantially the same, and Mr. Savory thinks he is justified in concluding:—

That patches of congestion and stagnation, with perhaps ecchymosis in the lungs and other organs, may be produced by the injection into the veins,—(1.) Of minute particles of solid matter suspended in water, which can undergo in the blood no decomposition or induce any chemical change, and consequently can only act mechanically. (2.) Of putrid fluids which have been previously carefully filtered, and so deprived of any solid particles. (3.) Of pus. That in either case the local effects are the same. The action of fresh pus in producing these effects is mechanical. Either its cells, or the clots which it may form in the blood, become impacted in

the capillaries. When putrid it no doubt acts in both modes. It has no peculiar or specific action when present in the blood. If putrid it will act as putrid matters generally do. Or its globules may, under certain circumstances like other foreign particles, become arrested in, and block up, the capillaries. In these experiments abscesses were not more readily induced by the injection of pus than of simple fluids, or even of solid particles.

The causes of the local congestions and suppurations in pyæmia are thus classified :

**Stasis due to mechanical action.** A blockade produced by the impaction of solid particles. According to their size :

**Arterial embolism.** By fragments too large to pass through the smaller arteries.

**Capillary embolism.** By fragments small enough to pass into the capillaries.

**Stasis due to change in the blood produced by the admixture of morbid fluid.** The local effect of blood-poisoning properly so called.

**Capillary obstruction.**

**Stasis due to a combination of the two above-mentioned causes.**

In all cases stagnation and congestion first ensue. The subsequent changes, whether towards resolution, suppuration, or gangrene, are determined by—

The action of the morbid fluid, or obstructing substance.

The changes it provokes.

The constitution and state of health of the individual.

Experiments upon animals and clinical observation are uniformly parallel in their results up to the production of the local effects,—the spots and patches of stagnation and congestion. The worst cases, those in which death is most rapid, reveal afterwards the least signs of local disease. There is no time for the local effects to supervene; the whole mass of the blood is so poisoned and spoiled, that it kills outright. It is in the milder cases only that they have time to develop, and pass through their subsequent changes.

These local effects seen in the lungs, or any other organ, both in experiments and clinical observations, are, in the first instance, spots or patches of congestion, more or less intense; the spots are, for the most part, well defined and regular in shape, with usually a circular outline upon the surface of the organ. They are of a dark red or livid color, sometimes slightly raised, and often indurated. In the larger patches these characters are usually less marked; they are less defined, and of a more dusky hue. These spots and patches, after remaining awhile, may either clear up, and gradually disappear, or may pass on into suppuration. In the latter case they gradually soften towards the centre, until a puriform fluid oozes out when a section is compressed; afterwards there is a distinct collection of pus in the centre, with a halo of livid indurated substance, gradually passing outwards into healthy tissue. In the same organ after death all the stages may be seen. These purulent collections at length become more concrete at the circumference, while towards the centre they are still fluid. Finally they may become caseous throughout and resemble tubercle; and, if life lasts, pass through still further degenerative changes.

The pathogeny of pyæmia has also lately been investigated by Dr. J. J. Woodward, U. S. Army. The results of these observations were com-

municated to the American Medical Association at their meeting, June, 1866.

This distinguished pathologist remarks:

“Careful examination of the secondary foci has failed to show me emboli impacted in the arterial twig, by which the region of the focus is supplied. On the contrary, the vessel continues generally quite patulous even after it is involved in the substance of the morbid nodule. This I have observed in a case of foci in the lungs consecutive to syphilitic supuration of the tibia, as well as in a number of similar cases due to wounds and amputations. I have investigated the branches of the portal vein leading into similar foci in the liver in cases of ulcerative dysentery, and with like results. On the other hand, it has frequently occurred to me to see the most diverse coagula in the veins leading from an amputated limb, where death has happened from other causes, without any pyæmic symptoms, and where the most critical examination of the body has failed to discern any secondary foci. As far as my own personal observations have gone, pyæmic phenomena have been invariably connected with the primary occurrence of local septic processes. I do not mean to lay this down as an unchangeable law; my observations have not been as yet sufficiently numerous to make me positive of more than that this is, at least, an important and frequent group of cases. In cases of wounds, the local conditions brought under my notice were, chiefly, sloughing of the edges of the wound, and the peculiar gangrene of the marrow which has so generally been miscalled osteomyelitis. . . . . The first phenomena noted are the peculiar coagulation of the blood in the bloodvessels, and the granular aspect of the tissues under the microscope, which show that the part is already dead and that decomposition has begun. The subsequent changes are purely chemical, and the normal elements become less and less recognizable amidst the host of actively moving molecules set free by the putrefactive alteration. . . . . In cases which prove fatal by inducing pyæmia, no line of demarcation, no barrier of inflammation limits the gangrenous portion; and the veins leading from the affected bone are usually full of coagula, which have entered into a form of putrefaction quite similar to that going on in the marrow. The ultimate result of this change is a yellowish or greenish-yellow fetid fluid, in which the microscope recognizes nothing but actively moving molecules with bright centres and dark borders. I have once or twice seen the veins leading from the flaps of a sloughing stump in a similar condition; but, in most of the cases to which my attention was drawn, the veins affected proceeded from the diseased bone itself. The putrefactive change going on in the marrow is transmitted through the coagulated blood-mass in the veins by actual continuity.

“The femoral vein, the saphena, and their branches, were sometimes stuffed with thrombi throughout a part of their extent, but in all the cases I found the veins from the bone, filled with their fetid contents, were clearly traceable to the point where the trunk with which they were connected discharged into the femoral, or one of the larger branches, with no thrombi intervening on the cardiac side. The granular yellowish fluid could sometimes be traced some little distance towards the heart. In short, there was clear anatomical proof of the introduction into the torrent of the circulation of the putrefying debris of the coagula which had formed in the veins leading from the affected bone. As for the detaching of larger fragments which should deserve the name of emboli, I found no actual anatomical proof, though abundant possibility

for such an occurrence existed. What was to be seen discharging into the femoral vein, was simply the putrefying liquid described, a liquid more viscid than blood, which could not be expected to circulate as readily as blood, and which might readily, I admit, although I saw nothing of the sort, carry with it more coherent fragments of the involved coagulum, but which, in any case, might be expected to be arrested in the capillaries of the lungs, and if so arrested, to set up there by actual contact, a similar form of change. It is also easier to conceive how a part of such a viscid, putrid fluid, which had passed through the capillaries of the lungs, might subsequently be arrested in the systemic capillaries, than it is to account for the frequent occurrence of *fever* in the liver and kidneys after amputations of the thigh, on the supposition of solid emboli.

"I do not offer this summary of my own observations as a solution of the question of pyæmia; but rather as a statement of facts, difficult to account for on the supposition of emboli, impossible to account for on the supposition of suppurative phlebitis, which must yet be fully embraced by any satisfactory explanation of the disease. The general series of the phenomena here sketched, as observed in connection with wounds, perfectly accords with what I have been able to observe in puerperal pyæmia, and in the pyæmia connected with dysentery. In the inflamed womb of the first group of cases, and the colon ulcers of the second, all the autopsies I have witnessed or made, demonstrated the existence of gangrenous or phagedenic, that is, septic, processes in connection with the local lesion. Without insisting upon any exclusiveness for the opinions which must necessarily spring from such cases, it is difficult to avoid the conclusion that local septic processes have a significant connection with the genesis of pyæmia, and that viscid septic liquids, derived from the degenerating primary coagulum, may at times play the part which Virchow ascribes only to solid emboli. These considerations assume still more importance when an attempt is made to arrive at some definite notion with regard to the *causes* of pyæmia.

"Truly it must be admitted that our knowledge of these causes is yet more unsatisfactory than our knowledge of the pathology of the disease. It is well known, however, that the conditions, under which it must certainly develop itself, are those under which almost all zymotic diseases attain their greatest malignancy; and it will be readily understood, if the views I have hinted are correct, that whatever favors the occurrence of septic processes in the local lesion may become a cause of pyæmia. Hence, it is in crowded hospitals, where ventilation, cleanliness, and diet are comparatively neglected, and among patients debilitated and disordered by previous exposure and privations, that pyæmia secures most victims, whether among wounded soldiers or parturient women. Under the same circumstances, erysipelas, hospital gangrene, and sloughing of wounds and sores, become common—very often indeed appear to stand in direct genetic connection to the subsequent pyæmia. Whether besides such hygienic conditions, to which, in modern times, attention has been most exclusively directed, there lurk yet other and more obscure momenta; whether, above all, as has been especially claimed by the obstetricians, there is a peculiar, pyæmic, epidemic influence, which exists at certain times and places, and is absent at others, is a question which can be first intelligently approached, when an epidemic of pyæmia shall have been observed under circumstances which completely fulfil every well-known hygienic requirement. Until then, to insist upon this aspect of the question, tends to encourage and excuse the neglect of conditions whose influence is beyond question."



Mr. Savory (*St. Bartholomew's Hospital Reports*, vol. ii, 1866) remarks: "The history of our knowledge of pyæmia is an instructive one. Although the ancient idea that the affection termed pyæmia depends on the presence of pus in the blood, is no longer generally received, yet it still continues to influence largely the views which are taken of its pathology. That the disease is still associated with what was called phlebitis, is due to the old idea that in this way pus is introduced into the circulation. It seems to be a great step onwards to recognize the fact that the disease may occur independently of the presence of pus, or of any affection of the veins whatever; that it is not due to any specific or peculiar matter formed in this or that particular part, but that it is the effect of blood-poisoning, due to the introduction into the circulation of morbid or putrid matter. No doubt, pyæmia is often associated with the formation of clots in certain veins, but this coincidence by no means implies the relation of cause and effect. When veins are found occupied by disintegrated clot after death from pyæmia, it has still to be shown that this is the cause of the disease. On the contrary, it can be shown, so far indeed as the most careful and critical examination can go, that pyæmia may occur independently of any affection of the veins whatever. And it is a question whether, in those cases in which the veins are plugged or inflamed, thrombosis and phlebitis are not the local, and pyæmia the general, effect of the same cause. There is no more evidence of any especial relation between pus and pyæmia than this, that pyæmia is the result of the passage of putrid fluid into the circulation, and that pus is of very common occurrence, and liable, like other animal fluids, to become putrid."

Mr. Savory, after an examination of our present knowledge of pyæmia, makes the following conclusions:

"Thrombosis may exist without any evidence of phlebitis, and very often occurs without being followed by pyæmia.

"Phlebitis may occasionally exist without thrombosis, and often occurs without being followed by pyæmia.

"Pyæmia often exists without any evidence of thrombosis or phlebitis; still oftener it happens without any evidence whatever that it has been preceded by either of these, or of any other affection of the veins."

It has, therefore, not been satisfactorily shown that either phlebitis or thrombosis stand, in any especial or peculiar manner, in relation to pyæmia, as cause and effect.]

From what has been written, it is therefore necessary to distinguish at least *three* forms of *metastatical dyscrasie*—namely, (1.) *Metastasis associated with the phenomena of embolism, tending to the development of multiple centres of inflammation, and their suppuration*; (2.) *Metastasis in which there is something over and above embolism, and quite independent of its occurrence, and which may get well if secondary abscesses do not form in important viscera—septicæmia, ichoræmia* (S. RINGER); (3.) *Metastasis combining the phenomena of (1.) and (2.)—pyæmia.*

In the first class of cases death generally ensues from exhaustion; and when the multiple centres are in process of evolution, the febrile phenomena are always increased—a distinct attack, with rigor, being excited and associated with each centre of development. Evening exacerbations of fever are distinctly marked, so that the



ranges of temperature during the course of the disease are similar to those of extensive suppuration attended with fever. The temperature, however, in some of these cases is not very high; but in both forms of metastasis mentioned the type of the fever is intermittent during the day (S. RINGER, BILROTH)—i. e., the temperature between the hours of elevation becomes normal; and Professor S. Ringer writes that he has never met with any disease in which this occurs except *pyæmia*. It is no doubt due to this circumstance being hitherto overlooked that such varied statements are current regarding the heat of the skin in *pyæmia*. In some cases, indeed, the temperature is never very high, nor the intermittent elevations very numerous, yet these are the kind of cases in which the internal organs are apt to be studded with secondary abscesses.

In the second class of cases of *metastasis*, in which intense febrile phenomena occur, quite independent of embolism, the temperature is very high, the intermittent elevations are numerous, and frequently there are no multiple centres of inflammation in visceral parts (RINGER). Though the exact chemical change in the composition of the blood has not been made out, yet, as it has been produced experimentally by the injection of filtered putrid fluids into the veins or intestinal canal, it may be fairly concluded that such cases are due to the absorption of putrid poisons in a gaseous or other fluid form, especially resulting from the decomposition of tissues. Tissue-change or metamorphoses in the febrile state may also establish sources of blood-poisoning (e. g., "Rheumatism"). Dr. Parkes states how the amount of urea rose in a case of *pyæmia* after amputation, from 3.1 for every lb. of body-weight to 4.26 grains. The weight of the body was 131 lbs. It fell to 110 lbs. in eleven days, and the temperature rose from 101° Fahr. to 105.6° (*On the Urine*, p. 178).

In surgical cases the phenomena commence to be expressed between the second and fourth day after an operation or injury. The tissue round the source of the mischief becomes red and œdematous towards the third day; and *ichor* may exude from the injured part, mixed with bubbles of gas. Disintegration spreads with great rapidity from the seat of injury; and where the part is to-day simply *œdematous*, to-morrow it will be an *ichorous* infiltration; and so it proceeds till death occurs. In some cases it may be looked upon as a progressive gangrene of areolar tissue (the acute purulent œdema of Perigoff). If thrombi form in the veins, and the phenomena of embolism occur, the danger is increased, and the case is more complex; but the *septic* phenomena, in the first instance, are quite independent of embolism. A very high temperature and a more continued fever are characteristic of *septicæmia*. The pulse rises (generally above 120), and the intense burning heat communicated by the skin of the patient is well known to the hand of the experienced surgeon. On the evening of the fourth day after an injury, Bilroth records the temperature at 37.3° C. — 99.5° Fahr. In the night the œdematous infiltration commenced, and on the ensuing morning the temperature was 40.1° C. — 105.8° Fahr. On the

eighth day the patient died, up to which time the temperature remained high, its lowest point being  $39^{\circ}\text{C.} = 102.2^{\circ}\text{Fahr.}$  A rapid and considerable increase of temperature on the *third* or *fourth* day, with delirium and a somnolent state, with an ichorous appearance of a wound, and œdematous infiltration around it, are certain signs of septicæmia. Albumen occurs in the urine in some cases, and sweats are common (BILROTH); the skin acquires a faint yellow color; and the breath a hay-like smell. The highest temperature recorded by Professor S. Ringer is  $107.4^{\circ}$  (*MS. Notes*). Some of these cases undoubtedly get well, although a very large proportion die; and the chances of getting well are greater in proportion to the freedom from embolism, and from intense febrile disturbance as indicated by temperature, and from the formation of secondary abscesses. Dr. Gillespie and Mr. Spence, Professor of Surgery in the University of Edinburgh, have recorded undoubted instances of recovery from cases of this kind; so also Mr. Prescott Hewett recounts ten recoveries within his experience.

**Treatment.**—The indications are—(1.) To remove all those conditions which favor *thrombosis* and the degeneration, metamorphoses, or putrefaction of blood-clots or tissue; (2.) To subdue the force of the circulating current, so as to prevent excitement during the long and slow metamorphosis of a clot when it has formed; (3.) To sustain the strength and allay the nervous irritability. Wine, brandy, rum, milk, strong animal soups, should be freely given from time to time, in small quantities. Opium should be given freely at night.

[Prof. Polli, of Milan, has proposed to treat pyæmia by the alkaline sulphites—the compounds of sulphurous acid with soda, potash, magnesia, and lime. Assuming that septic poisons introduced into blood poison it by acting as a ferment; satisfied, after an elaborate experimental investigation, that the sulphites have a remarkable power in arresting putrefactive changes, and counteracting the poisonous workings of putrid blood, pus, &c.; and having proved that they might be taken in large doses into the stomach without inconvenience or damage—being rapidly absorbed, diffused throughout the system, and eliminated without having undergone any chemical change, or at most a very slight one—Prof. Polli believed that in them he had discovered a valuable remedy against all septicæmic disorders. He proposed to give the sulphites in doses varying from twenty grains to a drachm, three or four times daily. Clinical experience has not, however, proved the value of this treatment. The writer has fairly tried it without any happy results; Dr. J. S. Bristowe (*Art. Pyæmia*, Reynolds' *System of Medicine*, vol. i, p. 225), "that in the practice of Mr. Simon, at St. Thomas's Hospital, it has entirely failed;" and Dr. George A. Otis, U. S. A., in his Surgical Report (*Circular No. 6, Surgeon-General's Office, War Department, 1865*), speaking of the army statistical reports on the treatment of pyæmia, states, that their "conclusions are adverse to the therapeutic utility of the sulphites and hyposulphites in this disease."]

Free exposure of the patients to currents of fresh air—treatment in the open air, in fact—has been shown by Mr. Paget to be

the most promising of success in cases of pyæmia,—by far the most fatal of all *traumatic infections*, which kills nearly all whom it attacks.

### [CHRONIC PYÆMIA.

(DR. CLYMER.)

It is not uncommon to meet with cases, more frequently happening in the course of diseases than following surgical injuries, which present all the essential characters of pyæmia, as ordinarily described, but are much slower in progress, and less severe and perilous. To these cases Mr. James Paget proposes to give the name of *chronic* or *relapsing* pyæmia. He has called the attention of the profession to the subject in a paper published in the first volume of St. Bartholomew's Hospital Reports.\* Chronic pyæmia is not referred to in any of the Systems of medicine or surgery, yet it resembles the typical pyæmia in the formation of widely dispersed, shapeless, collections of pus; in the probability that these formations are due to some infection of the blood by the entrance of diseased, or septic, products; and, often, in the occurrence of rigors, profuse sweatings, phlebitis, and inflammations of joints. It differs from the acute type in that its course extends, continuously or with relapses, over many weeks or months, and is often free, at least, in its later stages, from all severe general disturbance of the health, and from nearly all risk of life.

Mr. Paget believes that the difference between groups of cases of acute and of chronic pyæmia is not greater than those between cases of acute and chronic tuberculosis. Sameness of designation is, he thinks, in both cases alike justified by the rule, that differences in degree do not constitute or prove difference in kind.

The local evidences of chronic, are more often than those of acute pyæmia, seated exclusively, or chiefly, in different parts of the same tissues; they are more frequent in the trunk and limbs than in internal organs; and when seated in the veins are most frequently found towards the close of the disease.

The nearest affinities of chronic pyæmia are with rheumatism, through gonorrhœal or urethral rheumatism (vol. ii, p. 66); with simple or single abscess-formation after fever; and with hectic fever. Yet with very rare exceptions, the diagnosis from all these is, in practice, clear.

The **Prognosis** is usually favorable, especially when there are long intervals between the successive local manifestations of disease, and no evidence of serious pulmonary disorder. The more natural the pulse and breathing, and the less the sweatings, the greater, as a rule, are the probabilities of getting well.

The **Treatment** is good food in this disorder, careful nursing, abundance of fresh air, and a moderate use of stimulants and tonics. Mr. Paget thinks that the influence of the liquor potassæ is worthy of consideration.]

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\* [Cases of Chronic Pyæmia. By James Paget, F.R.S., St. Bartholomew's Hospital Reports, vol. i, London: 1865.]

## CHAPTER X.

## DISEASES OF THE RESPIRATORY SYSTEM.

## LUNG DISEASES.

THE acute inflammatory diseases of the pulmonary apparatus are more or less distinctly defined according to the tissue which they implicate and the symptoms to which they give rise. The three structures which mainly take part in the constitution of the lung-substance being—(1.) The *bronchial tubes*, terminating in (2.) The *pulmonary air-cells, vesicular structure, proper substance, or parenchyma of the lung*; and (3.) The *membrane covering this parenchymatous part*, forming a portion of that serous sac interposed between the lungs and the walls of the thorax. That portion of the serous sac which immediately invests the lungs is known by the name of the *pulmonary pleura*, while that which is applied against and invests the parietes of the thorax is known as the *parietal or costal pleura*.

One or more of those pulmonary structures may be associated in the processes and results of inflammation. Thus the bronchial membrane may be inflamed, when the disease is termed *bronchitis*; or the substance of the lung may be inflamed, the disease being then called *pneumonia*; or the pleura may be inflamed—a condition which is described as *pleuritis*. It is rare, however, in practice to find that these morbid states are so completely isolated. More frequently, for instance, with *pneumonia*, or inflammation of the substance of the lung, there is associated more or less inflammation of the air-tubes (*bronchitis*), on the one hand, constituting *broncho-pneumonia*; or there coexists inflammation of the investing pleura (*pleuritis*), constituting, on the other hand, *pleuro-pneumonia*. The physician can now distinguish each of these elementary conditions by definite symptoms; and by observing the combination of physical phenomena, their association with general symptoms, and the sequence of their occurrence, he is able to determine how far any given pneumonic affection involves one or more of the structures which compose the lung; and to direct the treatment of the case accordingly. It is therefore necessary to describe the phenomena of inflammation in each of these structures in detail.

## PLEURISY.

LATIN Eq., *Pleuritis*; FRENCH Eq., *Pleurésie*; GERMAN Eq., *Pleuritis*—Syn., *Rippenfellent-zündung*; ITALIAN Eq., *Pleuritide*.

**Definition.**—*Inflammation of the serous membrane that lines the cavity and covers the viscera of the thorax. It is characterized at its outset by a febrile chill, followed by an acute sharp pain in some part of the chest,*

frequently called "a stitch in the side," as it is usually confined to one spot about the lateral regions of the thorax. The acts of respiration are performed rapidly, and are not completed. A dry short cough supervenes, and the pulse is hard and quick. The natural serous secretion of the pleural sac is arrested in the first instance, but soon becomes increased in quantity, and of an inflammatory type, the exudation tending to assume the corpuscular character. The effusion more or less rapidly increases, and may ultimately assume a sero-purulent character; and the parietes of the corresponding side of the chest may dilate accordingly.

**Pathology and Morbid Anatomy.**—The inflammatory phenomena begin in the sub-pleural tissue, whose vessels enlarge and admit red blood, and shortly afterwards the red blood penetrates the web of the pleura itself, and the process is more or less diffuse. At first a number of red dots may be visible, which at length are so multiplied as to become confluent and form large patches, which spread till perhaps the whole of the *pleura pulmonalis and costalis* is in one continuous state of inflammation. The membrane is in all cases of a bright red or arterial color, slightly thickened from interstitial deposit, and easily detached, from the diminished cohesions of the sub-pleural fibrous tissue.

If the diffuse inflammation be of any intensity, the secretion from its surface is in general suspended at first, and the membrane is dry. In this state the inflammation may terminate by resolution, or serum may be poured out, forming the *serous inflammatory effusion*. The quantity of serum thus effused is extremely various. In some cases it hardly exceeds a very few ounces, while in other instances it amounts to many pints, separating the usually opposed surfaces of the membrane, and distending the cavity of the pleura, and compressing the lung. Laennec is of opinion that the time of effusion after the commencement of the inflammation is often very short, as he has detected ægophony and absence of respiration, as well as of thoracic resonance, an hour after the patient has first felt pain in the side. If the effusion be considerable, the lung becomes collapsed, contains no air, and therefore no longer crepitates; the vessels are devoid of blood, while the bronchi, even to the large trunks, are evidently contracted; still, if this lung be inflated, it enlarges more or less perfectly. Again, should the pleuritic effusion be less in quantity, some fluid appears spread all over the lung; but the greater quantity is collected at the lower portions of the chest.

Accompanying either of the previous forms, or existing *per se*, the *fibrinous inflammatory lymph* may predominate, and adhesion of the opposed surfaces ensue. In many cases the lymph is loose and watery, rendering the serum turbid or flocculent; but in other cases it is more solid, and adheres with great tenacity to the opposite membrane, becoming organized at both surfaces. The organization of these membranes is rapid, and is often effected in the course of forty-eight or even twenty-four hours. If the patient dies shortly after an attack of acute inflammation, these adhesions are found soft, easily lacerable, and extensible. If, however, he survives a



longer period, the adhesions are often of great tenacity, are indurated, and with difficulty separated from their attachments. The extent of membrane affected with adhesions is sometimes limited to a small portion, and sometimes extends over the whole surfaces of the cavity; but their most common seat is over the anterior lobes of the lungs, or the portion of pleura from the mamma to the axilla.

**Empyema.**—The pleuritic inflammation sometimes terminates in suppuration; and should the pus be in such quantity as to accumulate in the cavity of the chest, the disease is termed *empyema*. Empyema may be true or false; it is said to be true when the pus is secreted by the pleura, and false when it results from the bursting of an abscess of the lung into the cavity of the chest. The quality of the pus in true empyema varies from a genuine laudable pus to a sero-purulent fluid. In quantity, also, it varies from a few ounces to many quarts, filling the entire cavity of the chest. Under these latter circumstances the side of the chest is dilated, and the intercostal spaces are widely separated and bulging.

Effusion of pus may take place into either cavity of the chest, but the left, perhaps, is the more common. The phenomena accompanying empyema of the left side are remarkable; for, besides the lung being found collapsed, and not so big as the fist, the heart is sometimes seen transposed as far over on the left side as it usually is on the right. In cases, however, in which *paracentesis* has been performed, and the pus has been drawn off, the heart is observed to return to its place, while the lung, less completely collapsed, may be bound down to the upper and lower portion of the chest by long and multiple adhesions.

**General Symptoms of Pleurisy.**—Like other inflammations of the lungs, pleurisy may be acute or chronic. The acute form of this disease may be preceded by fever, but often no such antecedent is present. Its local symptoms, however; in most cases are strongly marked, the patient suffering with severe *continued pain* in the affected side, of “a dragging, shooting character,” which is greatly exasperated by coughing or forced inspirations, movement, pressure, and percussion, so that the lungs can only be imperfectly filled with air. The seat of the pain, however extensive the inflammation, is generally limited to one point; and this point is usually about the centre of the mamma, or just below that part, towards the lateral attachments of the diaphragm. While the pain is constant, it nevertheless sometimes remits, and with the occurrence of effusion often totally disappears. The tongue is commonly white, but the pulse varies, perhaps according to the form of the inflammation and its intensity. If the disease be limited to an effusion of lymph or serum, the pulse is seldom more than 90 to 110, but “hard and concentrated in impulse.” Either form of *pleurisy* is generally accompanied by a short, troublesome cough and some expectoration. The respirations are increased in frequency—phenomena of more constant occurrence than even the local pain—and, unless dyspnoea exist, are unnoticed by the patient. While one *respiration* is performed, only *three* beats of the heart, in place of *five* or *six*, occur. The patient likewise is

for the most part restless, and lies on the affected side; but if the effusion is great, the lung is compressed, which increases the general uneasiness, as well as the oppression of the breathing; and the patient, instead of lying on his side, now lies on his back, or sits propped up in bed. If he recovers, the fluid effused is absorbed with greater or less rapidity, and his amendment is proportionally retarded or accelerated. In fatal cases, although the lung may for a time become accustomed to the altered state of things in the chest, yet fresh effusions occur, which shortly terminate the life of the patient.

The duration of the disease is such that acute pleurisy sometimes terminates in a few hours, sometimes in a week or ten days, while cases have been met with in which many months have elapsed before the pleuritic effusion has been absorbed and the patient restored to health.

**Symptoms of Empyema.**—Again, if the inflammation is about to issue in the formation of pus, the pulse is extremely small and frequent (from 120 to 150), while the restlessness and anxiety of the patient are greatly increased. There are cases, however, of empyema in which the patient suffers little pain, or any more marked symptom than usually awaits the last stages of phthisis. In some instances he is for a time even capable of walking about the ward of an hospital or in a bed-room. Supposing, however, empyema to have occurred, any acute pain which may have existed subsides, but the anxiety of the patient is increased, and his state of collapse shows his imminent danger. If the constitution be less affected, the symptoms vary according to the side of the chest which is the seat of the empyema. If it be on the left side, for example, the heart is often transposed, and felt beating as far over on the right side as it usually does on the left, and the pulse is small and frequent. If we now bare the chest of the patient, we find the affected side enlarged, sometimes œdematous, with projecting intercostal spaces. As the lung is now greatly compressed, no respiratory action is seen on that side, which is entirely at rest. If *paracentesis* be now performed, the heart is restored to its place as the pus flows; but as the lung for the most part only imperfectly expands, the affected side, even in the most favorable cases, contracts, and the spinal column, pressed upon by an unequal weight, acquires a lateral curvature, the shoulder sinks, and the patient is greatly and permanently deformed.

Auscultation and percussion are equally valuable in determining the amount of effusion. If serum or pus be effused to the amount of a pint, for instance, the lung is displaced to that extent; and consequently the lower portion of the chest, when struck, returns a dull sound, which extends as high as the level of the fluid. If we now auscultate the patient, the respiration is also lost below the level of the fluid. Besides these results, the voice gives very striking indications of the lung becoming so far condensed from the pressure of the fluids; for we very constantly have *bronchophony*, and occasionally *argophony*.

If the chest be completely filled in empyema, the respiratory

sound is altogether wanting; so is *ægophony* and *bronchophony*, and the containing cavity returns a dull sound at whatever part percussed. Under these circumstances, and especially if the heart be displaced, the affected side will be seen entirely motionless, rounded, and distended; and when these signs are present, there can be no doubt that the distension is due to the presence of serous effusion or pus. But the physical signs of pleurisy are much more delicately varied according to the anatomical conditions of the disease. For example, at the most early stage, when the serous secretion is at first arrested, the expansion of the walls of the chest is diminished, as may be proved by measurement. The percussion-sound, however, is not perceptibly altered, and respiration is weak, because imperfectly performed. The characteristic *friction-sound* of inflamed serous membranes may, perhaps, now be detected, if listened for in the *infra-mammary* or *infra-axillary* regions. When the secretion has returned, increased in quantity, the signs continue as described, but the clearness of the percussion-sound becomes diminished, and the *friction-sound* is of a rubbing or grating character. The period of inflammatory effusion is now established, and the *infra-mammary* and *infra-axillary* regions become more or less bulging; the projection of the intercostal spaces of the affected side during both respiratory acts becomes most obvious; the thoracic vibration from the voice is abolished where the fluid intervenes, and so also are friction-murmurs there. The area of dulness, and of the peculiar sounds, may be changed by altering the position of the patient. The natural respiratory murmurs become greatly intensified above the level of the effusion. When the effusion exists on the right side, the sounds of the heart are more clearly audible than in the natural state in the right axillary region, because the lung is more solidified by the pressure of the effused fluid.

Such are the more salient symptoms which mark the progress of pleurisy. The disease, however, may exist without any of the general symptoms. There may be neither local pain, cough, dyspnoea, nor febrile action, and yet effusion may have occurred to such an extent as to have reached the clavicle, while the patient remains utterly unaware that his chest was the seat of disease (WALSHE). The physical signs alone reveal the disorder—which is termed *latent pleurisy*—a form of disease which had no existence in nosology previous to the time of Laennec. Whenever, therefore, the least suspicion exists of disease in the chest, especially in elderly persons, or those liable to constitutional affections, percussion and auscultation must never be neglected.

**Causes.**—The causes of pleurisy have an influence over its course; therefore a due appreciation of them is essential to a correct prognosis and treatment. The inflammation is sometimes said to acknowledge an idiopathic origin, as when the pleurisy is believed to arise from exposure to cold; but it usually results during the progress of some *specific* or *constitutional* disease. Exposure to cold, especially to currents of air, when the person is heated, is a frequent exciting cause; but many now express their entire disbelief in cold being able to establish pleurisy in a healthy person (HYDE SALTER,

FULLER). When exposure to cold is followed by inflammation of the pleura, the disease is most likely associated with some morbid condition of the blood; and without doubt blood-poisoning is one of the commonest morbid causes of serous inflammations. For example, *anæmia*, *pyæmia*, the specific toxæmia of *eruptive fevers*, the *materies morbi* of *rheumatism*, *gout*, *scrofula* (*tubercular pleurisy*), *carcinoma*, *Bright's disease*, *alcoholism*, *syphilis*, or even *retained excreta*, with *pyrexia* as the result of *overfeeding*, are all apt to set up serous inflammation. All serous inflammations, in truth, especially point to states of blood-poisoning; as during the progress of typhus or puerperal fever, or during Bright's disease, or the pleurisy may acknowledge some adjacent irritation, as *pneumonia*, constituting *pleural pneumonia*, or the irritation of some growth in the lungs, such as *tubercle* or *cancer*.

**Prognosis.**—Simple idiopathic pleurisy on one side of the chest, occurring in a person whose lungs are not chronically diseased, almost always terminates favorably, if taken in time and treated judiciously and with energy, and if the effusion has not been copious; but when it occurs as a complication in other diseases, the result may be doubtful, especially if air finds its way into the cavity (*pneumothorax*).

**Treatment of Pleurisy.**—In acute pleurisy, during the first stage, or that of *hyperæmia*, the best practitioners of all times and of all countries have taken blood from the arm, provided the strength be good and the symptoms sthenic; and if, says Laennec, after one or two bleedings, the pain in the side and fever have not abated, blood should be taken from the side by leeches or by cupping. The practitioner should also remember that effusion often takes place after bleeding, during the subsidence of the inflammation, so that the breathing is often more oppressed, and the symptoms for a time aggravated, although the condition of the patient is in reality improved. The lung, however, soon gets accustomed to this new state of things; and the fluid in a few hours beginning to be absorbed, the symptoms are generally ameliorated. Hot poultices and leeches should be always employed when pain or inspiration is present, of a "catching" or "stabbing" nature. *Tartar emetic*, says Laennec, is in general well supported in pleurisy, and contributes powerfully to subdue the inflammatory tendency; but, nevertheless, when the pain in the side and fever have ceased, it loses further power over the disease; at least, it does not appear to promote the removal of the fluid effused, so that its use must generally be abandoned as soon as the acute symptoms have passed away.

With respect to the application of blisters, Laennec objects to their use until the acute stage is past; but when the pain has ceased for some days, and absorption of the fluid proceeds slowly, and the disease promises to become chronic, a succession of blisters may be applied.

Such is an outline of the treatment recommended by Laennec, and which is still to be followed out. The points aimed at by venesection being relief of pain, and moderation in the force and frequency of the pulse, the patient ought to be bled in the upright

posture, and the blood should be allowed to flow in a full stream until he can take a deep breath freely, or till he feels faint and exhausted. From ten to twenty ounces may be necessary to accomplish this end, according to the severity of the case and the nature of the constitution. After free evacuation of the bowels has been effected, *calomel* to the extent of producing the *slightest* mercurialization, is the most beneficial line of treatment, to be followed in *certain cases only*—namely, those which do not acknowledge any *constitutional diseases* as their cause. The more rapidly slight mercurialization can be produced the better; and hence, writes Dr. Walshe, during the first six hours, small doses of *calomel with opium* (*a grain and a half of the former, combined with a sixth of a grain of the latter*, or more, if the pain continues acute) should be given every half hour, while mercurial ointment is rubbed into the skin of the affected side near the axilla every fourth hour. Dr. Fuller recommends *half grain or grain doses of opium* every *three or four hours*, in combination with *one or two grains of calomel, and half a grain of digitalis*, and to have *the whole side covered with a piece of linen spread with mercurial ointment*: over this is placed a poultice covered with oiled silk (*l. c.*, p. 184). The patient must be carefully watched, so that neither *ptyalism* nor *narcotism* is produced. The moment mercurial action has been established, the further administration of the mineral must cease. Dr. Bennett has not met with a single instance where the good effects of calomel have been unequivocal (*op. cit.*, p. 654).

Opium and digitalis are advised to be continued after the use of calomel has been suspended; and with them may be given two grains of *squills* or of nitrate of potash, which will act beneficially as a diuretic; or after twelve or more hours very small quantities of *tartar emetic* in solution may be given at night, combined with small doses of *opium* and *ippecacuanha*, to allay cough and general irritation. After the febrile action has in some measure subsided, and the active stage of the disease is at an end, a blister may be applied over the lateral region of the chest, but *not* over the seat of pain; and if the fluid continues to accumulate, the blisters ought to be repeated, so as to maintain a surface at a distance from the affected part in a constant state of counter-irritation. Diuretics ought at the same time to be freely given. The *compound tincture of iodine* in doses of *twenty minims freely diluted*, is a valuable medicine at this juncture; and so also is the *liquor iodum* of the British Pharmacopœia of 1867.

In the chronic stage of pleurisy, physicians are agreed as to the necessity of generous diet and tonic remedies in aid of any diuretics and absorbents which may be employed. If the patient be kept too low after the stage of active inflammation has subsided, or be unduly depressed, it will not only be impossible to induce absorption of the fluid, but there will be great danger of its becoming sero-purulent in character. The unfavorable issue of pleurisy in its chronic stage is in many cases attributable to a want of tone in the system, caused by the treatment adopted; the patient being kept too low, or he is overmuch purged, or is in some other way unduly de-



pressed, so that the system is unable to exercise its reparative power. A more generous diet ought to be given, and the general health sustained by quinine and other tonics. The use of diuretics and absorbents, ought at the same time, be steadily persevered in. When a succession of blisters has failed in relieving the patient, *ioduretted lotions*, or *ioduretted ointments*, combined with the internal administration of *cinchona* with *tincture of iodine*, *iodide of potassium*, and small doses of *bichloride of mercury*, *nitre*, *acetate of potash*, *squills*, *digitalis*, and *cantharides*, have each in their turn effected the desired object (*l. c.*, p. 186). The fluid has been gradually reabsorbed, and recovery has ensued. The formulæ most useful in such cases are—(1.) A diuretic in the form of a pill composed of *digitalis*, *squills*, and the mass of *pill hydrargyri*, of each a grain and a half; *nitrate of potash* (twenty grains), combined with *tincture of the perchloride of iron* (fifteen minims), three times a day, may at the same time be given (FULLER, CHAMBERS). Professor W. T. Gairdner recommends as a diuretic the *cream of tartar* electuary, in which the *cream of tartar* is mixed in equal proportions with treacle, honey, or marmalade, and in some cases flavored with a few drops of peppermint oil. The dose is a teaspoonful repeated as often as the stomach will bear it, or as the urgency of the case demands.

(2.) A lotion, to be applied over the chest by *spongeopiline* or by *lint* covered with *oiled silk*, composed as follows:

R. Hyd. Bichloridi, gr. iv; Tinct. Iodinii Co., ʒiv—ʒvj; Glycerini, ʒiij; Aquæ Distillatæ, ʒivss; Ft. Lotio.

(3.) One or other of the following ointments may also be rubbed in upon the skin, over the side of the chest: namely—

R. Hyd. Bichloridi, gr. iv—v; Ungt. Iodinii Co., ʒiv—ʒvj; Adipis, ʒiv—ʒj; Ft. Ung.; or—

R. Hyd. Bichloridi, gr. iv—v; Potassii Iodidi, ʒij; Aquæ Distillatæ, q. s. ut Hyd. Bichlorid. et Potassii Iod. solventur; Adipis, ʒj; Ft. Ung.

Professor W. T. Gairdner and Dr. Fuller are the two most recent authorities who notice the treatment of pleurisy by puncturing the chest and letting out the fluid—an operation to which the name of *paracentesis* has been given. With regard to acute pleurisy, Dr. Gairdner infers, from his own experience, that “for the mere saving of life in the acute stage of the disease, the operation is not necessary in any but a very small minority of the cases which have not yielded to remedies” (*Clinical Medicine*, p. 374). But a large proportion of the cases recorded in Dr. Gairdner’s excellent work, just quoted, occurred before he began to adopt the method of withdrawing the fluid practised so successfully by Dr. Bowditch, an American physician. In consequence of the great facilities afforded by this method for withdrawing fluid from the chest, Dr. Gairdner’s views regarding the operation have undergone considerable qualification. He now substantially gives in his adhesion to the principles and practices of Dr. Bowditch, and claims for therapeutics a cor-

respondingly extended field of usefulness. He has only performed the operation in acute pleurisy in cases of *great distension*, after other remedies had a fair trial without effect; and has avoided doing it in cases of partial or moderate effusion.

As a means of relieving the chest in chronic pleurisy and empyema, the operation is a useful one; and the practical question is as to the time when the operation should be performed. The condition of the patient's health and respiration, and the absorption or non-absorption of the effused fluid, are the aids to a decision for or against the performance of the operation. As long, writes Dr. Fuller, as the breathing is not seriously embarrassed, and the general health does not decline, so long are we justified in making full trial of our remedies. But as soon as extreme shortness and distress of breathing, or lividity and anxiety of the countenance, denotes serious interference with the functions of life, delay is no longer justifiable, and it becomes our duty at once to give our patient the chance which the operation affords.

The space between the fifth and sixth ribs, counting from above downwards, should be selected (if adhesions are ascertained not to exist there), being the most depending part of the chest when the patient lies on his left side, the more usual position in this disease.

The operation is described under the following subject, namely:

#### HYDROTHORAX.

**LATIN** Eq., *Hydrothorax*; **FRENCH** Eq., *Hydrothorax*; **GERMAN** Eq., *Hydrothorax*; —**SYN.**, *Brustwassersucht*; **ITALIAN** Eq., *Idrotorace*.

**Definition.**—*Watery fluid or serum in the cavity of the pleura (of either or both sides), rarely occurring in the absence of pre-existing disease of the pleuræ, lungs, heart, or great vessels, nor without the influence of some specific or constitutional disease—"passive dropsy of the pleura."*

**Pathology.**—In hydrothorax the cavity of the chest, on being opened after death, is found more or less full of watery fluid or serum, which being removed, the pleura is seen sometimes healthy, but more generally of a dark color, in consequence of a quantity of venous blood being congested in the vessels. The fluid may be effused into one or into both cavities. It may also be limpid and colorless, like water; but more commonly it is citron-colored, and contains fibrine, much albumen, and sometimes urea, in cases of *Bright's disease*. The quantity effused varies from a few ounces to many pints; eight and nine pints are not unusual; and Laennec states that he once removed twelve pints from the right side of the pleural cavity. When the quantity of fluid is large, the lung is compressed towards its roots, and placed generally in the groove formed between the sides of the bodies of the vertebræ and the heads of the ribs to their angles. It is sometimes so flat as not to be more than half an inch in thickness. It may be compressed against the sternum if previously-existing adhesions to that region have

fixed its position there. When hydrothorax is secondary, almost every chronic affection, either of the liver, kidney, or heart, may be found coexisting at the same time. Occasionally it is the result of extremely slight pleuritis, rarely of severe pleurisy, and in these cases the serum is more flocculent, contains more albumen, and portions of lymph are often also seen adherent to the *pleura pulmonalis* or *pleura costalis*; the two pleuræ are also often more or less united.

**Symptoms.**—The effusion may take place either gradually or suddenly. In the former case it may be so slow that the lung is able to adapt itself to the presence of the accumulating fluid, and the symptoms will consequently be much less marked, although the effusion be large. In the latter case the functions of the lung are almost at once suspended, the countenance livid, and the breathing greatly disturbed. When the effusion is slow, the symptoms are,—difficulty of respiration, which is carried on rather by the shoulders and diaphragm than by the intercostal muscles, some expectoration, lividity of the face or lip, œdema of the legs, and either a very full laboring pulse, or one that is small, frequent, and intermitting: the urine also is extremely scanty. As long as the effusion is moderate the patient can lie flat in his bed without experiencing any inconvenience. In the event, however, of the effusion being so considerable that the function of the lung is entirely suspended, the patient is unable to lie down, from the sense of suffocation produced by the fluid gravitating towards the root of the lung, and compressing the larger bronchi, and he therefore sits propped up by pillows, with his head bent forwards. When hydrothorax is symptomatic, or consecutive of affection of the heart or of other disease, it is generally preceded by swelling of the legs or eyelids, by the urine being plentiful and albuminous, or scanty, high-colored, and loaded with the usual salts, and indeed by most of the symptoms of dropsy generally. In these cases the effusion seldom takes place into the chest till a few days before death, rendering the agony of death doubly painful and suffocating.

When the effusion is moderate, auscultation gives bronchial respiration, some mucous rhonchus, and *bronchophony*, and occasionally that condition called *ægophony*, which is a broken sound like the bleating of a goat, or the notes used in the exhibition of "Punch," and which is heard as though the patient was speaking at the end of the stethoscope, but not through it. This singular phenomenon is heard only when the instrument is placed about the level of the effused fluid. When the effusion is more considerable, the respiration is almost tracheal, there is neither *bronchophony* nor *ægophony*, and a dull sound is returned over a greater part of the chest. Again, if the patient's chest be bared, there is no expansion on the side of the seat of the effusion, the respiration of that part being carried on altogether by the shoulders and diaphragm; and should the effusion be excessive, the affected side bulges out, as in empyema, and its intercostal spaces are enlarged and prominent. If air exists as well as fluid, succussion of the patient gives the sound of the splashing of fluid, and sometimes the patient can produce this phenomenon by shaking his body himself.

**Diagnosis.**—The absence of pain and of the other symptoms of inflammation distinguishes this disease from *acute pleurisy*. Should, however, the pleurisy be chronic, it is impossible to distinguish the two diseases except by the previous history. The diagnosis, also, between *hydrothorax* and *œdema of the lung* is not always easy.

**Prognosis.**—Some cases of hydrothorax recover, but the prognosis is in all cases extremely grave and doubtful.

**Treatment.**—The treatment of hydrothorax is of great difficulty, from the many causes on which the effusion may depend, and also from the almost uniformly intractable nature of the disease. The general principles of the medicinal treatment to promote absorption of the fluid have been given under the section on the treatment of pleurisy; and it only remains here to notice the great facilities afforded for the removal of the fluid by the operation recommended by Dr. Bowditch, with the apparatus devised by Dr. Morrill Wyman, of Cambridge, U. S. The apparatus consists of a trocar a little larger than the ordinary exploring trocar, and a silver canula, with a stop-cock in silver, as light and small as possible, capable of being connected with a syringe by an intermediate piece of brass, also provided with a stop-cock, the two cocks working the same way, and acting as checks upon each other. Such instruments may be had of [Messrs. Codman & Shurtleff, Boston, and surgical instrument makers in the United States,]; and a drawing of the instrument may be seen in vol. xxiii, p. 348, of the *American Journal of Medical Science*; also for Jan., 1863; also vol. xx, Oct., 1850.

During the operation the patient should be seated, when possible, sideways on a chair, or astride the chair, with his face to the back of it; or, if unable to rise, he ought to be brought so that the affected side may be made to incline slightly over the edge of the bed. The most appropriate spot for puncture is between the seventh and eighth, or the eighth and ninth, or the ninth and tenth ribs, in a line let fall from the lower angle of the scapula; but as a rule let the trocar be introduced as low down as possible, consistently with the safety of important organs in the chest or abdomen. The exact position of the liver and spleen must be determined first in every instance. Laennec himself once transfixed the diaphragm and pierced the liver, and that through the *fifth* intercostal space. An enlarged liver or spleen may be detected as high as the fifth rib. Dr. Watson once witnessed an operation in which the trocar was pushed through the diaphragm into the spleen, which was usually large. The patient died a day, or two afterwards of peritonitis. Just under the lower angle of the scapula is a spot easily reached, and where the muscles are thin. But in selecting the precise intercostal space, Dr. Bowditch chooses one about an *inch and a half* higher than the line on a level with the lowest point at which the respiratory murmur can be heard in the healthy lung of the opposite pleural cavity. Having pressed the forefinger of the left hand deeply into the intercostal space, the trocar, with its canula, should be plunged through the tissues at the depressed part, keeping as near as possible to the upper edge of the lower of the two ribs. The point of the instrument should be raised rather than depressed, so

as to avoid injury to the diaphragm, liver, or spleen. The skin need not be incised before puncture. Having withdrawn the trocar, leaving the canula, the double-valve syringe is to be applied, and the effusion slowly drawn away, until the lung has undergone as much expansion as it can endure with safety. This will be indicated by a sense of dragging distension or pain. When the canula is removed, the wound contracts and closes so completely that no lint or dressing is required.

In the hands of the American physician, Dr. Bowditch, and Professor W. T. Gairdner, of Glasgow, and Dr. Budd, of London, this operation has been the means of saving many lives. It is comparatively harmless, gives but little pain, and in the opinion of these eminent physicians it is an operation which ought never to be allowed to fall into disuse by the profession. The effect of the operation is to relieve the mind as well as the lung of the patient from great oppression. Although before the operation he is quite weak, he is often able after it to get up and walk. Digestion becomes at once improved, and strength is rapidly regained. The cough, however, is apt to augment during the first few days; the pulse also retains its quickness; friction-sounds occasionally become developed; and several months may elapse before the vesicular murmur becomes properly re-established in the lung.

Whenever the pleural cavity has become distended with fluid, and the dyspnoea is great, Dr. Bowditch recommends operation without delay. When thus performed early, it prevents a long and tedious illness, and future contraction of the chest. It should also be resorted to in all chronic cases when the effusion does not disappear after a reasonable time and the use of appropriate remedies. Dr. Bowditch has performed the operation 150 times on 75 persons, and has seen it done in 10 other cases. Out of the 75 cases, 29 recovered completely, and apparently in consequence of the operation, which was generally performed after severe symptoms had set in; and in all these cases the tapping seemed to be the first step towards recovery. In 26 of the 75 cases the fluid obtained at the first tapping was *serum*; and 21 of these cases made good recoveries. If the fluid afterwards became *purulent*, an almost certain fatality attended such a change. Of 6 such cases, 4 died; and the two others were likely to die when Dr. Bowditch wrote. In 24 of the 75 cases, *pus* flowed at the first tapping; and 7 of these recovered, and 7 died. In such cases relief is always obtained; but the tendency remains to a fistulous opening or to phthisis. If the fluid at the first tapping is sanguinolent, thin, and of a dark red color, not coagulating, it forebodes an almost certainly fatal result; and is generally associated with some malignant disease of the lung or pleura. Of 7 such cases, 6 died; and the seventh was still lingering when Dr. Bowditch wrote. A mixture of bloody purulent fluid at the first operation is usually fatal. A fetid gangrenous fluid is very rare, is obviously of bad omen, and betokens gangrene of the pleura and lung.

The operation may require to be repeated. Dr. Bowditch has done it *eight times* in six weeks to the same patient—himself a



physician. One lady he tapped *nine times* in eight months and a half, commencing when she was four months and a half pregnant, and when orthopnœa was threatening death. She was delivered of a living child at the full time, and recovered. In one remarkable case a youth had obscure symptoms for nine months. Dr. Bowditch recognized by the physical signs "latent pleurisy," or "idiopathic hydrothorax," as some may style it. Four pints of fluid were removed at one time, and the lung was fully expanded in forty-eight hours. In three weeks the patient was well, and he continued so.

Dr. Bowditch, in a letter to Dr. Gairdner, thus states the general results to which he has arrived in the use of this operation (*Clinical Medicine*, p. 720): "I now never operate unless I find some distension or rounding out of the chest, and filling up of some of the intercostal spaces, so that the chest presents a uniform curve, and not alternate depressions and elevations, as in the healthy chest. I operate under the following circumstances when I feel certain there is fluid:

"1. When there is *severe permanent dyspnœa—orthopnœa*—however acute the disease, if I find fluid filling the pleural cavity, or nearly filling it.

"2. When there are occasional attacks of *orthopnœa* threatening death, even if there be not sufficient to fill more than half of the cavity. If the fluid seems to be the cause of the dyspnœa, I operate, because occasionally I have lost a patient when waiting for more extensive physical signs. This rule I apply to acute and chronic cases.

"3. I use the trocar after three or four weeks of ineffectual treatment, without any absorption being produced.

"4. In chronic idiopathic hydrothorax, a latent pleurisy, with simply physical signs to indicate *extensive* effusion, but when the rational signs are either very slight or none at all, save a general malaise and weakness."

In the experience of Dr. Watson the operation of *paracentesis thoracis* in simple pleurisy is not to be performed unless the life of the patient is in jeopardy—that is, in cases "in which the effusion continues and increases, and the side, instead of shrinking, enlarges; the functions of the lung on that side are entirely abolished; nay, the use of the remaining lung is greatly interfered with by the pushing over of the mediastinum; and the patient is in imminent danger of suffocation." In such cases the oppressed lung must be relieved by "*letting the fluid out*" (Watson's *Lectures*, 4th edit., vol. ii, p. 128). Again, he writes, "Life is plainly in jeopardy when the vital functions of the lungs or of the heart are greatly hindered; when symptoms present themselves of approaching death by *apnœa* or by *syncope*. If we discover no cause for those symptoms except the increasing pressure of liquid pent up in the pleura, we are warranted in ascribing them to such pressure, and bound to act upon that persuasion." Also, if death by *asthenia* appears inevitable, the patient losing ground from day to day, and when all other means of getting rid of the pent up fluid have failed, the patient should

not be denied the *chance* which the operation affords. Also, "whenever the effused liquid consists of *pus*, it should be let out" (*l. c.*, p. 130). With the arrangements of Dr. Bowditch's syringe, we are enabled to remove fluid *before* the *false membrane* thickens over the compressed lung, and so makes it difficult to expand again. The trocar and small canula take the place of an exploring needle, which Dr. Watson does not object to use, regarding its use as a "minor diagnostic puncture;" and if the lung is not covered by an inexpandible *false membrane*, it will gradually expand as the fluid is withdrawn, the operation being done with the greatest slowness possible.

By the peculiar construction of the exhausting syringe, Dr. Bowditch has been enabled to evacuate the chest much more completely than by any other method, and to prevent entirely (and with absolute certainty) the admission of air. "It appears to me," says Dr. Gairdner, "to be in every respect an improvement so important that it may be said to open up a new history for the operation of *paracentesis thoracis*; and I trust it will receive in this country the attention which is due to it" (*Clinical Medicine*, p. 380). On the other hand, and by Dr. Bowditch's own showing, it does not appear that the entrance of air into the cavity of the pleura produces dangerous symptoms. He has never found this to be the case, even when air has been pumped into the chest. Why, therefore, resort to so elaborate an arrangement of stop-cocks, syringes, and canulæ, and tubes? Dr. Fuller regards such instruments as practically inoperative. "The lung," he says, "is seldom able to expand freely at once; and as the chest-walls will not yield beyond a certain point, air must be allowed to find ingress into the pleural cavity, or the fluid would not flow out. I have seen two of the most perfect of these stop-cock instruments employed; and so long as precautions were taken to exclude air from the pleura, so long they failed to draw off more than a few ounces of the fluid: directly air was admitted, the liquid flowed through them freely. Therefore, however desirable it may be theoretically to exclude air from the pleura, it is practically impossible to do so if we wish to relieve our patient" (FULLER, *l. c.*, p. 192). But Dr. Fuller agrees in the view that "when acute inflammatory action has subsided, the admission of air does not necessarily excite suppurative action of the pleura,—nor is it found to interfere with the re-expansion of the lung. He believes that the lung is very slow in regaining its due expansion—more than five days. Yet he agrees also that every precaution should be taken to prevent admission of air (as it is certainly conducive to suppurative inflammation) when the grooved needle has shown that the effused liquid is serous (*l. c.*, p. 193). Hence, I think, we come back to Bowditch's plan as the best. "When, on the contrary, the grooved needle has proved the existence of *pus*," Dr. Fuller believes "that the admission of air is not of the slightest importance." But here, again we know that *pus* formed internally and not in contact with air, undergoes changes when exposed to air, which renders it not less imperative that we should, if possible, prevent its admission; therefore, if the lungs are not bound down by adhesion, or prevented from expanding by the thickness of the

*false membrane* which covers it, here again Bowditch's syringe and stop-cocks enable the fluid, even when purulent, to be withdrawn without air being admitted.

But when the air has been admitted, and when the fluid within the cavity of the chest has become changed thereby—is purulent and perhaps fetid—the evacuation of the contents by “drainage” seems the most efficient remedy. This operation consists of introducing, through the opening made in the chest for removing the fluid, “a fine, long iron probe, somewhat bent.” It “is then directed towards the lower and back part of the pleural cavity,—the lower the better. If the end of the probe be made to press against the side of the thoracic walls, it can be felt from the outside, through the intercostal spaces, though perhaps obscurely, owing to thickness and toughness of the false membrane. The lowest and most appropriate site in which the probe can be felt having been selected, an incision is made upon the end of the probe, which is then brought through the opening thus made. A strong piece of silk thread is passed into the eye of the probe, and drawn through the two openings, and the drainage tube—an India-rubber tube, perforated at frequent intervals, in the way recommended by Chassaignac for the treating of sinuses—being firmly tied to one end, is then drawn through by means of the silk: the ends of the tube are then tied together, and the operation is complete. . . . The openings in the chest-walls are thus always free; the matter is discharged drop by drop as it forms, so that if the tube be suitably placed, there is never any collection of pus in the thorax; no time is given for decomposition, and the pus, therefore, is discharged in a healthy and pure state” (Goodfellow and De Morgan, in *Med.-Chir. Trans.*, vol. xlii). These are just the conditions required in cases of empyema; and by means of such a tube fetid fluid may be got rid of, and the cavity of the pleura washed out with warm water, containing a weak solution of *permanganate* of potash (Condy's fluid), of the strength of *two fluid drachms* to the pint of water (FULLER). Dr. Watson is also of opinion that if the corrupt and corrupting mass, in two cases which he cites, had been duly removed, the patients would have had a much better chance of life. With such a tube and a syringe, offensive gases and fluids may be got rid of.

### PNEUMOTHORAX.

LATIN Eq., *Pneumothorax*; FRENCH Eq., *Pneumothorax*; GERMAN Eq., *Pneumothorax*; ITALIAN Eq., *Pneumotorace*.

**Definition.**—A collection of air or gas in the cavity of the pleura, generally coexisting with fluid in the same cavity,—when the condition is sometimes termed hydro-pneumothorax.

**Pathology.**—Such a collection of gas or air within the cavity of the pleura, on either or both sides, occurs under any of the following conditions (FULLER, *l. c.*, p. 199):

1. When no communication exists between the pleura and the external air, it is then presumed to be due either to the spontaneous

evolution of gas from decomposing fluid in the pleura, from gangrene of the pleural membrane, or from the secretion, generation, or exhalation of air from the pleura—a phenomenon of doubtful occurrence *per se*.

2. When a communication takes place between the pleura and the alimentary canal, as in cases of softening and perforation of the œsophagus or stomach.

3. When a communication occurs between the pleura and the atmosphere, through an opening in the chest-wall, the result of penetrating wounds of the thorax or of parietal abscess.

4. When a communication occurs between the pleura and the bronchi; either the *result of violence*, rupturing the lung-substance, and tearing the pulmonary pleura; or the *result of disease*, causing perforation of the pulmonary pleura from without inwards, as in cases of *empyema*; or from within outwards, by ulceration. Such ulceration may be due to *tuberculous disease*, to *hydatid cysts*, *cancerous growths*, *pneumonic gangrene*, *metastatic abscess* or *bronchial gland abscess*; or, lastly, the lesion may be the consequence of *emphysema*, or *pulmonary apoplexy*, or minute bronchial abscesses perforating the lung during the course of typhoid fever (GAIRDNER).

Numerous cases of pneumothorax are to be met with from the bursting of a pulmonary cavity—during inspiration; or from the opening of a pleural effusion into the lung. Indeed, tubercular ulceration of the pulmonary pleura is the efficient cause of pneumothorax in 90 per cent. of the cases in which air is found in the pleura (WALSHE, FULLER). Cases, also, are on record of pneumothorax ending in recovery, with remarkable absence of bad symptoms. Such a case has been described by Dr. Thorburn (*Brit. Med. Journal*, June 2, 1860), and is probably a unique example of pneumothorax occurring in a previously healthy man, and terminating in a recovery virtually complete, running its course without fever, and with singularly little pain or dyspnoea. Dr. Gairdner, also, in commenting on Dr. Thorburn's case, observes that the acute and terrible symptoms so characteristic of pneumothorax in typical cases are occasionally absent individually, or at least not of diagnostic value absolutely. The disease, therefore, is one which may be overlooked in such cases where the symptoms are so latent that the date of invasion cannot be determined; and instances of the occurrence of pneumothorax being marked by the severe sufferings caused by another disease are so common as to be now well known (GAIRDNER). Cases of phthisis which do not seem to depart from their ordinary course, yet during the course of which aggravations are apparent, are just the kind of cases in which we may find, on examination, the pleura of one side more or less filled with air, but in which no date can be assigned to the pneumothorax, which has thus been detected at a period more or less remote from its actual occurrence (GAIRDNER, *l. c.*, p. 391). To detect the existence of any minute opening in the pleura after death, the side of the chest affected should be filled with water, and if the lung is then gently inflated, bubbles of air will escape if a perforation exists, however minute. It commonly occurs in the area comprised between the

third and sixth ribs (WALSHE, FULLER, CHAMBERS), "motion being greater there than in any other part of the chest," and therefore the pleura is less likely to be protected by adhesions there.

**Symptoms.**—The signs of pneumothorax are marked deficiency of respiratory murmur, without dull percussion, and with metallic phenomena, such as "a clear, mixing click, as of water dropping into a well" (*tintement métallique*), audible with almost every inspiration, and succussion-sound in a more or less well-marked degree. Nevertheless, "it is extremely difficult," as Dr. Gairdner observes, "to say precisely what, in the present state of science, constitutes complete evidence, in a clinical sense, of the existence of air in the cavity of the pleura. The combination of *very marked* deficiency of respiratory murmur, with very marked euphonic respiration or metallic tinkling, would probably be accepted by the skeptical critic."

These phenomena only present themselves when the pneumothorax is very extensive; and such cases rarely occur except in conjunction with pulmonary tuberculosis, when it is generally fatal. "Pain may be only moderate in degree, or not characteristic. Dyspnoea may be merged in a more chronic affection of the chest; fever may hardly be observed amid the hectic of phthisis or of empyema; while the more special sensation of rupture, alluded to by some authorities, and the suddenly-felt rush of fluid or of cold air into the chest, as described by others, are certainly quite as often absent as not" (GAIRDNER, *l. c.*, p. 387).

The signs of water, as well as air in the chest, may be discovered within a few hours after the presence of air has excited sufficient irritation to establish the pleuritis and serous effusion.

"The general symptoms in typical cases are comprised in physical distress and mental anxiety, as expressed in the countenance. The complexion is pale and dusky, and the lips more or less livid. The voice is weak; the skin moist, and often covered with a cold, clammy perspiration; the pulse is quick and feeble; and the respiration is extremely hurried" (FULLER, *l. c.*, p. 204).

The physical signs in typical cases comprehend—

"Convexity of the affected side, with obliteration, widening, and even bulging of the intercostal spaces, immobility or diminished movement of the chest-walls, and inaction or diminished movement of the intercostal muscles, contrasting forcibly with the increased play of the opposite side of the chest and the energy of its intercostal action. *Palpation* informs us that *vocal fremitus* is diminished or altogether annihilated; that the intercostal spaces are more than usually elastic or resilient; and that the heart is more or less displaced. *Mensuration* confirms our impression respecting enlargement of the affected side, and the increased width of the intercostal side. *Percussion* elicits a clear tympanitic resonance, which sometimes changes its character, and becomes amphoric, and of a metallic quality over the trachea and larger bronchi . . . and the area of clear resonance on percussion may extend considerably beyond its normal limits, as the mediastinum, the heart, and the diaphragm are more or less displaced. . . . *Auscultation* furnishes different results, according as the amount of the effused air is larger or smaller. If the quantity of



air be small, the respiration sounds are weak and distant, and the vocal resonance is weak; if it be great, so that the lung is thoroughly compressed, the respiratory sounds and the vocal resonance are almost or altogether absent, except in the interscapular region, at the root of the larger bronchi, where diffused-blowing respiration, and diffused but loud vocal resonance, may still be audible" (FULLER, *l. c.*, p. 204).

Certain signs resulting from the coexistence of air and serous or other fluid in the same cavity, which are not met with either in pleurisy or pneumothorax when they exist independently of each other, occur with the combined condition named *hydro-pneumothorax*, and are characteristic of it. These are described by Dr. Fuller as follows: (1.) *Fluctuation*, which is felt by the patient as well as by the observer, when the patient's body is abruptly jerked or shaken; (2.) A ringing, splashing sound—the succussion-sound of Hippocrates—which is heard under the same circumstances; (3.) A remarkable metallic tinkling, which sometimes accompanies succussion of the patient, but which is also apt to accompany cough or inspiration, or a sudden change in the patient's posture (*l. c.*, p. 205).

**Prognosis.**—When it affects the whole or nearly the whole, of one side in tubercular disease, it is usually fatal after periods varying from minutes to weeks. *Limited* pneumothorax, however, is less fatal, as in cases, for example, in which the lung gives way by a mere *pin-hole perforation*, but the pre-existence or rapid formation of adhesions limits the escape of air to a part only of the cavity, and the pin-hole opening is sealed up. Pleurisy in such cases, therefore, in relation to pneumothorax, is not to be regarded as a fatal complication, but as a healing power (GAIRDNER, *l. c.*, p. 396), the formation of adhesions often anticipating perforation of the pulmonary pleura in cases of tubercular phthisis. These adhesions limit the escape of air, prevent the utter collapse of the lung when air does escape, and so maintains to some extent the function of the lung, which otherwise would be destroyed as a consequence of such collapse. Prognosis is most unfavorable in perforation cases resulting from disease in the tissue of the lung, as contrasted with the more favorable prognosis in traumatic cases, in which the chest-walls are wounded, but in which the lung is not wounded. And those cases, *cæteris paribus*, are most apt to run an untoward course where there is great accumulation of air and great displacement of the thoracic organs (FULLER).

**Treatment.**—Puncturing the chest-walls, to relieve tension, may give temporary relief, by allowing the air to escape; but as the relief is only temporary, the operation ought only to be done in cases where the dyspnœa is urgent, and the displacement of the viscera such as demands that relief which the operation is calculated to give. Otherwise, the treatment of pneumothorax is best conducted by small doses of morphia—*i. e.*, stimulant doses—frequently repeated, combined or not by *ether*, or *alcohol* in small doses, with a view to overcome collapse, relieve dyspnœa, and subdue pain. When reaction follows the collapse, as indicated by heat of skin, strength and hardness of the pulse, soreness and pain of the affected

side, local bloodletting by leeches, with saline aperients, may be used if the strength of the patient warrants such a line of treatment. Turpentine and poppy fomentations, followed by blisters, are also indicated if life is sufficiently prolonged after the primary inflammatory symptoms are subdued. In other respects the treatment is similar to that stated under pleurisy.

## DISEASES OF THE TRACHEA AND BRONCHI.

### BRONCHITIS.

LATIN Eq., *Bronchitis*; FRENCH Eq., *Bronchite*; GERMAN Eq., *Bronchitis*; ITALIAN Eq., *Bronchitide*.

**Definition.**—*Inflammation of the air-passages leading to the pulmonary vesicles, characterized by hoarseness, moderate cough, heat, and soreness of the chest anteriorly—symptoms which are more and more intense according to the severity of the disease. The natural mucous secretion is at first arrested, but subsequently it becomes increased in amount and altered in quality, tending to assume the corpuscular character.*

**Pathology and Morbid Anatomy.**—The mucous membrane lining the bronchial tubes may undergo the inflammatory process, followed by results peculiar to the texture of the part affected.

In diffuse bronchitis we find the inflamed portions of the mucous membrane of a deep venous red, and this redness may be general or partial, in spots, streaks, or arborescent forms. The more asthenic the inflammation, or the more feeble and cachectic the patient, the more livid and purple is the redness. The secretion of the tubes, at first arrested, is eventually increased in quantity and variously altered in quality. It becomes thin, watery, and frothy, and subsequently thicker and more consistent, assuming the appearance of pus. (See vol. i, pp. 94–101.) Abortive or young epithelial cells, loaded with serous effusions, and losing their vital cohesion with the basement-membrane, are rapidly and easily discharged, constituting the thin, watery, frothy, serous expectoration of bronchitis in its early stage. Fibrinous exudation subsequently abounds, and the expectoration becomes tenacious and more opaque, and even pus-like.

**Casts of the bronchial tubes.**—In a very few cases lymph is thrown out, forming a false membrane or cast of the bronchial tubes. (See under “Characters of Sputa.”) Since Dr. Baillie first described and figured these tubular expectorated products, cases have been minutely described by many observers, and especially by Dr. T. Peacock, of St. Thomas’s Hospital, in the *Transactions of the Pathological Society of London*. Figures of such casts are also to be seen in the description of a case published by him in the *Medical Times and Gazette* for 1854, p. 659. Such a form of bronchitis is known and described by the various names of *plastic bronchitis*, *bronchitis crouposa*, or *bronchité pseudo-membraneuse*.

Occasionally it has occurred that the expectoration of bronchitis is of a very fetid character, so that the case simulates gangrene of the lungs. An instance of this description (with other analogous cases) has been carefully described by Dr. Laycock, Professor of the Prac-

tice of Medicine in the University of Edinburgh. A chemical analysis of the expectoration in this case demonstrated the presence of *butyric and acetic acids*; and the odor was characteristic of the *butyrates of ethyl*. It resembled the smell of the May-flower or apple blossoms, but was combined with an odor of *fæces* (*Med. Times and Gazette*, May, 1857, p. 480). This condition is generally associated with dilatation of the tubes.

*Ulcers*.—In a small number of cases ulceration of the bronchial membrane occurs from within outwards, frequently associated with dilatation of the bronchial tubes, and constituting characteristic bronchial abscesses.

Many authors affirm that the bronchial membrane, when inflamed, is thickened, and more particularly at the points of division of the tubes, and that the various abnormal sounds depend on the degree of thickening of this membrane, slight alterations of diameter producing great alterations of sound. The thickening of the mucous membrane of the smaller bronchi may be so great as to cause a complete obstruction (ANDRAL). This thickened state of parts, however, is very difficult to demonstrate, and the different sounds so often heard in bronchitis are with much more reason ascribed to spasmodic contractions of the circular fibres; and there are good grounds for believing that a partial spasm of the tubes is in all cases connected with bronchitis, especially in its early stages; and which is also the chief cause of the narrowing of the tubes at particular points, in connection with sonorous and sibilant râles.

The bronchitis may affect one lung, or both lungs, or a part of a lung, and the upper lobes are more commonly affected than the lower ones. The larger bronchi are also supposed to be more commonly inflamed than the smaller ones. Hence it is that the more marked primary effects of bronchitis are most obvious in the bronchi towards the roots of the lungs; while the secondary effects which are apt to follow a prolonged or severe attack, such as *vesicular emphysema*, are mostly developed towards the pleural surfaces of the lungs, and especially towards those surfaces most directly under the influence of the expansion of the more movable portions of the thoracic parietes.

A result of bronchitis which in its morbid anatomy may be mistaken for the tubercle of pulmonary phthisis has been recently demonstrated by Zenker, of Dresden. It is described as catarrhal induration of the pulmonary air-vesicles, by the accumulation of epithelium carried into them from the adjoining smaller bronchi, and which eventually fills up and consolidates the vesicular structure of the lung where this takes place. Zenker is one who does not believe in the possession of epithelium by the air-vesicles; and the tortuous vessels seen in them like varicose veins, especially in *stenosis* of the mitral valve, he regards as the normal state of vessels which are naked and uncovered by epithelium.

One most direct, invariable, and important result of bronchitis in the adult is condensation of the vesicular substance of the lung, of a peculiar type, as a result of mucous or other obstruction in the air-tubes leading to the condensed portion. Professor W. T. Gaird-

ner has shown this, and that such a condensation is most apt to occur in bronchitis associated with asthenia, although in such cases the amount of obstruction may be small, as in patients debilitated by disease or by age. Such condensation is also produced by collapse of the pulmonary vesicles, the boundaries of this local condensation being distinctly mapped out by the interlobular divisions of the pulmonary lobules implicated. Such collapse, when extensive and sudden, is not only a frequent cause of death, but at the same time it is a fleeting, temporary condition of immense frequency, and important practically to distinguish from the condensation of pneumonia.

The morbid anatomy of bronchitis, which an examination of the air-passages discloses, shows that obstruction in the tubes may be due to fluid mucus, or even to solid fibrinous coagula, or more or less prolonged spasm of the circular muscular fibre, and that such obstructions tend to the production of pulmonary collapse; and if the obstruction be considerable, and persistent, large portions of the lung may be emptied completely of air in the course of a few hours. The mechanism by which a mechanical obstruction, such as mucous fluid or more solid substances, brings about this collapse, appears sufficiently obvious when it is observed that such obstruction is never absolutely complete. The air gradually finds its way out by the edges of the obstructing substance, impacted as it is more or less in a series of diminishing tubes such as the bronchi, and acting the part of the ball-valve of a syringe, so that when the obstruction is driven onwards towards the narrower tubes by the force of inspiration, occlusion is more or less perfect only in the one direction. The expiratory force, however, so long as there is air in the vesicles, constantly tends to dislodge the obstructing body by pushing it towards the wider end of the tube. While, therefore, the entrance of the air is constantly and more or less effectually opposed, its exit is always permitted, so that ultimately the air-vesicles of the tubes beyond the seat of obstruction become completely emptied, and they collapse. The result of such collapse is a condensation of the tissue of the lung—a condition which had previously been ascribed to a limited inflammation of the pulmonary tissue, known as *lobular pneumonia*, and which was commonly believed to be peculiar to infants. Such collapse and condensation of the lung, however, whether in the lobular or diffused form, is an exceedingly common lesion in the adult, as shown by Dr. West and confirmed by Dr. W. T. Gairdner, especially in old persons, in typhus fever and in fatal dysentery, and is always associated with a certain degree of bodily weakness. Dr. Gairdner has further shown, however, that it is almost invariably found as a concomitant of fatal bronchitis, and that it depends on the obstruction of the tubes, as just described. When this collapse becomes permanent, it leads to still more obvious and important results:

1. Like other parts which become useless, the collapsed portions become atrophied, leaving only a small amount of fibrous tissue in its place, the proper and special elements of the pulmonary tissue

having disappeared. Such an atrophy causes a diminution of the volume of the lung at the place where the collapse occurs.

2. By a definite law (which may be expressed thus: *That a compensation by increased volume in one or more parts of the thoracic viscera invariably occurs to make up for diminished bulk in another, the internal capacity of the chest remaining the same*) this pulmonary collapse and atrophy invariably leads to vesicular emphysema of the lung, and even tends to dilatation of the heart itself. The most constant result of collapse, however, is emphysema, so much so that the one almost never occurs without the other.

Bearing upon these statements, several general phenomena have been noticed, which may be thus shortly enunciated:

1. That emphysematous portions of lungs are almost invariably free from every diseased appearance except the dilatation of the air-vesicles, and the consequent stretching and disorganization of their parietes.

2. That the bronchi leading to them are usually quite free from obstruction.

3. That vesicular emphysema by *increase of volume* of those portions of the lung to which the air has access, compensates for the *diminished volume* of the collapsed portion.

4. That vesicular emphysema prevails in the opposite parts of the lung to those in which the direct effects of bronchitis are observed.

The following are the forms of bronchitis which may be clinically recognized: (1.) *Acute bronchitis* (a) *of the larger and medium-sized tubes*—(b) *Capillary bronchitis, and of the tubes generally*—the “*peripneumonia notha*” of the older writers; (2.) *Chronic bronchitis*; (3.) *Plastic bronchitis*; (4.) *Mechanical bronchitis*, such as knife-grinders’ disease—*carbonaceous bronchitis*, or black phthisis; (5.) *Bronchitis secondary to general diseases*, such as typhoid fever; (6.) *Bronchitis secondary to blood diseases*; (7.) *Syphilitic bronchitis*.

**General Symptoms of Bronchitis.**—Bronchitis, of whatever kind, is generally preceded by fever, but more commonly by symptoms of what is commonly called “*a cold*,” or “*a cold in the chest*.” It often commences without any previous illness, and the uneasy sensations, frequently commencing about the region of the frontal sinuses, gradually pass down the nasal mucous passages, and thence by the trachea and windpipe, are experienced in the chest, especially over the anterior region. The symptoms of bronchitis becoming developed are expressed by the hoarse, altered voice, the cough and expectoration, and are too palpable to allow us to mistake the nature and existence of the disease. In a very few instances of diffuse inflammation, especially in *capillary bronchitis*, the cough is dry and without expectoration; but far more generally it is accompanied by sputa. The sputa vary greatly according to the different degrees of inflammation, or according as that inflammation is acute or chronic, sthenic or asthenic. In acute cases it is at first a thin mucus, sometimes streaked with blood, then more opaque, and lastly purulent; in more chronic cases it may be merely a muciform saliva, or a gelatiniform mass; or it may be like the unboiled white of egg, so



tenacious that it may be poured from one vessel into another without separating. In other instances it is puriform, varying from a laudable pus to a red or green putrilage. When purulent, it is usually formed into sputa, but in a few cases it is thrown up in large quantities unmixed, as from an abscess. The quantity of matter expectorated also varies greatly; sometimes only a few sputa in the morning, at others half a pint or a pint in the twenty-four hours, while other patients actually die suffocated from the immense quantity which is suddenly poured out, causing obstruction of tubes and collapse of the vesicular structure of the lungs.

The cough is seldom accompanied by any pain in the inflamed membrane, and has many degrees of violence. It may occur in paroxysms, and the sputa be discharged after a violent effort, at night or in the morning, or at other definite intervals. Again, it may be incessant, harassing the patient at every instant, causing a sense of soreness or constriction of the chest, and sometimes severe pain at the ensiform cartilage, in consequence of the mechanical exertion of coughing.

The urine of bronchitis varies greatly, as the grades of the disease are almost infinite, from a very slight affection of the larger tubes to a disease involving all the smaller tubes of both lungs, accompanied, perhaps, by collapse of the air-cells in some cases, and impeding aeration to a great degree in all. In these cases the urea is in large amount, and the pigment is increased, while the chloride of sodium is sometimes as deficient as in the height of extensive hepatization; and it appears to Dr. Parkes that those cases in which the urinary ingredients are in extremely small amount are more commonly those of severe and general capillary bronchitis. The retention or non-excretion of the urinary substances seem to be more common in diseases attended with a considerable impediment to aeration than in other affections in which the absorption of oxygen is presumably less interfered with (*On the Urine*, p. 282).

With respect to the effects of the cough on the constitution, the patient, supposing the disease to be unconnected with any morbid poison or organic affection of the substance of the lung, suffers little in his general health, and often feels he would be well if he could get rid of "the cough." In other cases he loses flesh, ejecting every meal, from the violence of the cough, or he sinks into a state of marasmus simulating phthisis. His pulse is generally natural, although in some cases it is frequent; his bowels also are regular. In bad cases, however, the patient's nights are broken, and he sleeps towards morning; while in slighter cases he sleeps through the night, but is disturbed early in the morning by cough and expectoration.

The duration of bronchitis is uncertain. It sometimes terminates in a few hours, sometimes in a few days, ceasing with the cold weather that ushered it in. In other cases its duration is long, and it is with difficulty recovered from; thus often laying the foundation of other formidable diseases which may ultimately destroy the patient. In old persons it generally returns every winter, or lasts, with intermissions, during the whole year.

**Physical Symptoms.**—The natural and healthy respiratory *bruit* of an adult has been compared to the sound heard during the calm sleep of a healthy child. In bronchitis this sound in the adult is changed, and varies, in different cases, from a tolerably sharp sound, which, when multiplied from a number of bronchi similarly diseased, resembles a chirping sound, or the bass notes of the violoncello. The sounds thus embrace a musical scale of considerable compass, the principal and more marked division of which compose the *sonorous* and *sibilant râles*, as they are termed. The cause of the higher notes has been supposed to be owing to a thickening of the mucous membrane at the orifices of the various bronchial tubes, so that the natural *embouchure* is narrowed, and a musical wind instrument is thus formed. To those who have observed in the dead body a swollen state of the bronchial membrane, this explanation may seem satisfactory; but to those who have not seen such a phenomenon, it seems more easy to explain this morbid sound by the different degrees of contraction of the circular and longitudinal fibres of the bronchi, in the same manner as we observe contraction of the muscular fibres of the œsophagus, or of the small intestines, causing a constriction. Besides the alteration of tone of sound in bronchitis, its quality is also often affected by the presence of liquid matters within the cavity of the bronchi; and hence we have it interrupted and modified by the air passing through bubbles of mucus; and as the size of these bubbles, and their viscosity, vary, so the sounds vary. Hence a scale was established by Laennec, whose extremes are the “*râle muqueux*,” and the “*râle tracheal*,” the former representing the bursting of small slightly viscid bubbles, the latter larger ones of greater tenacity, and yielding a gurgling sound. Sometimes this mucus, instead of being fluid, hardens so as occasionally to adhere and play as a valve, giving rise to a clicking noise. These are the various morbid sounds heard in bronchitis; and the danger of this disease is denoted by the quantity of fluid effused, and may be judged of by the nature of the sound. The sharp chirping sound is more to be feared than the graver and deeper notes; for grave sonorous notes originate in the larger tubes, chirping, whistling notes in the smaller; and the danger in bronchitis increases in proportion as the finer bronchial tubes become involved (*Edin. Med. Journal*, 1864, p. 1114). When the sounds of expiration are also much prolonged, severe bronchial inflammation is indicated.

Percussion generally returns a healthy sound in bronchitis. An important physical sign is one which indicates a sudden disappearance of the respiratory murmur over a definite part or parts of the lung. Percussion sometimes shows, however, that the part still contains air; and therefore it is presumed that the disappearance of the murmur is due to obstruction of one or more of the bronchial tubes; and which, as already shown, may lead to *collapse*, *condensation*, *atrophy*, and *emphysema*. In some cases the respiration is greatly accelerated, varying from 36 to 50 or 68 in a minute, especially in cases of capillary bronchitis. The pulse at the same time rapidly loses its strength, and becomes excessively frequent—120 to

150. The ratio of the respiration to the pulse is therefore greatly altered from the normal standard—that is, about 4 or  $4\frac{1}{2}$  beats of the pulse for every respiration; and thus *pulse-respiration* ratios of 3.0, 2.5, or 2.25 to 1 are not uncommon.

**Diagnosis.**—It is hardly possible to confound bronchitis with any other disease; but there is often much difficulty in assigning its cause and distinguishing it from phthisis. The absence of great emaciation, and the clear resonance returned on striking the chest, are the most salient points in diagnosis.

**Treatment.**—When the symptoms of a “common cold” first express themselves, and even when the sensations have extended to the chest, as indicated by the hoarseness and tendency to cough, the disease may be at once subdued in a healthy person by a full stimulant but not narcotic dose of opium or morphia (i. e., *one grain of opium, or a fourth of a grain of morphia*) at bedtime; or by an alcoholic diaphoretic drink; or by *five grains of carbonate of ammonia, or ten to twenty grains of muriate of ammonia*; or, if the appetite is unimpaired, a full supper, followed by a moderate amount of some hot alcoholic stimulant, may have the same effect.

When the attack is sudden, and attended with marked depression, associated with aching pains in the head, back, and limbs, and much febrile disturbance, opium, if taken early enough, is a perfect remedy. “One-third of a grain of muriate of morphia ought to be taken at bedtime, and its influence will begin to be felt in from twenty minutes to half an hour, by the gradual disappearance of the sense of intense weakness, the relief of the pains, and that peculiar feeling of thorough and evenly distributed warmth of the whole body which is so different from fever on the one hand or chilliness on the other” (ANSTIE, *op. cit.*, p. 120). Natural sleep ought to supervene, and the morning ought to find the patient well.

If such remedies are delayed too long, the object to be aimed at next is to induce a copious perspiration and a continued action of the skin and kidneys, in the first instance, by small doses frequently repeated, of *antimonial* and *ipecacuanha* wines, or *nitrate of potash*, or *acetate of potash*, as well as *bicarbonate of potash* and *aqua potassæ*, combined at a much later period with tinctures of *squills* and *hyoscyamus*.

In the early stage of the disease we have seen that the bronchial membrane has its normal moist condition altered to a very dry state; and the object of treatment is to bring about a return of the naturally moist condition. This is best effected by the inhalation of the vapor of hot water, the use of *tartar emetic* in doses of *one-twelfth* to *one-sixth* of a grain, the use of *ipecacuanha* in *one-quarter* or *half-grain* doses; and these may be aided by *hemlock*, *henbane*, *aconite*, or *hydrocyanic acid*, in appropriate doses. From the frequent inhalation of steam great benefit is derived (EASTON).

Abundant experience has shown that general bleeding in acute *bronchitis* uniformly weakens the patient, without greatly influencing the disease. Neither has medicine any very marked effects in the cure; for although some persons rapidly get well under a given

treatment, yet many similar cases, under exactly the same treatment, will run on for weeks, and perhaps for months, without any amendment. In the most acute cases of bronchitis, however, some blood may be taken from the chest, with great caution not to take too much, either by cupping between the shoulders or by leeches, and in general from ten to twelve ounces are sufficient. It is only in cases of congestion of the brain, heart, or venous circulation threatening asphyxia, that general bloodletting is imperative. Next to bloodletting, *tartarized antimony* administered in solution in doses of *a sixth or a quarter of a grain, every three or four hours*, conduces to free secretion, and generally to mitigate the symptoms of the disease. Dr. Fuller regards *digitalis* as a useful adjunct to the antimonial treatment; and the air-passages should be fomented by the inhalation of moist warm air, as by the steam of hot water, the secretions being at the same time stimulated, so that the bowels act freely. When the expectoration becomes thicker and less copious, the antimony may be decreased, and *squills*, or *ipecacuanha*, with *paregoric*, given. After this a blister should be applied to the chest; and on its being removed, a large linseed poultice should be placed over the blistered part, and be continued for many hours, which will not only keep the ulcerated surface open, but gratefully foment the part and relieve the patient. The bowels should be freely evacuated by a purgative dose of calomel, combined with *compound jalap powder*, and they should subsequently be kept in regular and gentle action by some neutral salt, such as the *sulphate of magnesia* in the *liquor ammoniæ acetatis*, combined at the same time *with some nitrate of potass*. The *compound jalap powder* is a most useful remedy when the system will bear it. Edema is greatly relieved by its use, whether of the lungs or of the body generally. The neutral *citrates*, *tartrates*, or *acetates* of the alkalies are useful eliminating remedies.

After these means have subdued the severity of the symptoms at the outset, expectoration should be promoted by such remedies as *squills ipecacuanha* and *tartar emetic*, combined with *hyoscyamus* or *conium*. Opium in narcotic doses (*i. e.*, above a grain) is inadmissible if the evacuation of the loaded air-passages is to be promoted, because its tendency, in a narcotic dose, is not only to diminish the secretion, but to paralyze the action of the mucous passages in eliminating that secretion. When large doses of opium have been given at this stage, death has been known to follow, and necroscopic examination has revealed the air-passages loaded with frothy serous mucus, and the air-cells congested and collapsed. It is not till after secretion has begun to diminish, in acute cases, that opium may be prescribed with benefit in stimulant doses, and it is then to be given in the form of the solution of the salts of morphia, added at bedtime to the doses of the *cough mixtures* so usually administered. If the disease shows a disposition to pass into the chronic stage, *quinine*, with *squills* and *conium*, may be administered, or a draught containing full doses of *cinchona*, with *five grains of carbonate of ammonia*, or *ten to twenty grains of the muriate of ammonia*, and *thirty or forty minims of the compound tincture of benzoin or of the bal-*

*sam of Peru*, will generally facilitate expectoration and relieve the dyspnoea. If a "heartly cough" is not attended by easy and free expectoration, but the chest remains loaded, powerfully stimulant expectorants may be given in aid of other remedies, such, for example, as the *decoction of senega* or the *misturæ ammoniaci* (FULLER). The patient throughout the treatment should remain in a room where the air is kept moist by the evaporation of boiling water from large flat dishes near the bed, and the temperature of the air should be maintained at 63° to 68° Fahr.

In chronic cases of bronchitis, especially in patients "who have made considerable progress in the journey of life, a lower tone of the system generally prevails, and a greater laxity of aërian membrane—particularly with excessive secretion, often muco-purulent"—characterizes such cases, and in them, after blistering, and perhaps poulticing the chest repeatedly with mustard poultices, the treatment in general should be more tonic. The *camphorated mixture* or *paregoric* and stimulant expectorant remedies are indicated for occasional but not for constant use. It was in these cases that the late Dr. Easton, Professor of Materia Medica in the University of Glasgow, so well showed the necessity of using remedies which will promote expectoration of the secretion which accumulates in the bronchial tubes. Such remedies are those which tend to invigorate the general system. Besides the selection of a beneficial climate, and the use of nourishing, easily-digested food, stimulating embrocations may be rubbed not only over the chest, both before and behind, but along the sides of the neck.

In the Meath Hospital at Dublin a liniment composed of the following ingredients is extensively employed by Drs. Graves and Stokes, and is recommended by them, as well as by Dr. MacLachlan:

R. Spt. Terebinthinæ, ℥iij; Acid. Acet., ℥iv; Vitelli Ovi, j; Aq. Rosæ, ℥iiss.; Ol. Limon., ℥j; *misce*.

As a rubefacient, it is to be applied morning and evening, when it generally reddens the skin and produces small pimples. In several cases the secretion of the kidneys is increased during its use (MACLACHLAN).

Of tonic remedies, which are invaluable, my friend the late Professor Easton, from his extensive experience, put most reliance on the influence of *nux vomica*, *iron*, and *cinchona*. He was in the habit of prescribing them in the form of a syrup composed of the *phosphates of strychnia*, of *iron*, and of *quinia*, so that, in doses of a *teaspoonful* three times a day, each dose shall contain the *thirty-second part of a grain of phosphate of strychnia* and *one grain respectively of the phosphates of iron and quinia*. (Formula for the preparation of this compound, see page 95, *ante*.)

Combined with these remedies, the inhalation of *slightly* irritant vapors has a beneficial effect, as of *vinegar*, *turpentine*, *chlorine*, and *iodine*. Inhalation of vapor is often unsatisfactory, on account of the difficulty of getting an apparatus to hold a sufficiently large



volume of boiling water. In the practice of my friend Dr. Fergus, of Glasgow, I have seen an admirable arrangement for inhalation, which overcomes this difficulty. It consists of a globular glass flask, about eight inches diameter, and six inches deep, having a wide mouth, a closely-fitting cork, carrying an inlet tube which descends to the bottom of the vessel, and an outlet tube, to which a flexible mouth-piece is fixed. It is used as an Eastern uses a *nargilé* for smoking through water.\*

Of the fetid gums, *ammoniac* in particular is a useful remedy. An emulsion of *gum ammoniac* in *diluted nitric acid* is a combination from which decided beneficial results are obtained. Professor Easton gave the following formula for its prescription—namely, *a hundred and twenty grains of the gum ammoniac dissolved in two fluid drachms of diluted nitric acid and twelve ounces of water*, compose a mixture of which an ounce may be given in gruel three times a day (*Glasgow Med. Journal*, Oct. 1, 1863). On the contrary, it is often advantageous to administer astringent remedies, and one of the most useful is *tannic acid* in doses of one to three grains two or three times a day, as originally recommended by Dr. Alison, of Edinburgh; or the *oil of cubebs* to the extent of ten drops three or four times a day on a piece of sugar.

Acute bronchitis is very apt to be latent in old people, and to be complicated with gastric or gastro-enteric inflammation (MACLACHLAN). The treatment must therefore be modified to meet such a contingency. When there is tenderness at the pit of the stomach, nausea, and failure of the appetite, with the general condition approaching a typhoid state, in persons beyond the meridian of life, the pectoral symptoms are often apt to be marked by such associated disorder. The stimulants of food (by *enema* in the form of soup, if unable to be taken by the mouth) and of alcohol, are the main remedies necessary from the very beginning. Abstinence cannot be enforced with safety. Leeches must be applied to the pained gastric region.

When the disease is associated with a tendency to gout, colchicum must be given. "It allays the cough, promotes the flow of urine, keeps up a regular alvine discharge, and can be given much more generally than squills, because it does not produce that feverishness which results from the use of the latter remedy, and can therefore be employed where there is considerable fever" (FORBES). It requires to be administered with great caution in the aged and infirm (MACLACHLAN).

In the protracted bronchitic affections of the aged, diuretics are of great service; and the following formulæ are recommended by Drs. Maclachlan and Stokes, as well suited in a variety of cases of senile chronic catarrh:

R. Decocti Senegæ ℥vij; Potassæ Nitratis, gr. iij; Tinct. Camph. Comp. vel Tinct. Conii, ℥xx; Spiriti Ætheris Nitr., ℥ss.; Oxymellis Scillæ, ℥ss. Fiat haustus ter die sumendus (MACLACHLAN).

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\* These inhalers are made of glass, so prepared as to receive boiling water without breaking; and are fitted up with tubes ready for use by Mr. Hugh Reid, chemist, 428 Argyle Street, Glasgow.

**R.** Liq. Ammon. Acet., ℥iij; Potassæ Acetatis, gr. xx; Aceti Scillæ, f℥ss.; Spirit. Æth. Nitr., f℥ss.; Tinct. Camph. Co., ℥xx; Mist. Camph., ℥vj; Syrupi Aurant., ℥j (MACLACHLAN). Fiat haustus ter die sumendus.

**R.** Decocti Senegæ ℥v; Tinct. Camph. Comp. Scillæ, āā ℥ij; Syrupi Tolut., ℥iv. Sumat ℥ss. vel ℥j ter die (STOKES, MACLACHLAN).

When gastric irritation prevails, the administration of balsams, gum resins, and terebinthine remedies must be suspended.

[CAPILLARY BRONCHITIS—*Suffocative Catarrh.*

Syn, LATIN, *Bronchitis Capillaris, Peripneumonia Notha*; FRENCH, *Bronchite Capillaire, Catarrhe Capillaire*; GERMAN, *Bronchitis Capillar.*

(DR. CLYMER.)

**Definition.**—*A dangerous inflammatory (so-called) affection of the smaller bronchial tubes, happening at all periods of life, and in all constitutions, but more common in children and old persons, and in those of feeble health; though occurring as a primary disorder, it is frequently secondary, complicating continued fever, the exanthemata, &c.*

This is a distinct form of bronchitis, always of serious import, and too frequently not recognized, or not distinguished from other pulmonary disorders of less mortal tendency. Most systematic authors seem to regard it as almost proper to the two extremes of life—infancy and old age—and its occurrence in youth or adult age as exceptional, and then only amongst the weakly. The writer's experience leads him to believe that it is more common in adults of both sexes than is generally admitted, and is not limited to those of feeble health or infirm from previous disease, but may happen in persons of vigorous and sound constitutions. The late Emperor of Russia, Nicholas, died of it in the full strength of manhood, and Dr. T. K. Chambers mentions a fatal case in his practice about the same time, of a clergyman of "large frame and great height" (*Clin. Lect.*, p. 246). The writer's observations have satisfied him that capillary bronchitis was not infrequent amongst our soldiers during the late war, especially the colored troops, though it was often diagnosticated, and was usually reported, as pneumonia, or as ordinary acute bronchitis. In young children it is sometimes met with as a secondary or intercurrent affection in continued fever, and often complicates the exanthemata, particularly measles, and is called by Trousseau broncho-pneumonia. It happens too as an idiopathic disease in infancy. When coincident with collapse of the lobules, it has been described as lobular pneumonia.

**Symptoms.**—An attack of capillary bronchitis may begin with shivering, though an initial chill is much less frequent than in pneumonia. Headache is often complained of, the tongue is furred, the face pallid, and the expression anxious, often from the outset. There is a feeling, with an aspect, of general debility frequently from the beginning, and out of proportion to the objective symptoms, the patient having the appearance of being in the first stage of continued fever, though the peculiar besotted look is wanting. The breathing soon becomes hurried and somewhat laborious; an incessant hacking cough sets in, which is generally dry, with the occasional expulsion of one or more grayish, or yellowish-gray, stiff pellets; or there is scarce, frothy expectoration. The pulse-rate is quickened, the skin dry, and its temperature variable. (There

is yet no reliable record of the body-heat.) These symptoms soon worsen; the breathing becomes more accelerated and difficult, and the breathlessness is harassing. The ratio of the respiration to the pulse is always high, the respirations going up to sixty or even seventy in the minute, with a pulse-rate of 120 or 130, the pulse-respiration ratio being changed from 4.5 to 2. The supplementary respiratory muscles are brought into vigorous action, and the supra-sternal, mastoid, scalenal, and infra-xiphoid spaces, and the base of the chest along the insertion of the diaphragm, are forcibly sucked in at each inspiratory effort. The face betokens great suffering and is of a bluish hue, the lips are purple, the conjunctiva has a bluish-gray tint and is turgid, and the skin of the extremities is of a dusky hue, and frequently at times cool and moist. There is great exhaustion. In many cases these symptoms are for awhile paroxysmal, with intervals of comparative comfort between the exacerbations. If the disease progresses, all the phenomena due to deficient oxygenation of the blood are aggravated; the cyanosis deepens, the facial anxiety is extreme and distressing, the surface is livid and damp, words are spoken with great difficulty, the dyspnoea is intense, and all the respiratory muscles are forcibly working; the patient is unable to lie down, though completely worn out. A frequent, short, moist cough, coming on in spells, replaces the ceaseless hack, and expectoration becomes free, the sputa being brought up during or after each fit; they are frothy, and aerated throughout, or spongy at the top, ropy, and adhering together as a single mass when turned out of the vessel. As death approaches, the pulse usually becomes more rapid, and is large and compressible, and then small and thready. The respiratory efforts are less violent, the number of respirations begin to lessen, and the pulse-respiration ratio falls, and approaches to a more natural standard; the deepening asphyxia probably making the patient less sensible of the respiratory wants, and carbonic narcosis may possibly, in some cases, retard the heart's action, and decrease the pulse-rate. Towards the end, both the cough and the expectoration cease, and death happens from apnoea.

The *physical signs* of capillary bronchitis are, *percussion-sound* at first natural over the whole chest; subsequently it may be slightly exaggerated over the superior and anterior regions, from the emphysematous state of the air-cells; and in some cases there are limited spaces of dulness, owing to collapse of the air-cells of one or more lobules, caused by obstruction of a bronchial tube; towards the close there may be diminished percussion-resonance on account of pulmonary oedema, and excessive accumulation of the morbid products in the air-cells and terminal air-tubes. *Auscultation* in the beginning shows the respiratory murmur clearer and somewhat exaggerated in the upper portions of the lung, with diffused dry rhonchi over the chest, the sonorous rhonchus being often quite loud and musical, and the sibilant of high pitch; if the respiratory murmur is not masked by the rhonchi, it is, in these regions, feeble; as we proceed downwards, and as the disease progresses, the distinctive rhonchus of capillary bronchitis—the subcrepitant—is heard, particularly in the posterior inferior regions. This fine moist rhonchus is mainly an inspiratory sound, though audible in expiration, and is described as resembling the continuous bursting of innumerable small unequal bubbles, or the sound produced by squeezing a not too wet sponge close to the ear. Heard at first about the base of the lungs, it soon becomes audible over nearly the whole chest, above and below, front and back, moving always upwards; it is from the beginning symmetrical. If the larger air-tubes are more involved, a mucous or submucous rhon-

chus may replace the dry rhonchi in the middle regions. When the lobules are collapsed, the signs of localized condensation of the lung-tissue can rarely, if ever, be made out, on account of the existing rhonchi. *Vocal resonance* is increased. There is no doubt bilateral local expansion of the chest, often of large extent, and Dr. Flint says that he has known the anterior superior and middle regions, in a young child, to become largely dilated, presenting the heteromorphism of long-continued and great emphysema, which disappeared on recovery; but owing to the violence and labor of the respiratory act, it is very difficult, and generally impossible, to make any accurate measurement.

The course of the disease is rapid; in children generally lasting from three to six days; in adults from a few days to two weeks; in old persons it rarely exceeds a week after the acute symptoms have set in. In young children capillary bronchitis sometimes comes on very insidiously, the symptoms being for awhile those of an ordinary catarrhus affection, and exciting little attention, although there are really distinctive traits present almost from the outset,—drooping, pallid face, quickened breathing, and spells of coughing, preceded and followed by wheezing. Dyspnoea suddenly happens, and chest-exploration shows extensive capillary bronchitis. In pulmonary emphysema of long standing, with extensive vesicular dilatation, intercurrent localized capillary bronchitis is very apt to happen, but it, so far as the writer's observations go, never becomes general, and though for a time aggravating the symptoms of the original disorder, is not a serious affection.

**Prognosis** is unfavorable, especially when capillary bronchitis supervenes upon any existing disorder which limits the respiratory area, and when in such cases it is general. In infancy and old age it is very fatal.

**Diagnosis.**—From simple acute bronchitis it can always be easily distinguished, both by the general symptoms and physical signs. Acute bronchitis is nearly always preceded by symptoms of “a cold,” and its course is downwards and peripheral, the proximal larger bronchi being first invaded, and afterwards the smaller distal ones. In capillary bronchitis the morbid action begins in the minute tubes near the external surface of the lung, and extends in an inverse direction,—towards the large bronchial branches. The extreme frequency of the respiration in capillary bronchitis is another distinction, for in ordinary bronchitis it is not usually much hurried. The sputa too are characteristic. But it should be remembered that bronchitis of the larger bronchi may extend to the smaller ramifications, and that capillary bronchitis may supervene upon and complicate simple bronchitis. The diacritic phenomena, both objective and subjective, of capillary bronchitis and pneumonia, are so well marked, that it is difficult to account for the frequent errors of diagnosis. It is of much practical importance to distinguish the two disorders, both as regards prognosis and therapeutics. The initial chill is much less constant in capillary bronchitis than in pneumonia, rarely amounting in the former to more than shivering, and there is reason to believe that the body-temperature is never so high. In the bronchial disorders the debility is earlier, the physiognomy different—more anxious and livid—the respirations quicker, the febrile reaction greater, and the sputa, when present, distinctive. Pneumonia is commonly unilateral, capillary bronchitis bilateral. The physical signs of the two affections are unlike, especially those shown by auscultation, the characteristic rhonchus of each affection having individual qualities. When collapse of the lobules has taken place in capillary bronchitis, the differential physical diagnosis may be less marked, but even here the rational symptoms and the



presence and diffusion of the symmetrical pathognomonic rhonchus should prevent error. Occasionally a case of acute phthisis is met with that at first sight bears some resemblance in the objective symptoms to capillary bronchitis; one is mentioned by Dr. Flint; but physical exploration and a history of tuberculosis will indicate the real nature of the disease. In plastic bronchitis there is subcrepitant rhonchus, but it is limited and never general, and the expectoration is characteristic.

**Morbid Anatomy.**—On opening the chest the lungs do not collapse; their surface at the upper anterior portions often show recent emphysematous enlargement of the air-cells, and is pale, the rest of the surface anteriorly being of a dull red, with patches of bright red; posteriorly the color is darker, and of a chocolate hue. The surface may be uneven, some lobules being quite prominent. The lungs are frequently pulpy and crepitant, but often somewhat doughy in parts, retaining the marks of the fingers, from oedema of the connective tissue. On cutting into the lung it is spongy, and of a deep dull red hue. At times it is dry, and requires to be squeezed to press out a viscid, opaque, grayish-white, or light straw-colored, frothy, muco-purulent matter; or it may be very moist, and a yellowish glutinous matter runs out on cutting into the lung-tissue. When this matter is examined microscopically, it is found to be made up of pus-cells and epithelium-scales suspended in a clear fluid; there are no blood-cells (WATERS). The lining membrane of the larger bronchial tubes is of a uniform dark rose-red hue throughout. The air-tubes are in parts dilated. The little yellow spots sometimes met with, resembling crude miliary tubercles at first sight,—pointed out by Rilliet and Barthez, and called by them and Fauvel, purulent granulations,—are dilated air-vesicles or lobulets, filled with the puriform matter just described, which flows out when they are cut into, the air-cells collapsing. In young subjects the bronchial glands are red, friable, and swollen.

**Treatment.**—If a happy issue of this disorder is to be looked for, the treatment must be strictly conservative; spoliative remedies lessen, if they do not destroy, the chances of recovery. Like similar pathogenetic diseases, it is what is styled self-limited, having an inevitable cycle to travel over. The tendency to death is by apnoea, from the imperfect oxygenation of the blood—the area of available respiratory surface being greatly diminished, both from the morbid state of the minute bronchi, and the accumulation of diseased products in them. The patient must be properly and adequately nourished, and stimulants are often required, at least at times. Tonics should be early administered. The chest may be covered with hot poultices, and an oil-silk jacket worn over them. Dry cupping, in some instances, has seemed to give relief to the urgent symptoms. The writer's experience is favorable to the use of the muriate of ammonia—two grains every two hours—either alone, or in combination with the chlorate of potash. The carbonate of ammonia has long had a certain reputation in capillary bronchitis, especially in the later stages. Whatever effect it may have is probably not due to its supposed stimulant properties, but to specific action upon the diseased tissue and its products.

The stimulant expectorants may be given in the later stages, but it is very doubtful if they are so efficient as alcoholic stimulants at this period. Surer results will probably be got from the administration of pure brandy or wine, particularly French Burgundy. Inhalations of steam generally give much comfort, and often diminish, at least for a while, the distressing breathlessness. Dr. W. Y. Gadberry, of Missis-



sippi, reports tranquillizing effects from the inhalation of sulphuric ether. Emetics—and such as will not depress the system should be chosen—are often necessary, particularly in children, to clear out accumulated secretions from the air-tubes.]

[PLASTIC BRONCHITIS—PSEUDO-MEMBRANOUS BRONCHITIS—  
BRONCHIAL POLYPI—CASTS OF THE BRONCHIAL TUBES.

LATIN, *Polypus Bronchialis*; *Bronchitis Crouposa*. FRENCH, *Bronchite Pseudo-membraneuse*. GERMAN, *Croupöse Bronchitis*.

(DR. CLYMER.)

**Definition.**—*A bronchial affection characterized by the production and expectoration of fibrinous exudation, in the form of membrane or casts, with catarrhus symptoms in the air-tubes, happening at all ages, but more usually between twenty and fifty years, and generally associated with some diathetic disorder, as rheumatism, gout, or scrofula, or with an aneurismal or other tumor pressing upon the bronchi.*

This not common form of bronchitis should not be confounded with cases of diphtheria and croup where the membrane extends from the lungs and trachea to the air tubes. It may be acute, or chronic, or intercurrent.

The first recorded case of plastic bronchitis is in the *Acta Eruditorum*, Leipsic, 1682. Tulpius mentions it (1685), and also Clarke (*Phil. Trans.*, vol. xix, 1697). It has been described by Morgagni, Senac, Michaelis, Cheyne (*Ed. Med. & Surg. Journ.*, 1803), Iliff (*Lond. Med. Rep.*, vol. xviii, 1820), Starr (*Med. Gaz.*, vol. xxv), Cazeaux (*Bul. de la Soc. Anat.*, 1836), Nonat (*Archiv. Gén.*, 2ème serie, 1837), Watson, Fauvel, 1840, Thore, fils (*Archiv. Gen.*, 4th ed., 1849), Fuller, Peacock (*Trans. of the Path. Soc. of London*, vol. v, 1854, and *Times & Gazette*, vol. ii, 1854), Michel Peter (*Gaz. Héb.*, 1863), Valleix (*Guide du Méd. Prat.*, 4th ed., 1866), and Dr. Stephen Rogers, of New York (*Transactions of the State Medical Society of the State of New York*, 1866).

**Symptoms.**—Like all diseases of whose pathogeny little is known, and where the result only of some ill-understood morbid condition is recognized, the described symptoms will be found to vary much in kind, degree, and duration. In an analysis of 30 cases, the attendant general and local symptoms in 11, were those of slight catarrh, and did not attract especial attention until the appearance of the peculiar sputa; in 9 the general symptoms were acute, like those of pneumonia or simple bronchitis; in 2 there was a low type of fever resembling typhoid; and in 7 the initial symptom was hæmoptysis. The symptoms immediately precedent of the characteristic expectoration are: a sense of constriction about the chest, with short breath, and a dry, hard, paroxysmal cough; these may last for several days. The breathlessness suddenly becomes urgent and alarming, the lips are blue, the face is swollen and livid, the extremities are cold and discolored, and after a severe fit of strangling cough, the membrane is expelled, usually along with the white, glairy, adhesive sputa of simple bronchitis, or with a little blood, or it may be quite dry. Immediate relief commonly follows, and lasts until there is fresh accumulation and another exacerbation, followed by detachment, expulsion, and alleviation. There may be several paroxysms in the twenty-four hours; in chronic cases they often gradually lessen in severity, and the membrane is after awhile expectorated with but little difficulty. Even in some acute cases, there would seem to have been only a temporary aggravation of

the symptoms of bronchitis just before the plastic expectoration, which, easily brought up with a hem or a rising in the throat, and a feeling of sickness, was expelled by the mouth or through the nostrils with slight effort, and without the hard, ringing cough, or threatening suffocation.

The *physical signs* of plastic bronchitis, most generally met with in upper chest regions, are : natural resonance, though it may be diminished over the affected region (FULLER) ; respiratory murmur at first rude, with sibilant and sonorous rhonchi. A distinct click (called by Cazeaux *bruit de soupape*) is occasionally heard (CAZEAUX, BARTH, CANE, GORDON, FULLER) ; a limited subcrepitant is met with, though not so frequently as Fauvel has reported, many, if not most of his cases having been instances of capillary bronchitis with tough viscid expectoration. Obstruction of the air-tubes may happen even to complete occlusion, causing collapse of the air-cells of one or more lobules ; there are then the signs of pulmonary consolidation,—dulness, and absence of all sounds over a circumscribed space. The moist sounds are frequently heard after expectoration of the membrane.

The membrane may be in shreds, like bits of skin, or in pellets, or in cylindrical casts, like boiled maccaroni, and delicately branched, of a grayish-white color, or of a rose-tint, or brownish-red, resembling decolorized coagulum.

In some cases the expectoration of plastic membrane ceases after a few days, and recovery is rapid and good. In others, the acute symptoms abate, but false membrane continues to be occasionally expelled, after fits of cough and breathlessness, for several weeks, with intervals of comfort, and, in some instances, of apparent good health. The coughing spells are often brought on by excitement or exertion. Cases are recorded where this disorder has lasted during one, two, three, or more years, either continuously, or at certain seasons. One instance is related in which there was catarrhal fever for four successive winters, each attack lasting five weeks, and ending in the expulsion of false membranes, the patient during the intervals being quite well. In Dr. Fuller's case there was a recurrence of catarrhus symptoms every winter for eleven years, accompanied by the expectoration of small pieces of plastic matter, after which time the disease was not limited to the winter months, but followed any exposure to cold or damp, or change of weather. Relapses may happen. In the chronic form the general appearance is unhealthy. The subjects of this disorder are habitually short-breathed and pallid, with livid cheeks and lips, and incurvated nails.

**Prognosis** depends upon circumstances, and upon the presence and nature of any complicated pulmonary disorder. In 34 cases analyzed, 20 seemed to have ultimately recovered perfect health, and 10 to have died sooner or later ; in 10 it is stated that false membranes continued to be expectorated at intervals for a long period. The acute form of plastic bronchitis runs its course rapidly to a happy or fatal issue, and in adults, however alarming or urgent the symptoms, usually ends in recovery, particularly when the false membrane is freely expectorated ; or it may pass into chronic bronchitis, with occasional exacerbations from fresh cold from time to time, which subside after membranous expectoration. When the general and local symptoms are slight at the outset, the disorder is apt to be of longer duration.

**Diagnosis.**—The expectoration of plastic membrane in rounded pellets or cylindrical casts is really the only distinctive symptom of this disorder. The diacritic signs between plastic and capillary bronchitis are usually sufficiently marked, the former being generally localized in one lung, and

one region, whilst the latter is symmetrical and shows a great tendency to extend; the subcrepitant rhonchus, when met with in plastic bronchitis, is limited; in capillary it is diffused. From pseudo-membranous croup and diphtheria it is distinguished by the local symptoms and physical signs, as well as by the character of the false membranes expelled.

**Nature and Causes.**—Plastic bronchitis happens oftener in males than in females; in 44 cases 33 were males and 11 females. It is not limited to any period of life, though it is most common in middle age. Cheyne thought that it was most frequent in old persons, and Fauvel believes that it is most often met with in infants, and is with them a very fatal disease. In 34 cases where the age is given, there were: from 5 to 20 years, 11 cases, from 20 to 50, 21, and from 50 to 60, 2. Morgagni mentions a case in a man seventy-eight years old. In some instances the health is excellent until the sudden onset of the disorder; generally, however, it occurs in persons of delicate constitution, and who are suffering, or have suffered, from pulmonary disease. In some of the recorded cases an inherited strumous diathesis is stated, or a liability to catarrhus affections or asthma, or there had been previous hæmoptysis. Some had suffered from scurvy, others had been gouty. It is not infrequently associated with rheumatism (BUCKLER), and has apparently been produced by the pressure of tumors, malignant or benign, upon the lung-tissue, or by an aortic aneurism.

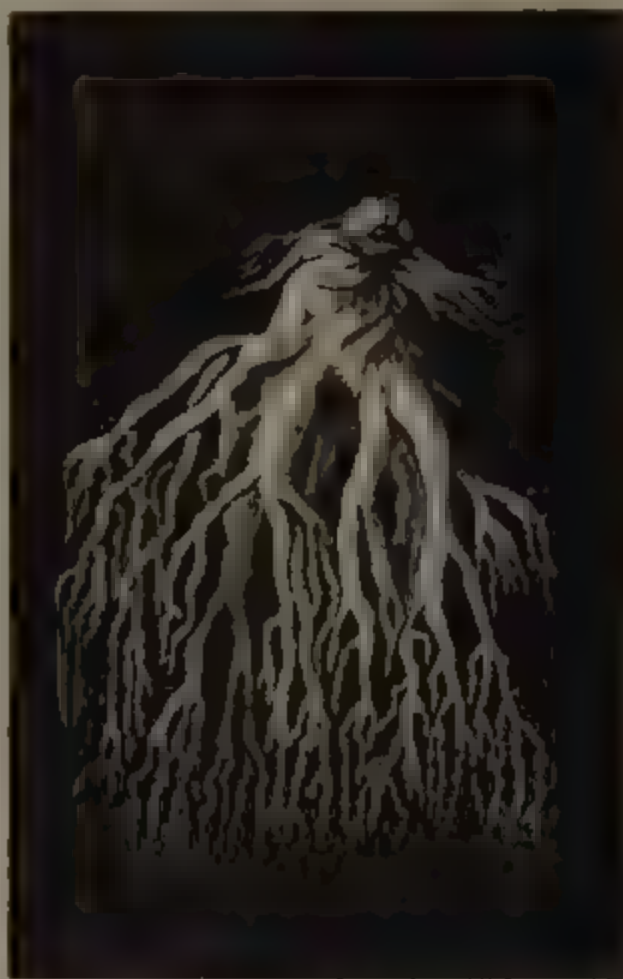
The characteristic expectoration was described by both Ruysch and Morgagni, and is figured by Cheselden in his *Anatomy* (1722). Michælis, and Cheyne\* divide bronchial polypi into two classes: (1.) A moulded coagulum of blood: (2.) Condensed, lamellated, solid, or tubular membrane. The second class belongs to that form of fibrinous exudations where the blood corpuscles escape along with the liquor sanguinis, instances of which are met with in pericarditis and pleurisy (WATSON, PEACOCK). The membrane when expelled may be in shreds; or in the form of oblong or rounded bodies, sometimes as large as a filbert, which when macerated in water for a short time gradually expand into a stem and branches; or like cylindrical casts of the bronchi, varying in diameter, from a crowquill to a writing-pen or drawing-pencil, from one to three or four inches in length, with small divergent branches, and minute terminal points, resembling some vegetable roots and their radicles or beard. As many as ten distinct subdivisions have been seen: they come from the second, third, and fourth bronchial ramifications. The color is dull white, or occasionally brownish, from the admixture of blood. The casts are composed of tough, concentric fibro-membranous laminæ, between which a fine probe may be passed, and which seem to have been deposited in successive layers. The main stems may be solid or hollow; often there is no distinct cavity in the central trunk, but the larger branches in places are hollow.

The wood-cut represents one of the casts expectorated in the case of Dr. Stephen Rogers, of New York (*Transactions of the Medical Society of the State of New York*, 1866); their number was from one to half a dozen a day, through a period of about three and a half months. In the opaque semi-fluid mass, the arborescent character of the casts was quite distinct, but was thoroughly brought out by an admixture with, and gentle agitation in, water. They were always cylindrical or solid, and varied from  $\frac{1}{8}$ th to  $\frac{3}{4}$ ths of an inch in diameter at the base of the cast, the extreme length of some of the larger ones reaching nearly four inches in length. The case was one of malignant disease of the lungs.

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\* [*Ed. Med. and Surg. Jour.*, t. iv, 1803.]

FIG. 39



Cast in plastic bronchitis, one half natural size, involving bronchi of an entire lobe of lung—(S. KODAK.)

The microscope shows these casts to have a pretty uniform structure. At first sight they seem to consist of parallel and nearly straight fibres, intermixed with numerous rounded bodies about the size of blood-corpuscles. On a more accurate examination, the fibrous appearance is found to be deceptive, and to be produced by the edges of delicate, transparent, nearly structureless laminae, closely and concentrically arranged. Between these laminae the corpuscles exist in large numbers, more or less globular, for the most part uniform in texture and solid, but sometimes having a somewhat irregular cavity. Acetic acid produces expansion and partial solution of the laminae, but has no evident action upon the corpuscles. Towards the terminations of the minutest ramifications of the casts there are found, mixed more or less abundantly with these structures, compound granular cells, irregular clusters of oil-globules, and ovoid bodies containing pigmentary matter (BRISTOWK, *Trans. of Pathological Society of London*, vol. v, 1864): these are fibrine-exudations, which have undergone some degenerative process.

There is no evidence that the formation of plastic material in the bronchi is due to any extension to, or involvement of, the submucous tissue in the morbid action; nor is there any proof of excess of fibrine in the blood. The pathogeny of this disorder is to be sought for in some peculiarity of individual constitution, or in the cause giving rise to the disease; and it must, for the present at least, be looked upon as the result of a specific morbid process, and should be more fully and carefully studied.

**Treatment**—In the acute form, muriate of ammonia and the alkalies may be given, with an occasional emetic, and the inhalation of the vapor of hot water, and local counter irritation. If the general symptoms are



typhoid, stimulants, tonics, and nourishment are required. In the chronic form the muriate of ammonia and iodide of potassium (THIELFELDER), with tonics, arsenic, cod-liver oil, and a residence in a warm climate, are recommended. Warm clothing should be worn, and particular attention paid to the state of the skin. From the relief afforded by breathing warm dry air, and remaining in a warm room in the case of Dr. Fuller's patient, the Turkish bath might be tried. The patient should be carefully protected against damp and sudden changes of weather.]

DILATATION OF THE BRONCHI.—Syn., BRONCHIECTASIS.

LATIN Eq., *Dilatatio* ; FRENCH Eq., *Dilatation* ; GERMAN Eq., *Erweiterung* ; ITALIAN Eq., *Dilatazione*.

**Definition.**—A cylindrical, or fusiform, or saccular dilatation of a bronchial tube, or of several tubes, at one or more points, or through a considerable portion of the tubes.

**Pathology.**—The true bronchiectasis arises quite independently of all other pulmonary affections; but there are several minor varieties which result from other changes in the lungs, such as from whooping cough; suffocative capillary bronchitis; structure of bronchi; long-standing indurations of lung-substance, tubercular or inflammatory; the remains of chronic tubercular cavities, or abscesses in the lung-tissue.

The most important forms of the true bronchiectasis are—(1.) *The general or uniform*, in which there is a cylindrical or fusiform dilatation of a tube, or of several tubes throughout considerable stretches of their extent; (2.) *The saccular*, or ampullary, in which there occurs an abrupt dilatation of a tube at a particular point, or at several points. When dilatations exist in neighboring bronchi, communications may become established between them, and to such an extent that several bronchial tubes open into one common cavity. Wasting of the muscular and elastic coats is a usual result, and there is degeneration of their tissue; and in consequence the dilated tubes open into each other without any truly ulcerative process, as shown by Dr. G. T. Stewart, from whose elegant description of "*Dilatation of the Bronchi*," in the *Edin. Med. Journal* for July, 1867, this account of *Bronchiectasis* is taken. Copious yellow or inspissated mucus, sometimes with casts of tubes, and often very fetid, exists in the dilated tubes, associated with crystalline fats and fungi. In some cases the tissue of the lung is condensed, from cirrhosis or fibroid degeneration of the lung, which may proceed to more intense induration, ulceration, and even gangrene. The site of *bronchiectasis* is usually the lower lobe and the middle lobe of the right lung. It occurs towards the apices sometimes. Usually it affects many bronchi and occurs in both lungs. The essential element of the lesion in bronchiectasis is atrophy of the bronchial walls, which rapidly yield to the pressure of air. The enfeebled and dilating condition of the bronchi favor the accumulation of the mucus secreted by the mucous membrane, which, accumulating and undergoing decomposition in the dilated cavities, leads to inflammation, and the forma-



tion of villous processes, to increase of connective tissue, and to further consolidation of surrounding lung.

**Symptoms and Clinical History.**—The disease comes on insidiously; but gradually symptoms of bronchitis become well marked. Breath and sputum become fetid, and general health is impaired. Decomposition of the secretion is followed by lung consolidation, ulceration, abscess, or gangrene. Perforation of the pleura, empyema, or pneumothorax may prove fatal; or death may result from exhaustion due to the constant discharge of the sputum. A peculiar febrile disturbance resembling *septicæmia* may also terminate life. Unfortunately, the tendency is to a fatal result; but recovery may take place—1st. *From cretification of the contents of the dilated tubes, and the conversion of their walls into a sort of fibrous capsule*; 2d. *From penetration of the pleura and thoracic parietes and discharge of the contents outwards*. Dr. Stewart considers the disease as probably hereditary and constitutional.

The most characteristic symptoms are, *the odor of the breath, the characters of the sputum, and the cough*. The fetor of the breath is something distinct and different from that of gangrene, and is not present in all cases, and not till decomposition of the bronchial secretion has taken place. *Blood* is sometimes expectorated with the sputum, sometimes in large quantities, as in phthisis, at other times merely in streaks over the expectoration. *Cough* is frequent, and occurs in paroxysms, but is moist, soft, and usually quite painless; and after the fit of coughing, large quantities of the sputum are brought up with difficulty. Exertion induces dyspnoea. *Auscultation* gives varied sounds—cavernous, moist râles, sometimes even gurgling, and the site of these sounds aids to distinguish them from those of phthisis, also the history and progress of the case.

**Treatment.**—Opiates, to relieve cough; balsamic remedies (*tolu, tar, turpentine, copaiba, cubebs*), and astringents like *catechu* or *rhatany*, with the use of counter-irritants and the inhalation of variously medicated vapors, are all useful aids in ameliorating the condition of the patient. Inhalation of disinfectants capable of being so used, such as *creusote, carbolic acid, sulphur, vapor*, and the like, are of special service.

[The foregoing article of the Author being a digest of Dr. T. Stewart Grainger's contribution to the morbid anatomy and symptomatology of Bronchiectasis, in the *Edin. Med. Jour.*, 1867, the Editor's section on Dilatation of the Bronchi in the first American Edition is reprinted.]

## [DILATATION OF THE BRONCHI—BRONCHIECTASIS.]

FRENCH, *Dilatation des Bronches*; GERMAN, *Bronchiectasie*.

(DR. CLYMER.)

**Definition.**—*A variety of chronic bronchial disorder, whose special anatomical character is dilatation (cylindrical, globular, or mixed), of the second, third, or fourth divisions of the air-tubes of one or more lobes, generally the lower and middle, with atrophy of their muscular and elastic coats, more often in the right lung, sometimes affecting both lungs. at-*

*tended with cough and fetid muco-puriform expectoration, and most frequent at or after middle life.*

This change in the calibre and structure of the air-tubes is more frequent than generally stated by systematic writers, and is especially interesting and important for the reason that in many cases its symptoms and signs closely resemble those of one of the stages of pulmonary tuberculosis. Much remains to be done in the study of its nature and pathogeny.

**Causes, Nature, and Morbid Anatomy.**—Bronchial dilatation, in the present state of our knowledge, may be regarded as a consecutive disorder, following acute and chronic catarrhus affections, collapse of the pulmonary lobules, pneumonia, and pleurisy. It is supposed to be most common in persons of a lymphatic habit. It is met with at all ages, but is more frequent after forty. In 24 cases there were: between 5 and 20 years, 4; between 20 and 40 years, 6; between 40 and 70, 10; and over 70, 3. In 27 cases, 12 were males and 15 females (LEBERT), though the difference in the proportion of the sexes is generally considered much greater. In Lebert's 27 cases, the disease was limited to one lung in only 9, or one-third, the right lung being affected in 5 and the left in 4, and the bronchi of the opposite lung being more or less dilated. In 43 cases observed by Barth, it was unilateral in 23; its site was the right lung in 11, the left lung in 10, and in 2 the lung was not noted. In 20 the bronchi of both lungs were involved, in 10 to nearly the same extent, more marked in the right lung in 7, and in the left lung in 3.

There are three forms: (1.) Uniform and cylindrical, tubes naturally the size of a crowquill becoming as large as the finger; (2.) Globular, or pouch-like, of the size of a pea, filbert, walnut, or hen's egg; a variety of this form is a series of small spherical dilatation—the tubes intermediately having its natural calibre—giving to it a beaded look; (3.) Mixed.

The dilatation is rarely confined to a single bronchus, or one of its branches, but generally includes all the bronchial ramifications of a lobe, but in different degrees. It sometimes forms a pouch, or cul-de-sac, on the surface of the lung. It seems rarely to begin in a primary bronchus, but usually in the second, or third, or fourth divisions, and sometimes in those of lesser size. The enlargement usually begins at the origin of a tube, though sometimes this may be narrowed. The dilated branches may abruptly contract near its terminal extremity, and become obliterated, or end in a spindle-shape or spheroidal sac. Where bronchial dilatation is in both lungs, it may be limited to the larger tubes, about their origin; sometimes all the bronchi of a single lobe, not homolateral, in each lung may be affected, as the upper right and the left inferior lobes.

The dilated bronchi generally contain some puriform matter, similar to the expectoration. The tissue of the tubes is more or less changed in texture; the mucous membrane is commonly thickened, granular, often softened, sometimes ulcerated, and of a dark, or livid red color. There is rarely any false membrane; but a cheesy, friable substance is occasionally met with. The most constant and distinctive nutritional changes are in the muscular and elastic tissues, which are thinned, and lose their individual elementary characters. The lung-tissue about the dilated bronchi is condensed, indurated, shrivelled, and almost always altered in structure, having undergone more or less degeneration, probably fibroid. In a case reported by Dr. Legendre, it was of scirrhus consistence, and resembled, in color and density, the tissue of the uterus. When one lung is affected in greater part or wholly, its bulk is usually much les-

sened, while that of the opposite lung is increased, it occupying from one-fourth to one-third more of the thoracic cavity. The evidences of pulmonary emphysema are found in a large proportion of cases.

**Symptoms.**—Bronchial dilatation may exist for some time, and to a large extent, without affecting the health, the strength being good, the complexion natural, no anxious look, and little or no loss of flesh, the patient complaining only of having had for some time a paroxysmal cough, with occasional acute catarrhus attacks, and more or less expectoration. The symptoms, however, are generally in proportion to the number of implicated air-tubes, the size of the dilatations, the condition of the mucous membrane, and the amount of disabled lung-tissue. In most cases the face is more or less pallid or livid, there are weakness and fatigue on exertion, marked emaciation, a constant feeling of discomfort about the chest, breathlessness—amounting to oppression on lying down, particularly on the affected side, or after fits of coughing—and a constant racking cough, day and night, relieved by expectoration, which is abundant, and sometimes excessive. The properties of the sputa vary according to the nature of the changes in the tissue of the tubes and air-sacs. They may be those of chronic bronchitis,—muco-puriform, opaque, greenish-yellow, aerated on their surface, or quite purulent and offensive; but the characteristic expectoration, when the disease is advanced, and tissue degeneration has set in, consists of small clots of purulent matter, less coherent and compact than the nummular sputa of phthisis, floating in an abundant, somewhat frothy, watery fluid, the whole having a curdled look, and a peculiar stinking smell, like that of sebaceous acid (SKODA). In this stage of the disorder there is often hectic, though in many cases no febrile excitement exists, unless some acute intercurrent disorder happens. Where breathlessness is much complained of, it generally indicates acute pulmonary disease, or emphysema, or heart complication. In 33 cases observed by Barth, heart disease coexisted in 16, with the usual symptoms of disordered pulmonic and systemic circulation—palpitations, dyspnoea, cyanosis, and œdema of the extremities.

The *physical signs* depend upon the form, size, and extent of the dilatation, and the condition of the surrounding lung-tissue. On *inspection*, depression may be seen over the site of the affected portion of the lung, but when this is at all marked there is reason to believe that it is generally caused by former pleuritic effusion, though where the greater part, or the whole of a lung is involved, there may be marked retraction of one side. The *application of the hand* detects increased fremitus. *Percussion* often fails to give any trustworthy sign. Skoda says that it is only of value if the dilated tubes are large and numerous, and the surrounding lung-tissue condensed, when tympanitic resonance, even to the degree of the amphoric or cracked-metal sound, will be heard. It is, however, doubtful whether this happens except in large globular dilatation. Ordinarily the percussion-note is dull, raised in pitch, and of tympanitic quality. The percussing finger feels diminished elasticity. The percussion signs also vary according to the amount of secretion in the air-tubes. Skoda thinks that *auscultation* in this disease is worth little unless frequently repeated, since it is only the continuance of the consonant rhonchi and bronchial breathing that gives any reliable information. The auscultatory signs modified by, and proportionate to, the local condition, are: feeble or absent vesicular murmur, harsh respiration, which may be blowing or cavernous, exaggerated vocal resonance, bronchophony, and cavernous voice, or sometimes pectoriloquy. These

alterations of the breath-sounds are constantly masked, or temporarily replaced by rhonchi of various degrees of moistness even to gurgling, which are best heard by making the patient breathe deeply, or after coughing.

The *duration* of this disorder is generally long. Commonly progressive, it may become stationary, or, in rare cases, so far as relates to the symptoms, greatly amend. Patients with bronchial dilatation generally die of complicating diseases, acute or chronic. It greatly favors attacks of acute bronchitis or pneumonia. Organic heart disease is frequently coincident. In two of Barth's cases death was caused by sudden and excessive hæmoptysis, no tubercles being found in the lungs. Pulmonary tuberculosis or cancer may shorten life; in 43 cases, 11 were tuberculous and 11 cancerous. Superficial dilated bronchi may open into the pleura, and be followed by empyema and pneumothorax.

**Prognosis.**—When the dilatation occurs in young persons, is limited, not progressive, and hæmatisis is not interfered with, the disease causes but slight and temporary discomfort, and persons may live on for years, pursuing their callings. The prognosis is unfavorable in proportion to the age of the subject, the extent and degree of the bronchial and lung lesions, the presence or absence of complicating disorders, the state of hæmatisis, and the general condition.

**Diagnosis.**—Skoda regards the characteristic sputa as the most reliable diacritic sign; but these do not always exist. When the physical signs are unilateral, and particularly on the right side, and at the base of the chest, they indicate bronchial dilatation rather than tuberculosis. When unilateral, but generally diffused over the lung, in bronchial dilatation they are less marked toward the superior regions. In pulmonary tuberculosis, with excavation, the opposite lung, especially at the summit, is usually affected; this is not the case in bronchial dilatation. The general symptoms, and the history of the case, should generally aid in a differential diagnosis.

**Treatment.**—Muriate of ammonia, nitric acid, the balsams, sulphur, sulphur baths, arsenic, cod-liver oil, tonics, iron, &c., are all recommended, and amendment, if not a cure, sometimes follows their use. Skoda says the best treatment is the inhalation of vapor impregnated with turpentine or tar. The fetid breath may be corrected by inhalations of creasote (G. STEWART). Drugs, however, can only be immediately palliative, and an arrest of the morbid process can only be hoped for by attention to the nutrition of the patient, according to rules already laid down.]

## DISEASES OF THE LUNGS.

### EMPHYSEMA.

LATIN EQ., *Emphysema*; FRENCH EQ., *Emphyseme*; GERMAN EQ., *Emphysem*; ITALIAN EQ., *Emfisema*.

**Definition.**—(1.) Vesicular Emphysema—*A misshapen enlargement of the air-cells of the lungs, by dilatation of them, attended with gradual effacement of the functional bloodvessels distributed over their walls; anæmia of the lung in the affected parts, tending to dilatation of the right side of the*

heart, with anasarca. (2.) Interlobular Emphysema—Air infiltrating the meshes of the connective tissue of the lungs.

**Pathology.**—Some confusion has arisen from the use of the term "*Emphysema*" (which means the presence of air in the connective tissue) to designate dilatation of the air-cells of the lungs. Here the air is where it ought to be; but the air-cells are too large and misshapen, and contain too much air; and being limited in this way to the vesicles of the lung, this form of the disease was named by Laennec "*vesicular emphysema*." In 1698 the disease was identified and well described by Sir John Floyer as existing in broken-winded horses; and Dr. Baillie, in subsequently describing enlargement of the air-cells of the lungs, refers to Sir John Floyer's description as applicable to the lungs of the human subject.

The dilated air-vesicles vary from the size of millet-seeds to that of Barcelona nuts, or even larger; but when they form a great expansion, it is probable that many air-vesicles are dilated into one common cavity by rupture of the partitions which separate them from each other. The dilated vesicles may be seen clearly through the pulmonary pleura: they also protrude from the surface of the lung. The emphysematous parts are pale, and sometimes quite white; the tissue is drier than normal; it cannot be easily emptied of air, resembling the lungs of a reptile rather than those of a human being. It possesses fewer capillary vessels; and they become obliterated by distension of the air-cells. The lung is therefore anæmic, and contains less moisture than the normal lung. It is so dry and light that it floats much higher in the water than a healthy lung.

**Causes.**—Several theories have been put forward to explain the mechanism which produces emphysema. Dr. Elliotson considers a want of due expansion of the lungs as the most common cause of emphysema. "Whatever prevents any one part of the lungs from expanding when the thorax expands—whether it be a material obstruction of the bronchial ramifications, or a compression of them, or whatever else, it will occasion those parts which remain dilatable to keep dilated in a corresponding increased degree, in order to fill up the vacuum which the expansion of the chest occasions. When we inspire, we dilate the chest, and the air rushes down the trachea, and the lungs follow the dilated portions. If there be any part [of the lung] that will not dilate, then other parts are over-dilated to fill up the vacuum; and in that way those parts which we distend are *over-distended*, in order to compensate for the want of distension in other parts; and when once *over-distended*, they are often unable to recover themselves, just as is the case in other parts of the body—the urinary bladder, for example. I presume it is on this account that dilatation of the air-cells is so common in persons laboring under chronic bronchitis, especially where the membrane is most thickened, and where the secretion, if there be any, is tough and adherent, so as to produce obstruction" (*Practice of Medicine*, p. 851). Dr. Watson follows Laennec in believing that the dilatation in the outset is mainly due to the imprisonment of air within the cells under the influence of disease, such as imperfectly ob-



structed tubes, so that air enters the vesicles more readily than it can escape from them. More and more air then accumulates, and is incarcerated in certain air-cells, which, yielding to the distending force, lose their elasticity and become permanently large.

But, to confirm this view, it ought to be shown that the dilated air-cells are those belonging to the tubes in which the obstruction exists; and Dr. Elliotson is of opinion that they are not those in which there is obstruction. Dr. Gairdner, indeed, has very ably demonstrated this in connection with the occurrence of emphysema in bronchitis (see page 695, *ante*). The disease forms one of the most serious complications of *bronchitis*; and the tendency to vesicular emphysema appears to be hereditary—60.4 per cent. acknowledging hereditary transmission (FULLER).

**Symptoms.**—In typical cases of emphysema the patient is short-winded, and distressed by a constant sense of fulness and oppression at the chest, and generally seeks advice after suffering and discomfort have become too great for him to bear any longer.

The difficulty of breathing is often aggravated by spasm, as in *asthma*; and emphysema is a frequent consequence of that disease—the one reacting on the other, so that the phenomena of each are mutually aggravated.

The physical signs are, incompleteness of the act of expiration, the thorax remaining prominent and round over the emphysematous lung. In spare persons the clavicles are not well defined. Percussion, over the bulging parts especially, yields a peculiarly clear and resonant sound; and although there is thus shown to be abundance of air underneath the part which yields such a sound, yet the *vesicular murmur* of breathing is extremely indistinct, showing the air is not in motion there. It is shut up in the enlarged air-cells (WATSON).

The disease tends to impede the circulation through the lungs, and so to produce hypertrophy, with dilatation of the right side of the heart, venous congestion of the head and face, attacks of palpitation, paroxysms of cough and dyspnoea, œdema of the feet and legs, general anasarca; and dropsical effusion is a frequent termination of the disease.

**Treatment.**—Apart from the management of the bronchial congestion, on the principles already given under *bronchitis*, little can be done for the special treatment of *emphysema*. If bronchial spasm prevail, Hoffman's anodyne may give relief. It is the *spiritus ætheris* of the British Pharmacopœia, [*spiritus ætheris compositus* (U. S. P.),] of which *thirty to sixty minims* may be prescribed in *camphor water*, or in *spiritus ammoniæ aromaticus*, or in *volatile tincture of valerian*; or it may be combined with stimulant doses (*one grain*) of opium; or with *twenty minims* of the *ethereal tincture of lobelia, belladonna, conium*; or the *ethereal tincture of Indian hemp and hydrocyanic acid* may each in turn be found of service (FULLER).

[The asthmatic fits may often be checked by the internal use of chloroform. The inhalation of chloroform or ether, or very dilute carbonic acid, is said to be relieving. Smoking belladonna, or stramonium, or

camphor, will often abate the distressing breathlessness, and Dr. Fuller says, that the seed-pods of the *datura tatula* smoked in a pipe, he has known to succeed after stramonium has failed. Trousseau strongly recommends the arsenical cigarette; and breathing the fumes of bibulous paper, which has been soaked in a saturated solution of the nitrate of potash and then dried, has sometimes wrought immediate relief of the paroxysm. Drinking a strong infusion of coffee, or iced-water, often quiets spasm. Nothing, however, is so sure as a full dose of opium given with sulphuric or chloric ether: R. *Tincturæ Opii*, fʒj. *Ætheris sulphurici*, fʒij. M. *Sixty drops every twenty minutes.* Dr. Fuller adds twenty drops of the *ethereal tincture of lobelia* to each dose.

The preparations of arsenic, and particularly the *arsenate of antimony*, have been lately recommended as not only ameliorating the condition of the lung-tissue, and making the patient comparatively comfortable, but as actually curative (*De l'arsenate d'antimoine dans l'emphysème vésiculaire des poumons*, par le Dr. Charles Isnard (de Marseille), 1868).]

Dry cupping between the shoulders often relieves passive pulmonary congestion; and if an attack is imminent, an emetic, or unloading the bowels by a dose of the *compound jalap powder*, may prevent its accession or moderate the paroxysm.

### PNEUMONIA.

LATIN Eq., *Peripneumonia*; FRENCH Eq., *Pneumonie*; GERMAN Eq., *Lungenentzündung*—Syn., *Pneumonie*; ITALIAN Eq., *Pneumonitide*.

**Definition.**—*Inflammation of the true pulmonary tissue, which in its acute sthenic form, uncomplicated by constitutional or specific diseases, runs a definite course, expressed by severe febrile symptoms, which come on suddenly, attaining in a few hours a great intensity, and which undergo a no less sudden abatement or improvement between the fifth and tenth day, in proportion to the severity of the disease; and while the local productive results of inflammation in the form of the lung-lesion are yet intense, but which are eventually removed.*

The natural course of pneumonia is materially modified by constitutional or specific diseases, especially if any organ, such as the kidney, the heart, or the liver, is involved; or it may be modified by the secondary contamination of the blood by absorption of lung-exudation in the latter stages of the disease, tending to inflammation of the other lung, to pleurisy, to pericarditis, or to blood-coagula in the cavities of the heart or great vessels.

**Pathology.**—In the continental cities of Europe about eight per cent. of all the deaths are caused by pneumonia; and while about three per cent. of all diseases are due to the same cause, about two per cent. of all cases of disease in hospital are referable to pneumonia. It is subject to more or less well-defined periodic fluctuations, and sometimes appears as if it were epidemic; while its prevalence has been observed to be very much coincident with that of typhus fever (Niemeyer, *New Syden. Society Year-Book*, 1862).

[The general prevalence of this disease makes it one of great interest. During the late war it was very common in both the United States and

Confederate Armies, and the mortality-rate was high. In the United States Army, during the year ending June 30, 1862, the number of cases of pneumonia reported was 11,061, and 2134 deaths; and during the year ending June 30, 1863, there were 20,466 cases, and 4957 deaths; total for the two first years of the war, 31,527 cases, and 7091 deaths; and this out of a total of 8098 deaths from all the inflammatory diseases of the respiratory organs, giving a ratio of deaths to cases of 1 in 6.8 in the Atlantic Region, and 1 in 3.8 in the Central Region (*Circular No. 6, Surgeon General's Office, War Dept., 1865*). In the Confederate Army during a period of nineteen months (January, 1862, to July, 1863, inclusive), 17.1<sup>1</sup>/<sub>10</sub>ths per cent. of the mean strength, on an average, had pneumonia; and it gave 2.7 per cent. of all cases of disease and wounds entered upon the field reports; and 3.15 per cent. of all the cases of disease and wounds entered upon the hospital reports. In a period of fifteen months (January, 1862, to March, 1863, inclusive), there were in certain general hospitals in the State of Virginia, 4774 cases of pneumonia reported, and 1261 deaths, a mortality of a fraction more than one fourth the cases, or more exactly 22.86 per cent., or 1 death in 3.78 cases. During four months, September, 1862, January, February, and March, 1863, 1527 cases of pneumonia were entered upon the registers of the general hospitals in and about Richmond, Va., with 405 deaths. For this period the ratio of deaths from pneumonia to the entire number of deaths from all causes was 19.22 per cent.; whilst on the other hand the cases of pneumonia during these four months was only 3.54 per cent. of the entire number of cases of all diseases treated. For four months, April, May, June, and July, 1863, there were 108,165 cases in all the general hospitals of Virginia, with 2705 deaths, the ratio of deaths from all causes to the entire number of cases treated, being 2.5 per cent., or 1 death in 39.98 cases. 21.29 per cent. of the deaths from all causes were from pneumonia, whilst it made only 2.16 per cent. of the entire number of cases of all diseases. 24.14 per cent. of the cases of pneumonia ended fatally, or 1 death in 4.05 cases. In the General Hospital at Charlottesville, Va., nearly one-third, or, more exactly, 31.9 per cent. of the cases of pneumonia were fatal, or 1 death in every 3.12 cases; the ratio of deaths from pneumonia to the entire number of deaths from all causes, 23.84 per cent., or 1 death from pneumonia in 4.17 deaths from all causes, including pneumonia. On the other hand, the cases of pneumonia were less than one-twenty-third of the total number of cases (4.32 per cent.), or, more plainly, 1 case of pneumonia in 23.13 cases of all diseases, pneumonia included. In General Hospital No. 1 (Confederate), Savannah, Ga., during a period of twenty-five months (December, 1861, to December, 1863, inclusive), more than one-third of the entire deaths from all diseases were caused by pneumonia, whilst the cases of pneumonia to all diseases was in the ratio of 1 to 19.32; a little over one-third, or 1 in 3.18, or 31.35 per cent. of all the cases of pneumonia were fatal (*J. JONES, U. S. Sanitary Commission Med. Memoirs, 1868*).

From January 1 to May 1, 1864, 784 cases of pneumonia, happening amongst the U. S. colored troops, were treated in the hospital attached to Benton Barracks, Mo., of which 156 died. Besides the above there were 675 cases of measles, of which 130 died, death being caused mainly by pneumonia (*IRA RUSSELL, U. S. S. Com. Med. Mem.*).

In the British Army during the Crimean war, the deaths from pneumonia to the number of cases of the disease is reported at 1 to 3.6.

From the foregoing statistics it would appear that the death-rate from pneumonia is much higher in armies than in civil life. Many of the reported

cases of pneumonia, in both the United States and Confederate Armies, were intercurrent affections, particularly in measles, chronic camp diarrhoea, and malarial toxæmia, and this measurably explains the great mortality, without reference to the methods of treatment. The writer is satisfied, from personal observation, as already intimated at p. 703, that many cases of capillary bronchitis were reported as pneumonia. The prevailing type of the disease in both armies was adynamic.]

The phenomena which are appreciable during the natural course of a disease which is uncomplicated and unmodified by any other disease or by treatment, afford the best means of studying its pathology. To Dr. Parkes, the Emeritus Professor of Clinical Medicine in University College, science is indebted for the records of such a case (*Med. Times and Gazette*, Feb. 25, 1860). The case was one of acute sthenic pneumonia, which occurred in a well-built temperate man of about ten stones weight, and which Dr. Parkes conscientiously believed could be left without any treatment (beyond the application of a few leeches) to the unassisted processes of nature.

The characteristic phenomena of pneumonia in the majority of cases are evolved in a regular and consecutive manner; and they may be considered under the three following heads:

1. *The Course of the Pyrexia as Measured especially by the Thermometer and the Pulse.*—In the case which Dr. Parkes records, the commencement of the pneumonia is dated from the *shivering*; and the patient was admitted to hospital on the *third day* of the disease, which was completed at four P.M. of Jan. 7, 1860. He was then intensely febrile, with flushed cheeks, constant cough, viscid bloody pneumonic expectoration, hurried breathing, with crepitation and bronchial respiration over the posterior base of the left lung. The conjunctivæ were a little yellow. Thermometrical observations in the axilla were taken hourly during the day—from nine in the morning till eleven and twelve at night—by Dr. Ringer and Mr. Miller; and the following Table gives the general results—records of the pulse and respiration being also given, for the purpose of comparison with the temperature and with each other.

During the *third* and *fourth days* of the disease the temperature was uniformly high, the difference between the maximum and minimum being only 1.6° Fahr. on the *third*, and 0.6° Fahr. on the *fourth day*. The *fourth day* was the most febrile, both as to mean temperature and as to the constancy of its height. The *fifth day* was scarcely less febrile, the mean temperature being only 0.2° Fahr. below the *fourth day*.

Towards the end of the *fifth day*, however, and during the commencement of the *sixth day* (from four to twelve P.M., 9th January), the thermometer decidedly fell slightly; then, during the early part of the *sixth day* of the disease (night of January 9), it made a great descent from 104.2° to 101° Fahr.; and after this time it never rose above 101.4°. During the whole of the after-part of the *sixth day* and the first part of the *seventh*, the temperature continued to fall, and at 11 A.M., January 11—the nineteenth hour of the

TABLE OF THE TEMPERATURE, PULSE, AND RESPIRATIONS.

FROM HOURLY OBSERVATIONS DURING THE DAY. MEAN NUMBER OF DAILY OBSERVATIONS,—14.

Date.	Day of Disease. (The disease commenced at 4 P.M.)	Temperature in Axilla. Hourly Observations during the Day.					Pulse. Hourly Observations during the Day.					Respirations hourly observed. Mean No.
		Mean.	Max.	Min.	Hours of Max.	Hours of Min.	Mean.	Max.	Min.	Hours of Max.	Hours of Min.	
Jan. 7.	Part of third and part of fourth day.	103.6° Fahr.	104.2°	102.6°	7 P.M.	9 A.M.	108	120	100	1 A.M.	2, 5, 10, and 11 P.M.	36
" 8.	Part of fourth and fifth days.	103.9°	104.2°	103.6°	6, 7, and 8 P.M.	11 A.M.	107	116	98	7 P.M.	11 A.M.	36
" 9.	Part of fifth and sixth days.	103.4°	104.2°	103.0°	1 P.M.	8 and 10 P.M.	106	112	100	4 and 6 P.M.	10 and 12 P.M.	48
" 10.	Part of sixth and seventh days.	100.6°	101.4°	99.4°	9 P.M. and 1 P.M.	11 P.M.	93	112	82	5 P.M.	10 P.M.	38
" 11.	Part of seventh and eighth days.	98.6°	99°	98°	3, 5, and 6 P.M.	11 A.M.	76	84	72	2 P.M.	4 P.M.	30
" 12.	Part of eighth and ninth days.	98.5°	98.8°	98.4°	1 P.M.	10 A.M., 2 and 4 P.M.	76	84	70	1 P.M.	9 A.M.	31



*seventh* day of the disease—it reached 98° Fahr. In thirty-six hours it had fallen from 103.2° to 98°, or no less than five degrees. Afterwards it oscillated for two days between 98° and 98.8°, but never rose above this latter point. The fever, in fact, had ended by crisis (a sudden and rapid defervescence), which is usually accompanied by a strong action of some eliminating organ, such as the skin, the kidneys, or the bowels; and in the case recorded by Dr. Parkes there were both profuse sweating and considerable urinary excretion; but whether more at the period of rapid fall than before is uncertain.

*The pulse* ranged from 120 to 100 during the first three days, and then fell to 90, 80, and 70. On comparing carefully the hourly variations of the pulse and temperature, it is quite clear that there is a connection between them, so that either simultaneously or often a little before or after, a fall or rise in the thermometer occurred, with a fall or rise in the number of the pulse. Not unfrequently the alteration in the pulse occurred before the change in the thermometer; and sometimes the pulse rose, though never greatly, when the temperature was falling. When the thermometer oscillated and finally fell, the pulse fell at the same time, and very uniformly. The respirations averaged 38 in the minute during the first four days, and 35 afterwards. They did not fall nearly so much as the *temperature and the pulse*; and were not nearly so good an indication of the course of the pyrexia. As the mean of the thermometer was not above 104°, as the mean of the pulse was not 120, and as the mean of the respiration was not 40, the case must be considered a slight one, according to the rule laid down by Wunderlich—namely, that the intensity of cases of pneumonia is to be judged of by the concurrence of these phenomena, so as to call all cases slight that fall *below*, and all cases severe that are *above* those averages in which *the temperature records 104°, the pulse more than 120, and the respirations more than 40 in the minute during the height of the disease*. It is only by a rigorous application of some such rule to the cases of pneumonia which are grouped together to furnish statistical data as a guide to treatment, that anything like trustworthy results can ever be obtained. Such results have not yet been so obtained; and it is scarcely to be expected that the task will be accomplished in our day.

2. *The Course of the Local Lung-Symptoms*.—When the patient was admitted, there was considerable crepitation, and some bronchial respiration in the lower lobe of the left lung. The hepatization increased, and was considerable on the fifth and sixth days, and its greatest amount was either at the period of the *defervescence* or was subsequent to it—the number of respirations being even greater after the temperature and pulse had commenced to fall than before, so that they appeared rather to run *parallel with the amount of the hepatization than with the general fever*. The sputa were most bloody during the *third and fourth days* of the disease—were less florid and more rusty on the *fifth and sixth*—after which they became less viscid and free from blood or hæmatine. The pain in the side disappeared on the *fourth or fifth day*. After the *seventh* or

*eighth day* the bronchial respiration began to lessen, and had ceased by about the twelfth to the sixteenth day. Harsh respiration, some *redux* crepitation, and a little sonorous *râle* were left for some days more.

3. *The Condition of other Organs.*—Having twice had rheumatic fever (eleven and two years before the pneumonia), the heart of the patient in one or other of the attacks had been slightly damaged, and he had slight aortic obstruction, with mitral regurgitant disease, but without any marked alteration in the size of the heart's cavities. There was supplementary breathing in the right lung, and about the angle of the right scapula was some slight suspicious bronchial respiration, as if some consolidation were there; but if so, it disappeared early.

The liver was not enlarged nor tender, but the conjunctivæ were very slightly yellow; and the yellowness disappeared at convalescence. There was thirst, loss of appetite, and dry, furred tongue.

The action of the eliminating organs was as follows: (a.) The skin acted profusely throughout, both before and at the period of defervescence; (b.) The bowels were rather confined, but the motions were said to be natural; (c.) The amount of excretion from the lungs was undetermined; (d.) The excretion from the kidneys was not determined till during the *fifth* and *sixth days* of the disease, the last day of the intense pyrexia. On this day there was a little albumen (one-sixth of the height of the urine in a test tube). There were no chlorides, and on the following day the albumen had disappeared. The other urinary ingredients were not determined; but there were copious deposits of lithates on the *fifth, sixth, seventh, eighth, and ninth* days of the disease, and probably afterwards, of which no note was made. On part of the *seventh and eighth day* eight grammes of chloride of sodium were taken by the mouth, and on the *eighth, ninth, and tenth* days, as on previous days, there was no chlorine in the urine. But on the *eleventh* day of the disease the chlorine began to reappear, was in some quantity on the *fourteenth* day, and on the *twenty-first and twenty-second* days of the disease it was so abundant as to yield seventeen to twenty-two grammes. The urates throughout were abundant, but their exact amount was not determined.

The records of this case of pneumonia have been thus fully given, in the hope that it may serve as a model for the investigation and recording of similar cases. Till such inquiries are fully carried out by many observers, working independently and uniformly, the statistics of pneumonia will get more confused than they now are, and will therefore become even of still less value.

It is now beginning to be appreciated by thoughtful physicians that the so-called physical signs are inferior in value to those afforded by accurate observation of the correlations of temperature, pulse, condition of the urine, and tendency to crisis; and these observations, so ably and clearly recorded by Dr. Parkes, are in unison with many similar records by Wunderlich, Metzger, Barthez, and others. They show that the crisis may occur on any day of the disease, but the usual dates are the *fourth, or sixth, or seventh* day.

The following is a tabular statement of those results:

TABLE.

Date.	Day of Disease.	Condition of Patient.	Amount of Urine in cub. cents. in twenty-four-hours (b.)	Urea in grammes in twenty-four hours. (a.)	Chloride of Sodium in twenty-four hours in grammes. (a.)	One kilogramme of body-weight excreted in twenty-four hours of urea, in grammes.
Jan. 10.	Part of sixth and seventh days.	First day of defervescence, temp. 100.6° Fabr.	980	85.56 (= 1321 grains).	—	1.245
" 11.	Part of seventh and eighth.	Temp. 98.6° Fabr. Complete defervescence.	—	—	—	—
" 12.	Part of eighth and ninth.	Complete defervescence. Absorption of exudation.	865	87.38 (b.) (= 1349 grains).	—	1.273
" 13.	Part of ninth and tenth.	do.	865	87.38 (= 1349 grains).	—	1.273
" 14.	Part of tenth and eleventh.	do.	—	—	Some amount unknown.	—
" 17.	Part of thirteenth and fourteenth.	Complete convalescence. Exudation gone.	1300	35.1 (= 542 grains).	0.040 grms. in 10 c. c. 5.2 (= 80 grains).	0.551
" 24.	Part of twentieth and twenty first.	do.	1267	30.2 (= 466 grains).	17.15 (= 265 grains).	0.474
" 25.	Part of twenty-first and twenty-second.	do.	1760	44.0 (= 679 grains).	21.95 (= 339 grains).	0.691
" 27.	Part of twenty-third and twenty-fourth.	do.	1910	40.1 (= 619 grains).	14.32 (= 221 grains).	0.630
Mean excretion of urea by one kilogramme of body-weight on the seventh, ninth, and tenth days during resolution,						1.263
Ditto ditto during the fourteenth, twenty-first, twenty-second, and twenty-fourth days' complete convalescence, . . . . .						0.586
Mean physiological excretion in men aged twenty-two, . . . . .						0.500

(a.) The urea and chloride of sodium were determined according to the method of Liebig, by Dr. Parkes' excellent Clinical Clerk, Mr. Smith. The chloride of sodium was not got rid of before testing for urea, but the usual correction was made. The amounts are given both in grammes and grains.  
(b.) The urine of two days added together, and the total halved.

*The Urine in Pneumonia*, [—is generally, of a deep yellow-red hue, flame-color.] (1.) The amount of *water* is lessened by a third or a half (600 to 800 c. c.). This is most marked during the early days, and is no doubt due to retention of water in the febrile body. During the stage of resolution the amount of water increases. (2.) *The urea* is greatly increased during the height of the disease—an increase not due to any other cause than the metamorphosis of tissue during the pyrexia; and in equal periods of time it is more abundant during the day than during the night; and is in larger quantity *before* than during resolution. Dr. Parkes, however, has found it to be very great during resolution; and the general fact must be admitted that *the greatest amount of urea coincides with the highest temperature, and that it arises from general increased metamorphosis.* (3.) *The specific gravity* is high (1025–1035) partly from deficiency of water, and partly from excess of urea; and as the chloride of sodium is absent, the specific gravity measures pretty accurately the amount of urea. (4.) *The uric acid* is increased, and

its free excretion is a favorable sign. (5.) *The urine-pigment* is increased two or threefold, or altered; and it tints the urates, when they fall, brown, red, or carmine. (6.) *The chloride of sodium* is diminished, or is entirely absent during the existence of the fever and the occurrence of the lung-lesion—i. e., during the early period, or at the commencement of hepatization. Even when given by the mouth at this period, it will not pass off as in health, but is retained, and will not appear in the urine (in Dr. Parkes' case, for more than forty-eight hours). It reappears during, or rather after resolution, but it does not always reappear directly resolution commences. It may still be retained for some days—as many as *eight* or *ten*—after which it is poured out in such quantities as sometimes to raise the specific gravity of the urine. This especially occurs in cases where there is little expectoration and no purging, although the water of the urine is increasing and the urea is diminishing. *Fifteen* to *twenty-five grammes* may be thus passed in the urine during twenty-four hours; and the period of its increase is some time after the excretion of urea has reached its acme, and is declining. There is thus ample evidence that the chlorine has been retained, and not excreted in excess through the skin; and even when *hydrochloric acid* is given to the patient, no chlorine appears in the urine; and Beale's observations prove that the lung exudation is very rich in chlorides, which are often largely excreted in the sputa. The retention of the chlorides thus seems to be intimately connected with the development of the lung-lesion or exudation; and their excretion is increased during the absorption and disappearance of the exudation.

[Redtenbacher, Wachsmuth, Neubauer, J. H. Bennett, and others, have within a few years called attention to the diminution or absence of the chlorides in the urine during the course of acute pneumonia, and their reappearance at the period of defervescence, and claimed that both the treatment and prognosis might be influenced by the amount present. Observation shows, however, that the fact is not constant, and is of little clinical significance; and that in other diseases the same variation in the amount of the alkaline chlorides is noticed. The presence or absence of sodium in the urine is probably more intimately related to the amount and kind of food taken, than to the several stages of the disorder. In three cases of pneumonia in old persons, two of which ended fatally, Bergeron found the chlorides present in the urine. In a case of pleuropneumonia in an adult, which had lasted already sixteen days, and where all signs of lung-solidification had ceased, but some pleural effusion remained, the same careful observer could obtain no chloride of sodium in the urine; on the twenty-first day there was a slight precipitate; on the twenty-third and twenty-fourth days there was no trace of it; on the twenty-fifth and following days, it reappeared and was abundant.\* In

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\* [On an analysis of incinerated portions of lung in the second and third stages of pneumonia, Dr. Bergeron obtained in 810 grains (20 grammes) of lung-tissue by weight a mean of about  $\frac{1}{4}$ th of a grain (4 centigrammes) of chloride of sodium. In a pneumonic lung weighing about 87 oz. (1150 grammes), about 22½ oz. (700 grammes) more than the sound lung of the opposite side; the total quantity of chloride of sodium abstracted from the blood was about 21 gr. (1 gr. 40).]

two cases of intermittent fever, an analysis of the urine by J. Moss gave the following amounts of chloride of sodium :

1. { Paroxysm, . . . 5.5 grammes.\*  
Apyrexia, . . . 6.4 " "
2. { Paroxysm, . . . 0.8 grammes.  
Apyrexia, . . . 7.6 " "

In two successive paroxysms of tertian ague, Ranke found the amount of chloride of sodium in the urine to be :

1. { Apyrexia, . . . 0.44 grammes.  
Paroxysm, . . . 4.18 " "
2. { Apyrexia, . . . 1.29 grammes.  
Paroxysm, . . . 8.10 " "

In a case of acute pneumonia, Wachsmuth analyzed the urine and ascertained the daily amount of urea and chloride of sodium present. Convalescence began on the twelfth day; up to that time the diet was rigid:

Day.	Urea.	Chloride of Sodium.
2 . . .	13.89†	5.88 grammes.
3 . . .	86.28	8.79 "
4 . . .	22.08	4.18 "
5 . . .	81.27	0.47 "
6 . . .	17.86	0.62 "
7 . . .	82.87	0.88 "
8 . . .	23.72	0.88 "
9 . . .	20.15	} traces.
10 . . .	15.67	
11 . . .	18.95	
12-14 (Convalescence.)	12.51	2.69 "
(Food.)		
15-16 . . .	25.70	13.73 "[

(7.) *The sulphuric acid* is also increased by about a third. (8.) *The phosphoric acid* and the free acidity are lessened.

Abnormal products abound in the urine. They are chiefly as follows: (1.) *Bile-acids*; (2.) Bile-pigment, with or without jaundice; (3.) Albumen is present in a large proportion of cases. The period of its occurrence is variable; but both Weller and Parkes agree that it is most common at the commencement of and before resolution—*i. e.*, at the height of the disease. It has been supposed that some of the absorbed exudation is got rid of in this way (J. W. BEGBIE); but in the cases observed by Dr. Parkes, the albumen in the urine always appeared *before* resolution, when the lungs were yet commencing to be consolidated; and in one case there were, at intervals of from *six* to *eight* weeks, three recurrent attacks of acute pneumonic consolidation, affecting different parts of the two lungs, and at the height of each attack albuminuria occurred; while in the intervals, and subsequently, the urine was quite free. Dr. Parkes, therefore, does not ascribe the appearance of albumen in the urine in cases of pneumonia to the absorbed exudation passing out by the urine in this form, but to the implication of the kidneys in the general congestion and exudation, the most marked local seat of which was in the lungs.

\* [Average amount excreted in twenty-four hours in health, about 11.5 grammes =177 grains.]  
† [Mean daily amount excreted in health, by adult males between 20 and 40 years, about 88.18 grammes,=512.4 grains.]



Of five cases in which albuminuria occurred during the height of the disease, no fewer than three died; while among seven *non-albuminous* cases observed by Dr. Parkes, only one died. While the mortality of all the cases recorded by him was *four in thirteen*, or 30.7 *per cent.*, that of the cases with albuminous urine was 50 *per cent.*, and with non-albuminous urine it was 14 *per cent.*; “*therefore albuminuria occurring before resolution appears to be an unfavorable sign*; and the very frequent occurrence of albuminuria,” continues Dr. Parkes, “in sthenic pneumonia is one of the most interesting facts in its clinical history. In no disease seen in this country, except, perhaps, in *maculated typhus*, is albuminuria so common as in *pneumonia*. *Bronchitis* of the large tubes, chronic phthisis, and emphysema present a most marked contrast to pneumonia in this respect.” It is not invariably connected with *extent* of local disease: cases of enormous consolidation, and with great dyspnoea, have not necessarily albuminous urine. [Bergeron found albuminuria very rare in pneumonia of old persons, even when of the severest kind. In the two cases he met with it, there was nephritis.]

In some cases, instead of being increased, the urinary solids are lessened, even in intensely febrile cases, with a heightened temperature of *five or six degrees*. Such is a result of retention, and not of diminished excretion (see vol. i, p. 75, *et seq.*); and in such cases some one or other of the following events happen: (1.) At a later period of the disease a large amount of some ingredient may be poured out by the urine, although the febrile symptoms have almost or entirely gone—*e. g.*, uric acid—the crisis by the urine of the older physicians; (2.) Towards convalescence spontaneous diarrhoea may come on; (3.) The recoveries are not so rapid as in cases in which the urinary excretion is large. The amount of the urine, indeed, is a good guide for prognosis. As in *typhoid fever* and other acute affections, it is probable that the products of metamorphosis are retained, and poison the blood.

*During convalescence* the urine generally augments considerably in amount, and (cases of previous retention of excreta being excluded) the *urea*, the *uric* and *sulphuric* acids diminish for a short period below their healthy range; the *chloride of sodium* increases, and an extraordinary amount is sometimes passed; but if the quantity got rid of by *sputa* or by *diarrhoea* has been large, the amount of *chloride of sodium* excreted by the urine will be proportionally less (Parkes *On the Urine*, pp. 270–279).

Decrease of temperature, as a rule, precedes the falling of the pulse, and, on an average, the *fourth day* is the day of *maximum* elevation in uncomplicated cases; the decline, beginning on the following day, becoming rapid, and attended with various well-marked critical phenomena on the *seventh day*; while the *minimum* is reached about the *ninth day*. In typical cases, throughout the attack, the temperature rises all day towards evening, and falls during the night towards the morning.

If a fresh lung-lesion or exudation commences, a reaccession of temperature will indicate its occurrence; but in all pure and simple cases the defervescence is a precipitate one, the temperature falling,

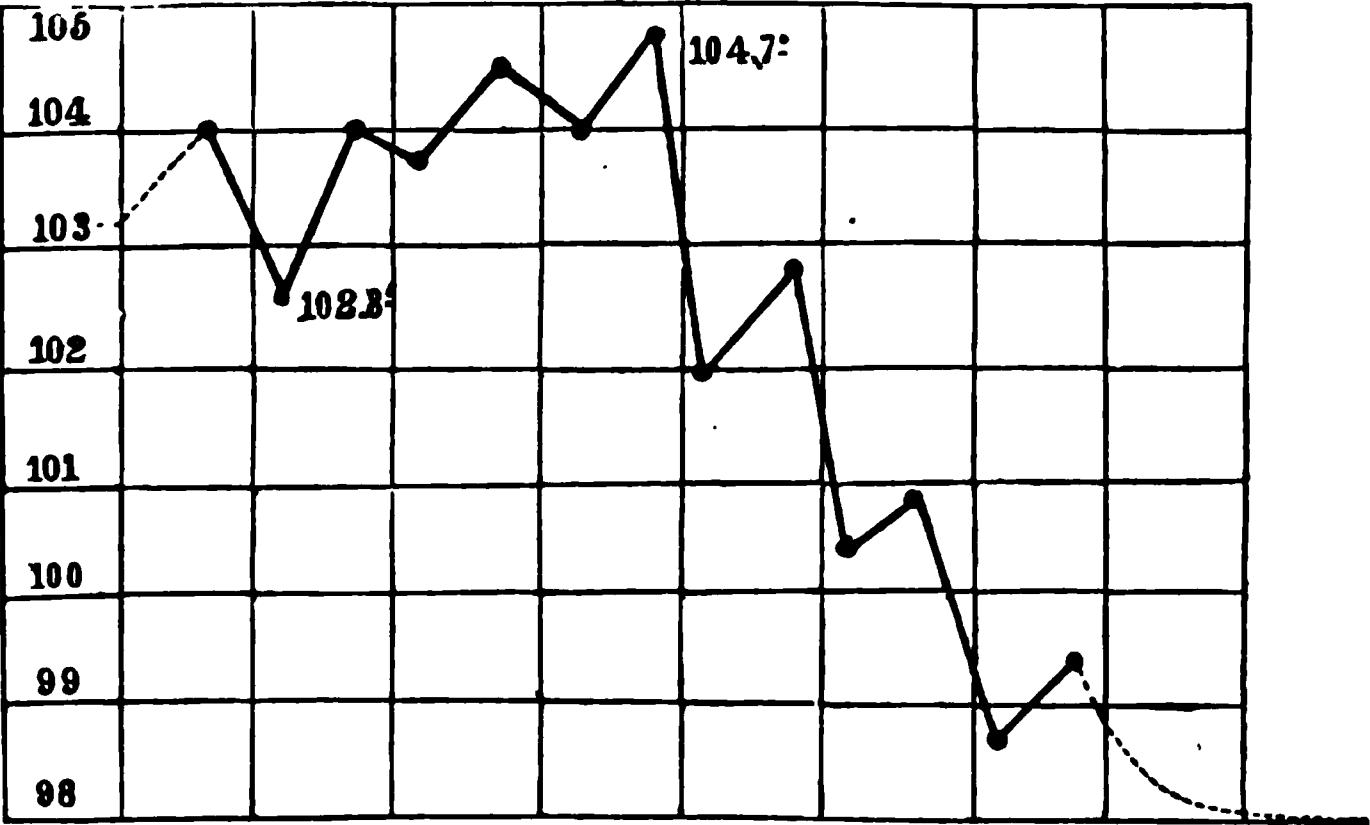
in the course of from *twelve to thirty-six hours*, as much as  $7^{\circ}$  Fahrenheit, or more degrees. In the majority of cases the decrease during the first *night* of defervescence is very considerable, small during the succeeding day, but again considerable during the following night (WUNDERLICH). If a diagram of the temperature in a case of pneumonia be projected upon paper by the student, as was done with several of the febrile diseases in vol. i (some of which it resembles, such as *measles*), he may perhaps better appreciate the statements that have been made regarding temperature in this disease.

[The thermoscopic cycle in a case of pneumonia with natural evolution, has three periods: (1.) Ascension. (2.) Stasis, or development. (3.) Decline. The first period is distinguished by the rapid and nearly continuous rise of the body-heat; there is, during this period, as in all febrile disorders, a slight fall in the morning temperature; but, while the thermometer marks some tenths of a degree less than it did on the previous evening, it shows a considerable rise from the temperature of the previous morning. The line of ascension rarely goes beyond the evening of the second day, for, generally at that time, and forty-eight hours after the initial chill, the maximum temperature is reached. During the period of development, it stays about the same, offering, however, some interesting oscillations. The type of the fever in pneumonia is not continuous. Thomas (of Leipsic) who has particularly investigated this point in the clinical history of the disorder, makes out four forms of the accompanying fever: (1.) Sub-continuous, the daily thermometric variations being expressed by two- or three-tenths of a degree; this is the least frequent type, and rarely, when present, lasts longer than one or two days. (2.) Sub-remitting, in which a fall of from one-half to one degree marks the remissions; this is the most common variety. (3.) Remitting type, where the oscillations of the temperature exceed  $1.5^{\circ}$ ; and (4), in rare cases, where the type of the fever is intermittent.

A few trustworthy observers have been enabled to note the body-heat almost at the outset of the seizure. In a patient, where it was recorded four hours after the initial chill, the thermometer in the axilla marked  $102.5^{\circ}$  Fahrenheit, and, eight hours later, that is, twelve hours after invasion, it rose to  $104^{\circ}$  (ZIEMSEN). Thomas (of Leipsic) found, nine hours after the onset, the temperature  $104.8^{\circ}$  Fahrenheit; in another case, twenty-three hours after the chill,  $105^{\circ}$ ; in a third, on the morning of the second day,  $106^{\circ}$ ; and in eight other cases, where the body-heat was taken on the evening of the second day, it ranged from  $102.2^{\circ}$  to  $104.4^{\circ}$  Fahrenheit. In a case examined by the late Dr. Waters on the first day, the thermometer rose to  $102.8^{\circ}$ . In double pneumonia, the thermometric curve does not rise higher than when one lung only is affected.

RANGE OF TEMPERATURE IN A CASE OF PNEUMONIA OCCURRING IN A MAN THIRTY-EIGHT YEARS OF AGE, DATING FROM THE FIRST EVENING OF THE ATTACK. THE RECORD INDICATES MORNING (M.) AND EVENING (E) OBSERVATIONS (Wunderlich).

Degs. 1st day 2d day 3d day 4th day 5th day 6th day 7th day 8th day  
Fahr. M. E. M. E. M. E. M. E. M. E. M. E. M. E.



LINE OF NORMAL TEMPERATURE 98° FAHR.

[Table showing Correlation of Temperature and Pulse in Pneumonia (WATERS).

Day.	Temp.	Pulse.
1st, . .	102.8°	123
2d, . .	102.3°	120
3d, . .	103.6°	122
4th, . .	104°	126
5th, . .	103°	122
6th, . .	102.8°	122
7th, . .	100	114
8th, . .	99	94
9th, . .	N.	78

Bergeron has carefully observed the body temperature in the pneumonia of the aged.\* He found the thermometer to rise rapidly and abruptly to 102°, 103°, and 104° Fahrenheit, and then fall to 102.10°, 102°, 101°, and even to 99°; it then rises from one-half to one degree, where it remains for some days, until the period of defervescence, when it suddenly falls several degrees. In cases terminating fatally, the thermometer oscillates between 104° and 102°, never falling below this, and in some cases immediately before death, reaches 105°. The difference between the morning and evening temperature was noted; generally, and it may be regarded as a favorable prognostic sign, the evening temperature is higher by from two-fifths to three-fifths of a degree, than that of the morning, and may exceed it one degree. In very severe cases, the temperature is occasionally the same, morning and evening, or it may be higher in the morning by a degree or more. The following table from Bergeron gives the temperature in a fatal case of pneumonia in an aged female.

\* [*Recherches sur la Pneumonie des Vieillards (Pneumonie Lobaire Aigue)*, par le Dr. Georges Bergeron, Paris, 1866.]

DAY.	Morning.	Evening.
February 25, .		104.2
“ 26, .	104.2	104.6
“ 27, .	103	104.8
“ 28, .	102.7	104.1
“ 29, .	104.8	Died.

In studying the thermometric phenomena of disease, the surest results will be obtained by taking the body-temperature in the rectum, when practicable, instead of in the axilla. There is constantly between the temperature of the rectum and that of the axilla, at the same time and in the same person, a difference of never less than one degree, and often of two or three degrees, and sometimes more. The mercury attains its status with the thermometer in the rectum in two or three minutes, whilst in the axilla it often continues to oscillate during eight or ten minutes. When taken in the axilla, it should always remain twenty minutes in situ. The same instrument should be used throughout in the observation of a case.

Reliable thermometers, of proper form and gradation, are manufactured by Mr. J. Ronchetti, 92 Fulton Street, New York. Messrs. Otto & Rynders, 64 Chatham Street, New York, import and have for sale Dr. Aitken's thermometers, made by Casella, of London.]

The natural history of pneumonia would be incomplete without some attempt to expound the causes of this tendency to a spontaneous favorable termination of uncomplicated cases, and their determinate duration. This cannot be better expressed than in the words of Dr. Parkes. First of all, he considers the following question:

“What is the exact connection between the lung-symptoms and the general pyrexia? The course of the two is certainly not exactly parallel. The complete consolidation seems to be posterior, in point of time, to the height of the pyrexia. It has even been supposed by Wunderlich that the exudation into the lungs coincides with the end of the pyrexia—that is to say, that the defervescence commences when the lungs become completely hepatized. I have not been able to satisfy myself rigorously on this point. If it could be satisfactorily made out, it would certainly imply that the exudation into the air-cells relieved or cured the fever—in other words, that the lung-disease is not a primary, but a secondary condition, and that it succeeds to and brings to an end, by purifying the blood, a condition of general pyrexia, arising from blood disease. Without believing that this relation is quite determined (if it were determined, the case would be settled), there is no doubt that the fever ends spontaneously, or very greatly lessens at the time when the inflammation of the lung is very great.

“There are at present two views more or less clearly defined which aim at explaining these phenomena, and which may be thus expressed—

“1. According to the first of these, there is a blood disease of some sort, of a nature not thoroughly known, but which consists, in part, in an augmentation of the fibrine, as in acute rheumatism. Increasing up to a certain point, and giving rise to the slight malaise which precedes all cases of pneumonia, it is at last brought to a head by some exposure,

by a dietetic error, or, by reaching a point at which the functions of the blood are seriously interfered with. Then ensues high general fever, from implication of the nervous system, and at the same time some organ or other is, on account of special affinity for the morbid blood, or from previous damage to its structure, specially irritated. In pneumonia the lung is the seat of election, and there is rapid hyperæmia and transudation of fluid into the air-cells. By this transudation the morbid blood is purified. The process is analogous to that of gout, in which a diseased blood gives rise to a local disease by the deposition of urate of soda in and about joints. When the localization and consequent purification is finished, then the fever ends. There remains the lung-exudation, which gradually softens down, is partly expectorated, partly absorbed; and, in the process of absorption, it may produce again secondary contamination of the blood, and certain affections of other organs, which constitute those secondary affections which sometimes complicate the after-course of pneumonia.

“The weak points in this hypothesis are the want of definite indications of the blood disease, and of its mode of production. There is some evidence on these points, but it is certainly not very great.

“Its strong points are the explanation it gives of the previous malaise; of the sudden outburst of fever, when the diseased blood implicates at last the nervous system; of the singular and rapid termination of the pyrexia at a time when the lung-lesion is yet intense; and of the enormous elimination of urea during the very first days before the lung-exudation has softened down.

“2. The second hypothesis is the exact contrary of the former. The lung-affection is supposed to be the primary lesion: it is a local inflammation produced by the (still obscure) causes of local inflammation, running the ordinary course of such inflammations, and giving rise to violent symptomatic pyrexia. The undoubted increase of fibrine in the blood is supposed to be not primary but secondary, to be caused by and to augment with the inflammation, and to be at its height with it. Virchow has stated that this fibrine is nothing more than the albuminous substance absorbed by the lymphatics of the inflamed and hepatized lung, and poured into the blood. The fever is believed to be entirely symptomatic of the local disease, and to be commensurate with its intensity and extent. The arguments for this view seem to be, that the lung-symptoms are remarkably early in manifestation, though they may not be very intense. Pain in the side and cough are very soon present, and sometimes occur even before the shivering and headache. The pyrexia, although great in the early days, is perhaps not greater than might have been produced by the condition of the lung; and as to the termination of the fever, this may be supposed to occur because the really true febrile stage of pneumonia is not the period of complete exudation, but the preceding period of intense hyperæmia. To say that the pyrexia is gone, when the lung-lesion is yet most intense, may be an incorrect expression of the fact; the consolidation may possibly, indeed, be most intense, but this may be merely the natural termination of that enormous hyperæmia and blockage of vessels from local changes of nutrition, which is in reality the essential disease.

“The difference between these two hypotheses would be this: the fever ends spontaneously, first, because the blood is purified, or, secondly, because the local disease ends spontaneously—*i. e.*, the active febrile-making local disease. This last assumption, however, is decidedly a very bold and hazardous one.

“Between these two views it is not very easy, nor perhaps is it desir-



able, yet to choose, for the blood has not yet been sufficiently examined. The only blood disease which has yet been indicated by the supporters of the first view, as anterior to pneumonia, is hyperinosis (excess of fibrine); and as hyperinosis occurs in acute rheumatism without pneumonia, it is evident that there must be some other cause, either in the blood or in the local structure of the lung, which locates the disease in that part.

“That hyperinosis is really anterior in pneumonia as in rheumatism, must, in spite of the opinion of Virchow, be considered likely, from the experiments, among others, of Professor Naumann, of Bonn: that it is not the only condition in either of these cases will be generally admitted. But what other blood affection is there? None has yet been indicated, to my knowledge, in the acute sthenic pneumonia of young persons without gouty or renal disease. But there is one point on which I have been trying to collect evidence for some years, but at present without sufficient success. It is well known how frequently the liver is affected in pneumonia, so that some amount of jaundice is not at all uncommon, and sometimes bile-pigment appears in the pneumonic sputa. I have also found in some cases evidence of liver affection for some time before the lung-disease, especially the so-called torpor with deficient biliary flow.

“Is there, then, any condition of the liver which adds something to the blood which ought not to be there? Taurin has been found in the healthy tissue of the lung; but in the hepatized lung it seems, from Verdel's observations on his pneumic acid (taurin), to be in excess. Is it some compound of this sort which, in combination with the hyperinosis, determines the localization of the blood disease, or produces by its irritation the inflammation of the lung? I know no facts whatever which can lead to a decision; but it is to be hoped that some competent person will soon undertake a more complete analysis of blood in the very first days of pneumonia than has yet been made.

“But, whatever be the facts as to the order of things in pneumonia, whether the lung affection is a mere localization of the blood disease, or whether the undoubted blood disease of the developed stage is merely produced by absorption from the inflamed lung, it is certain that the usual course of pneumonia is such as we witnessed in this untreated case, viz.,—(1.) There is an early period of intense fever, ceasing, if no complication be present, of itself, at a tolerably determinate time; (2.) There is a later period of lung hepatization, which softens down during a period of moderate fever, and is expectorated or absorbed. There are therefore two periods in pneumonia, and both have their dangers. The intense early fever may kill by its intensity; the exudation may kill, subsequently, by apnœa, or may contaminate the blood during softening to such an extent as to lead to renewal or increase of the fever and inflammation of other parts, or to coagulation of the blood in the heart or great vessels. Each period has its own dangers, and must have its own treatment.”

**Morbid Anatomy.**—Following Andral and Laennec, all subsequent writers on pneumonia are persuaded that a knowledge of the anatomical characters of the lung in this disease is essential to a perfect appreciation of the symptoms, physical signs, prognosis, and treatment of the disease. Both in the acute and chronic forms of diffuse inflammation of the substance of the lungs, the vesicular pulmonary tissue is more loaded with dark venous blood than usual, and its texture is more easily broken down than in health; air, however, still penetrates the air-cells, and consequently the lung still crepitates, swims in water, and, if washed, the color is nearly restored,

but it is doughy, pits on pressure, and is red or livid, and heavier than the normal lung.

This is the *first stage of pneumonia*—namely, that of *simple engorgement*. This state of inflammation may terminate by resolution, or it may pass to more complicated and dangerous lesions. Dr. Stokes is of opinion, however, that a *stage of irritation* has previously existed, so that the *first stage* described by Laennec would be the *second* described by Dr. Stokes, who has repeatedly seen a condition of the lung, regarded by him as the first or earliest stage of pneumonia, in which the lung-tissue is drier than usual, and of a bright vermilion color from intense arterial injection; but there is no engorgement (*On Diseases of the Chest*, p. 310). Skoda and Fuller are opposed to this conjectural view.

When effusion of serum succeeds, the lung is in the same gorged stage, but in addition it is loaded with watery fluid, so that on cutting into it the serous fluid mixed with blood streams from it as from a sponge. When its action is listened to during life, a lung in this condition no longer crepitates, and its bulk is enlarged; for it may be seen after death to have taken the impression of the ribs, and it does not collapse when the chest is opened. The lung is now technically said to be in a state of *red hepatization*, or, as Andral has termed it, *red softening*; for, although firm, its texture has lost its natural toughness, cohesion, and resistant power.

This is the so-called *second stage of pneumonia*. The diseased part readily breaks up by a thrust of the finger into its substance. This state has many degrees, the air-cells being either wholly or partially impermeable. In some instances the productive effects of inflammation (commonly called exudation) are very large in quantity, mixed with blood, and the more fluid portion can be readily separated, or, as it were, pressed out of the lung. In the other extreme of this form of inflammation the exudation forms an integral part of the lung, which then becomes so solid that, if cut, it represents with much accuracy a portion of the liver or spleen. In this state it contains at the diseased part little or no air, does not crepitate nor float in water; it cannot be injected, is of a deep venous color, while its texture is easily broken down and penetrated by the finger. The lung is still enlarged or swollen, and does not collapse when the chest is opened. It varies in color from a reddish brown or deep dull red to a violet hue, and is generally darker in the aged than in the adult.

[In 18 pneumonic lungs examined by Dr. R. Cresson Stiles there was an average excess of 2 lbs. over the weight of the opposite sound lung; 4 weighed between 35 oz. and 2 lbs.; 4 between 2 and 3 lbs.; 4 between 3 and 4 lbs.; 5 between 4 and 5 lbs.; and 1 weighed 5 lbs. and 11 oz., the weight of the opposite sound lung being 1 lb. 8 oz.—the weight of the exudation was 1 lb. 8 oz. (*Medical Record*, Oct. 1866). Grisolle has seen a hepatized lung weigh 5½ lbs. The excess is generally in proportion to the extent of pulmonary tissue solidified.

The following table, compiled by Dr. Ira Russel, gives the weight of the lungs in 50 fatal cases of pneumonia, and is taken from his valuable paper in the *United States Sanitary Commission Medical Memoirs*. Dr. Russell has found the weight of the healthy lung in the negro to be on an average four ounces less than in the white.

TABLE

SHOWING THE ORDER IN WHICH THE LUNGS AND SEVERAL LOBES WERE ATTACKED, THE STAGE OF THE DISEASE IN EACH LOBE AT THE TIME OF DEATH, AND THE WEIGHT OF EACH LUNG IN FIFTY CASES AMONG COLORED TROOPS AT WILSON HOSPITAL, TENNESSEE.\*

Number of Cases.	Order of attack of Right and Left Lung.		Order of attack of Lobes of Right Lung.			Stage of disease of Lobes of Right Lung.			Order of attack of Lobes of Left Lung.		Stage of disease of Lobes of Left Lung.		Weight of Right Lung.	Weight of Left Lung.	REMARKS.
	R. Lung.	L. Lung.	Superior Lobe.	Middle Lobe.	Inferior Lobe.	Superior Lobe.	Middle Lobe.	Inferior Lobe.	Superior Lobe.	Inferior Lobe.	Superior Lobe.	Inferior Lobe.			
1	1	2	2	N	1	R	N	P	N	1	N	R	26	20	Bronchitis.
2	1	1	1	N	1	R	N	P	N	1	N	R	24	26	
3	1	1	1	1	1	R	N	P	N	1	N	R	23	24	
4	1	N	1	1	1	R	R	C	N	N	N	R	16½	8½	Typhoid Fever.
5	2	1	1	1	1	R	C	R	1	2	R	C	21	24	
6	1	1	1	1	1	R	C	R	1	1	N	R	30	11	
7	1	1	1	1	1	R	C	R	1	1	N	R	42	22	Measles sequelæ.
8	1	2	1	1	1	R	C	R	1	1	N	R	34	37	
9	1	N	1	1	1	R	C	R	1	1	N	R	55	31	
10	1	2	1	1	1	R	C	R	1	1	N	R	50	23	Left Lung collapsed with 24 oz. effusion.
11	1	2	1	1	1	R	C	R	1	1	N	R	36	27	
12	1	2	1	1	1	R	C	R	1	1	N	R	38½	21	
13	1	2	1	1	1	R	C	R	1	1	N	R	20	21	Pleuritis Left Side.
14	2	1	1	1	1	R	C	R	1	1	N	R	50	24	
15	1	2	1	1	1	R	C	R	1	1	N	R	43	39	
16	1	2	1	1	1	R	C	R	1	1	N	R	22½	30	Effusion R Side 12 oz.
17	N	1	1	1	1	R	C	R	1	1	N	R	14½	42	
18	2	1	1	1	1	R	C	R	1	1	N	R	30	20	
19	1	2	1	1	1	R	C	R	1	1	N	R	25	24	Pleuritis Left Side.
20	2	1	1	1	1	R	C	R	1	1	N	R	23	44	
21	2	1	1	1	1	R	C	R	1	1	N	R	21	25	
22	1	N	1	1	1	R	C	R	1	1	N	R	39	16	Bronchitis of Left L.
23	1	2	1	1	1	R	C	R	1	1	N	R	44	23	
24	1	2	1	1	1	R	C	R	1	1	N	R	26	15	
25	1	N	1	1	1	R	C	R	1	1	N	R	30	11	Bronchitis of Left Lung.
26	1	2	1	1	1	R	C	R	1	1	N	R	27	20	
27	2	1	1	1	1	R	C	R	1	1	N	R	28	23	
28	2	1	1	1	1	R	C	R	1	2	N	R	21	49	
29	1	N	1	1	1	R	C	R	1	2	N	R	24	10	
30	1	N	1	1	1	R	C	R	1	2	N	R	49	12	
31	2	1	1	1	1	R	C	R	1	2	N	R	19	31	
32	2	1	1	1	1	R	C	R	1	1	N	R	41	18	
33	1	N	1	1	1	R	C	R	1	N	N	C	29	9	
34	1	2	1	1	1	R	C	R	1	2	N	R	35	16	
35	2	1	1	1	1	R	C	R	1	2	N	R	19	30	
36	2	1	1	1	1	R	C	R	1	2	N	R	38	40	
37	1	2	1	1	1	R	C	R	1	1	N	R	35	14	
38	1	2	1	1	1	R	C	R	1	1	N	R	43	26	
39	2	1	1	1	1	R	C	R	1	1	N	R	24	24	
40	2	1	1	1	1	R	C	R	1	2	N	R	15	28	
41	1	2	1	1	1	R	C	R	1	1	N	R	32	24½	
42	1	2	1	1	1	R	C	R	1	1	N	R	30	27	
43	1	2	1	1	1	R	C	R	1	1	N	R	43	24	
44	2	1	1	1	1	R	C	R	1	2	N	R	17	46½	
45	1	2	1	1	1	R	C	R	1	1	N	R	27	16½	
46	2	1	1	1	1	R	C	R	1	1	N	R	19½	34	
47	1	2	1	1	1	R	C	R	1	1	N	R	34½	26	
48	2	1	1	1	1	R	C	R	1	1	N	R	23	28	
49	1	2	1	1	1	R	C	R	1	1	N	R	29	21	
50	1	2	1	1	1	R	C	R	1	1	N	R	32	31	

From the above Table it appears that the right lung was attacked first in 32 cases, the left lung in 18; superior lobe, right lung, 21; middle lobe, right lung, 14; inferior lobe, right lung, 37, three lobes simultaneously, 11; superior lobe, left lung, 24; inferior lobe, left lung, 32; both lobes simultaneously, 14. Superior lobe, right lung, in stage C, 15; R, 11; G, 9; P, 6; N, 9. Middle lobe, right lung, in stage C, 12; R, 9; G, 6; P, 4; N, 10. Inferior lobe, right lung, in stage C, 14; R, 15; G, 9; P, 9; N, 3. Superior lobe, left lung, in stage C, 19; R, 9; G, 4; P, 3; N, 14. Inferior lobe, left lung, in stage C, 13; R, 15; G, 8; P, 4; N, 9.]

\* Figures 1 and 2 show order of attack; letter C, congestion; R and G, red and gray hepatization; P, pus or purulent infiltration; N, normal.

Resolution of red hepatization is by fatty degeneration of the exudation granule corpuscles, giving to the affected portion of the lung the appearance of gray hepatization (N. GUILLOT, A. CLARK, LEBERT, VIRCHOW, BENNETT, STILES). Bergeron found in examining properly prepared sections of pneumonic lungs of old persons, three and a half times more fat in the so-called third stage than in equal weights of the same lung in the second stage.]

**Suppuration** may follow this form of pneumonia, and the pus effused may be either infiltrated or limited, as if contained in an abscess. Infiltration is by far the most common; and although this form of lesion may occur suddenly, as a result of serous infiltration in unhealthy persons, or during the progress of general diseases, yet in the belief of most pathologists, it more generally follows the *red hepatization*. In this latter case the pulmonary tissue, red, dense, compact, and impermeable to air, passes to a gray color, and hence it is termed *gray hepatization* or *gray softening*, and is regarded as the *third stage of the disease*, following Andral and Laennec. The structure in other respects of either form of hepatization appears to be the same; for if the lung be examined with a microscope the same granulations (only they are white or gray, instead of red) are found. There are instances, however, in which these granulations are wanting, and then a gray smooth surface only is observed. Hence the *granular* and *non-granular* forms of hepatization described by some authors.\* In aged persons the granulations of a pneumonic lung are much larger than in the adult, depending, no doubt, on the increased size of the air-cells in persons of advanced life (MM. HOURMANN, DECHAMBRE, and Dr. MACLACHLAN). Hepatization in old people, with granulation, is nearly four times more common than non-granular or planiform hepatization (MACLACHLAN, *l. c.*, p. 279).

In the *gray* as in the *red hepatization*, the pulmonary tissue is easily torn, and the quantity of pus infiltrated is sometimes so great that, on cutting into the lung, that fluid readily flows from it: at other times the pus will not flow on a simple incision, but exudes by compression. The lung is solid and impermeable to air. It sinks in water, and has ceased to crepitate. The interspersion of red and dark points gives a granite-like appearance to the section of a lung which is in a state of gray hepatization. The lung is then more friable than in red hepatization. The finger sinks into the gray portions of the lung upon the least pressure, and when the gray texture is squeezed in breaks down into a pulp.

**Abscess of the Lung.**—Although pus is more commonly diffused through the pulmonary parenchyma, yet sometimes it is collected into an abscess. In the infancy of pathology physicians regarded phlegmonous abscess of the lung as a common and ordinary occurrence, but it is extremely rare; and Laennec, when he published the first edition of his work, had only met with six cases, notwith-

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\* [According to Dr. Bergeron and other recent observers, the distinction made by Drs. Hourmann and Dechambre of granular and planiform hepatization of the pneumonic lungs of old persons can no longer be accepted.—EDITOR.]

standing his extensive research ; and in the practice of other physicians, phlegmonous abscess of the lung is equally uncommon.

[Louis has seen it once, Chomel, in twenty-five years, three times, Andral once, Trousseau twice, Flint, in 133 cases, four times (diagnosis verified post-mortally in two cases), and Grisolle, in ten years of special study of the disease, not once. Chambers, in a record of 600 cases at St. George's Hospital, found it in three. In 750 cases treated in the hospital at Vienna, 1847-50, pulmonary abscess was observed but in a single case (FLINT).]

Abscess of the lung (although termed phlegmonous, to distinguish it from tubercular abscess) generally exists without any great intensity of inflammation, or other considerable alteration of its tissue.

[There may be one or more, of the size of an egg, if single, or of a filbert, or less, if multiple ; central or peripheral ; the cavity of irregular form, sometimes subdivided by imperfect septa or bands, the ragged cells intercommunicating, and containing whitish inodorous pus, or reddish or chocolate-colored, with bits of disintegrated lung. The walls are of gray or red indurated lung-tissue, the orifices of the bronchial tubes being visible ; or they are lined by a thin, gray, false membrane, about half a line thick, which may be firm or soft. A pulmonary abscess increases from the centre to the periphery, or several abscesses may come together ; it opens either into the bronchia or pleural sac. Pulmonary abscess is more frequent after fifty, and especially after seventy years of age. In 67 cases of pneumonia in old persons, Moutard-Martin met with it in 3 ; and of 70 cases at Bicêtre, Mercier saw it in 5.]

In the experience of Dr. Maclachlan, however, the rarified condition of the lung in the aged "seems to favor the formation of small abscesses, which are occasionally seen interspersed through the red and gray consolidated tissues, as if certain air-cells had broken down and coalesced during the plastic or suppurative process, and formed so many sacs for the reception of the effused or secreted matter" (*l. c.*, p. 279).

**Gangrene of the Lung.**—Pneumonia may terminate by gangrene, which is also as rare a termination as by abscess.

[Out of 305 cases observed by Grisolle not one ended in gangrene ; of 133 cases analyzed by Flint, it happened in one.]

It occasionally arises from excess of inflammation, but more commonly the inflammation which precedes this state is of little intensity, so that it rather approaches to anthrax, and may be of pestilential origin. The gangrenous portion may or may not be circumscribed, and it is found in the different states of gangrenous eschar, of deliquescence, of sphacelus, and lastly, of simple excavation, the dead portion having been detached and expectorated. (See "Gangrene of the Lung.")

**Pyæmic Inflammation and Abscess.**—In some classes of cases, as in the continued fever of typhus and typhoid, pneumonia occurs as a



local lesion, and is sometimes named “ typhoid pneumonia ;” so also in another class of cases, characterized by *blood-poisoning* or by *embolism*, the lung disease is a secondary result, and is said to be due to pyæmia. While in yet another class of cases, as in those where some morbid condition of the constitution prevails—as in the alcoholism of drunkards—a primary pneumonia in them may give rise to blood-poisoning and the phenomena of pyæmia, by the sanious decay of the exudation and its reabsorption during resolution, or its passage to the left side of the heart.

Pneumonia may be either single or double—that is, it may attack one or both lungs at the same time. Of the part of the lung attacked, inflammation of the inferior lobe is most frequent ; next, of the superior lobe ; while rarely the whole lung is inflamed.

[Pneumonia of the right lung is about twice as frequent as that of the left, and this holds good at all ages. The inferior lobe is more often the site than the superior, in the proportion of four to three ; of 264 cases the inferior lobe was the first to suffer in 133, the upper lobe in 101, and the middle lobe in 30. Pneumonia of the apex is twice and half times more common in the right than the left lung (GRISOLLE). Of 109 cases analyzed by Flint, pneumonia was limited to the lower lobe of the right lung in 29 cases ; to lower lobe of left in 25 ; upper lobe of right in 8 ; upper lobe of left in 3 ; extended over whole of right lung in 27 ; over whole of left lung in 9 ; lower lobes of both lungs in 8. Double primary pneumonia is very rare ; it occurred in one-sixteenth of Grisolle's and Louis' cases. Of 128 pneumonias in Dr. Chomel's ward, in 1838–39, 8 only were double.]

Brônchitis may take place without pneumonia, but in many cases pneumonia follows as a consequence. Pneumonia also may take place without brônchitis, but in general brônchitis accompanies it. Pneumonia also may take place without pleuritis, but it generally happens that the pleura is more or less affected. It is so especially in the pneumonia of pyæmia. In old persons primary pneumonia is mostly lobar, and the disease should be particularly looked for at the inferior and posterior parts of the chest. It runs its course very rapidly to purulent infiltration, and the *prodromata* are not distinctly marked. Parotitis is a frequent complication, and of bad omen, and the pneumonia may be marked by cerebral phenomena.

**Hypostatic Pneumonia** is that form which is apt to occur in those who are obliged to lie on their back for a length of time, on account of a fracture, paralysis, or the like.

Much speculation has been entertained with respect to the more particular seat of pneumonia ; some contending that the inflammation affects the connective tissue of the lung, others the air-cells, and others both.

[On examining, under a dissecting microscope, a section of solidified lung, it is evident that the air-cells are the seat of the exudation. By hardening the preparation in alcohol, solid casts can be picked out.]

It is quite certain, however, that the minute bronchial tubes are

not affected in slight pneumonia; for in such cases their divided extremities stand out in the midst of the inflamed part like so many white points. When the lung is more acutely inflamed, the bronchial tubes are red, and evidently greatly inflamed. There can be no doubt, however, from what we now know of the nature of the inflammatory process, and also from direct observation upon the tissue of inflamed lungs, that the minute elements of the vesicular and connecting tissue composing the parenchyma of the lung are from the first directly altered in their vital properties. (See vol. i, p. 81, *et seq.*)

[In the initial stage the walls of the air-sacs are intensely vascular, and somewhat thickened; an examination under the microscope of a dried section of lung, shows the capillary plexus of the pulmonary arteries, which alone supply them, greatly enlarged, if not more numerous, as Grisolle believes, and their walls in close contact with scarcely an interspace. Besides this extreme congestion of the pulmonary plexus there is at the same time active cell proliferation within the air-sacs.]

**General Symptoms of Pneumonia**—Pneumonia is generally preceded by some antecedent fever, by shivering more or less violent, and often by bronchitis. In a few cases, however, consolidation may be complete ere any primary affection is observed. In others, failure of the appetite, general weakness, and wandering pains in the limbs and chest, precede any definite attack by several days.

[In about one-fourth of the cases of acute lobar pneumonia there are the usual precursory symptoms of threatening illness; and in about one-sixth—in old persons the proportion is greater—catarrhus symptoms, often slight, precede the attack several days. The invasion is almost—four times out of five—constantly marked by a chill, varying from mere creeping to severe rigor, which may last several hours, or through the night, and recur once or twice during the first and second days. In old persons the initial chill is nearly invariable. Sharp, lancinating pain at or near the nipple of the affected side, is one of the earliest and most common symptoms; in 301 cases it was absent in only 29, and in 182 cases it appeared within the first twelve hours in 161, and in 17 of the remaining 21 before the end of the first day, and in 4 from the second to the fourth day. In 173 cases its site was at the nipple in 89, near it in 31, in the axilla, 2, at the base of the chest in 39, infra-spinatal region in 5, supra-spinatal region in 1, and in distant points 6. In 30 cases of pneumonia in old persons, pain was an initial symptom in 22 (BERGERON), and in 50 cases recorded by Durand-Fardel it was present at the beginning in 31. Old persons not unfrequently complain of dull pain about the nipple several days before the acute pneumonic symptoms appear, and Moutard-Martin mentions two cases where pain existed in the right side for several days, while the physical signs showed pneumonia of the left lung, pneumonia of the right lung appearing on the third or fourth day.]

The disease being set up, the patient is restless and uneasy; his respiration difficult and hurried—from 30 to 50 in a minute—according to the amount of lung whose function is suspended; his cough

frequent, and his expectoration streaked with blood; but notwithstanding this symptom he seldom, unless the pleura is affected, suffers pain, which consequently increases the danger. The aged, however, seldom complain of difficulty of breathing during the progress of the disease, whatever may be the frequency of the respiration, so that it is incumbent to count the movements of the chest in old people, to avoid all sources of error (HOURMANN, DECHAMBRE, and MACLACHLAN). Obscure and latent forms of attack, in the experience of these eminent physicians, are frequently met with in aged subjects laboring under chronic disease of the brain, heart, or some other internal organ. The pulse is full and frequent—from 100 to 120; the countenance livid; the nostrils dilated; the tongue and lips more or less livid, the former of which is coated with a white or yellow mucus.

[A patch of flush on one or both cheeks (*rougeur des pommettes*), more or less deep and livid, is almost constant during the first and part of the second stage, and by some authorities is said to be more marked in pneumonia of the upper lobes. When limited to one cheek there is no constant unilateral correlation between the cheek-flush and the lung-lesion, as has been insisted upon, particularly by Dr. Gubler, the site of the former being often on the side opposite to that of the affected lung; and Dr. Jaccoud has published five cases, including his own, confirmatory of this statement made many years ago by the writer (*Practical Treatise on Diseases of the Respiratory Organs*, Philadelphia, 1845, p. 256).]

The patient inclines to lie on his back supported by pillows. If he recovers, these symptoms are gradually mitigated; but should his case tend to a fatal end, the tongue becomes brown and typhoid, his pulse more rapid, profuse sweats break out all over his body, and at length his mind wanders, and he dies by *coma* or *apnœa*. There are many instances, however, where the course is widely different, and in which the patient, though evidently distressed by impeded respiration, has yet moments of cheerfulness; gets up, and may even walk about. But he suddenly dies, seized with a severe paroxysm of dyspnœa or of coughing, followed by collapse of the remaining healthy part of the lung.

In typical cases of acute pneumonia certain groups of symptoms are more or less constantly combined,—namely accelerated respiration, with more or less difficulty of breathing, and rapidity of pulse.

[Hurried respiration is an early and common symptom. Of 91 pneumonic patients (adults), not previously treated, whose respiration was observed between the second and sixth days of the disorder, in 38 the number of respirations oscillated between thirty and forty, in 36, from forty to fifty, and in 9 it rose to fifty-four and sixty. They rarely exceed sixty, yet in two patients, one of whom recovered, they were respectively seventy-five and eighty. They are generally in proportion to the amount of disabled lung-substance, though this is not invariable, for sometimes in extensive single, or in double, pneumonia there are not more than twenty-five or thirty respirations, and in very limited lobar pneumonia they have exceeded fifty with intense dyspnœa. Extreme breathlessness is uncommon in the beginning of pneumonia; when it occurs it is gener-

ally where more than one lobe is attacked, or the whole lung is solidified, or the pneumonia is double. Andral, Bouillaud, Hourmann, Dechambre, Day, and others, have stated that the dyspnœa was more intense in pneumonia of the upper lobes than in that of the lower; but the observations of Grisolles show that this is not rigorously exact. Bergeron found in the aged that the breathlessness was more severe when the lower lobe was affected, and in double pneumonia. Pregnancy and thoracic heteromorphisms increase the liability to violent dyspnœa, independent of the site and extent of the disorder. The pulse is usually much quickened, varying in most cases from ninety to one hundred and six beats in a minute, and has sometimes reached, in cases which have ended happily, one hundred and forty. Its maximum of frequency is about the third or fourth day. It may be large and resisting, or sharp, but generally loses its hardness as the disease advances. The rapidity of the pulse is, with rare exceptions, in direct ratio with the extent and severity of the disorder. In the cases of pneumonia in the aged observed by Bergeron, the pulse oscillated from seventy to one hundred and forty beats, but in most of his cases it ranged between ninety and one hundred and ten.]

These are so correlated that the severity of the case is best measured by observing the correlation of these symptoms. The cough is [often present from the beginning. In 90 healthy persons attacked with pneumonia, it appeared in 80 within the first twelve hours, in 6 at the end of the first, and in 4 at the end of the second day. It is rarely as painful as in acute bronchitis, nor accompanied with its sharp, burning and tearing pain, behind the sternum, and in the direction of the bronchi. In old persons it very often precedes the pneumonic symptoms several days; generally infrequent and slight, it is occasionally constant, harassing and paroxysmal.] At first [frequent, short, and] dry, but soon there appears a scanty, semi-fluid, gray, frothy, mucous expectoration, which becomes more and more viscid, and which, in the first stage of the disease, remains of a catarrhal yellow or white appearance; [and sometimes streaked with blood, but rarely of a reddish hue.] Afterwards, during the second stage, it becomes still more tenacious, reddish, or rust-colored; and the heat of skin increases, with thirst and marked prostration, and a pulse increasing in rapidity.

[The sputa vary in color and consistence. The several hues are produced by the more or less intimate admixture of blood with the exudation. When they are light-red or rusty, the union is less perfect, and specks, resembling brickdust, are seen on their surface. They are often of an orange-yellow, apricot, or sugar-candy tint; and sometimes greenish, brownish, or black, like licorice or prune-juice. Sometimes throughout they remain white, opaque, and catarrhal. The red and yellow are viscid, adhering to the sides and bottom of the vessel, semi-transparent, and highly aerated. Sometimes their tenacity is less, they are more albuminous, run together, forming a homogeneous mass, which may be turned out of the cup, leaving it nearly dry. Occasionally they resemble in consistence and color a solution of gum-arabic. When dark-colored, the expectoration is serous, and may be covered with a light white froth. From the third to the seventh day the sputa may contain small ramifying membranous concretions. This peculiar character of pneumonic expectoration was first noticed by the late Dr. Remak, of Berlin, and may

be recognized by diluting the sputa with water and spreading them out on a glass slide; the concretions are then seen in the form of small branching cylinders, which divide dichotomously and are slightly swollen at the points of division. Primitive reddish sputa generally become yellow in the course of the disorder, but it is rare to see those originally yellow acquire a rusty tint. Green and prune-juice sputa are as a rule always consecutive. Sometimes the expectoration is entirely wanting; according to Grisolle and Bouillaud, when this is the case there is pneumonia of the summit. Chomel mentions an instance where the expectoration was alternately white and rosy, resembling well-whipped white of egg, with a reddish, transparent, consistent liquid, chemical analysis showing a large proportion of albumen. Rusty sputa last from one to twelve days—mean five; primitive yellow, two to nine days—mean four; and when consecutive, their mean duration is about three days. Where the issue is happy, the expectoration becomes mucous or sero-mucous, but retains for a while its tenacity. In the *aged*, the expectoration during the first stage is not characteristic. Durand-Fardel saw a case where it was prune-juice from the beginning. The sputa subsequently become adhesive and amber-colored, sometimes rusty, or are homogeneous and greenish, and contain fibrinous concretions. Towards defervescence they are often brownish, or even of a prune-juice color.

Headache is present in about seven-eighths of the cases, appearing on the first day,—if it has not been prodromic—is frontal, acute, lancinating, or constrictive, attains its maximum of intensity before the fourth day, and generally ceases about the seventh day. More or less tendency to sweating is observed in most cases of pneumonia from the first days. Delirium not infrequently occurs; it may be mere wandering, or violent, or low and muttering; it often has no relation with the degree of severity of the febrile symptoms; and most frequently accompanies pneumonia of the upper lobe. In the pneumonia of old persons it happens in one-third of the cases (DURAND-FARDEL, BERGERON); in 10 of Bergeron's patients there was pneumonia of the summit of the left lung in 5, of the right lung in 4 = 9. The decubitus is not constant, and is more influenced by the degree of pain than by the site of the pneumonia. If the pain is severe, patients generally lie on their back, or upon the affected side; when it abates, they change their posture, and their movements are freer. When the greater part of one, or both lungs is solidified, and the oppression and breathlessness are in proportion to the extent of disabled lung-tissue, the decubitus is dorsal, with the head and shoulders elevated.]

Such are the general symptoms of pneumonia; and, except by their different degrees of intensity, it is difficult, if not impossible, to distinguish the different stages of inflammation from each other, without the aids of percussion and auscultation. The general symptoms of serous pneumonia, however, are the most marked; the uneasiness being greater, the respiration louder and more difficult, the countenance more lived and swollen, the cough more harassing, the expectoration more abundant, and the attempt to lie down impossible. A gangrenous state of the lung is determined chiefly by the intolerable fetor of the breath.

[A favorable termination is tokened by amelioration of all the symptoms, general and local, the sputa loses its peculiar characters, and is



more abundant and opaque, the respiration is freer and slower, and the skin becomes cool and moist, the body-temperature falling rapidly. In fatal cases typhoid symptoms set in, the breathing is more hurried and oppressive, or gasping, the countenance pallid, the lips livid, the pulse very frequent, irregular, and thready, the surface cold and clammy, the expectoration thin and dark-colored, or suppressed, there is low delirium or coma, and death happens by apnœa.

Convalescence from uncomplicated pneumonia is generally rapid; the digestive functions are speedily re-established, and the appetite is imperious, and strength and flesh are quickly regained. During the progress of the disease there is, particularly at the period of development, generally, a considerable and rapid loss of body-weight, sometimes to the amount of two pounds or more in the day (WACHSMUTH); so soon as defervescence is established this stops, and the gain in weight is immediate and often considerable from day to day. In some cases local symptoms persist for awhile; the cough lingers, and auscultation shows that the affected lung-tissue is not perfectly permeable. It should be borne in mind that the dropping of the body-heat, near or even to the natural rate, does not show that the lung has cleared up; normal temperatures have been taken where there have been crepitation and bronchial breathing all over the lung (WATERS). Pain in the chest may be felt continuously or at times for some weeks or even months. Relapses are very rare, and are of shorter duration than the previous attack.]

The duration of a case of pneumonia varies greatly. Laennec conceived that the diffuse inflammation lasted seven or eight days. We have seen that in simple, uncomplicated cases the crisis is reached about the fourth or sixth days (WUNDERLICH, PARKES, METZGER).

[According to the observations of Wunderlich, Thomas (of Leipsic), Jaccoud, and others, defervescence begins in one-fifth of the cases of pneumonia, between the third and fifth days, inclusively; in three-fifths, between the fifth and seventh days, inclusively; and in the last fifth, after the seventh day, and usually from the seventh to the ninth.]

Andral records the duration of the disease in the adult at eleven days; Chomel and Laennec, at from seven to twenty days; Bouillaud, from eight to twelve days; Hourmann and Dechambre, fourteen days. [In 30 cases observed by Flint, the shortest duration was five days, and the longest twenty-three; the mean being a fraction over twelve days.] The mean duration in the aged, of 109 carefully recorded cases by Hourmann and Dechambre, was found to be nine days and seven-tenths; and Dr. Maclachlan agrees with them in considering the progress of pneumonia to be more rapid in the aged than in the adult. More generally, however, taking all its forms, cases of pneumonia terminate between the seventh and the twentieth day. [There is no tendency to defervescence on certain critical days.]

[TABLE SHOWING DURATION IN FIFTY FATAL CASES OF PNEUMONIA. (I. Russell.)  
DEATHS IN HOSPITAL, FEBRUARY, 1864.

INFLAMMATION OF LUNGS.	
No. of Days Sick.	No. of Cases.
2-6	6
7	3
8	2
9	5
10-11	2
13	3
14	3
15	3
16	3
17	2
19	1
20	2
21	3
24-28	6
33-38	2
40	2
60 and 72	2
Total number of cases, . . . . . 50	
Average number of days sick, . . . . 17	
" " "rejecting last 3, 13.62	

**Physical Signs of Pneumonia** are to be appreciated by *inspection, palpation, mensuration, percussion, and auscultation*. The physical signs of pneumonia, in the adult and in the aged, are so dependent on the morbid changes which have been described in the lung in the various stages of the disease, that a description of them is best given in correlation with the morbid anatomy of the disease. In doing so, the descriptions given by Dr. Fuller in relation to adults, and by Dr. Maclachlan in relation to the aged, will be mainly adhered to.

On striking the chest of a person in health it returns a certain hollow resonant sound, demonstrating it to be partly filled with air. Also, if we place the ear to the chest, we hear certain sounds, on inspiration and on expiration, which are termed the respiratory or vesicular murmur (as described at page 557, *ante*). In pneumonia these natural sounds are altered, the sound on percussion being rendered duller than natural, while the bronchial respiration undergoes still more remarkable alterations; and these modifications enable us, by percussion and auscultation, to determine the nature and seat of the disease. During the *first stage*, inspection of the chest shows that the costal movements are not materially diminished, unless the motions of respiration are restrained by pain. [*Measurement* sometimes shows local thoracic dilatation.] It is only by percussion and

auscultation combined, that the existence of pneumonia can be determined.

By *percussion* a peculiar euphonic resonance may be emitted by that portion of the chest at which pneumonia is beginning, which ceases after the commencement of exudation, to be replaced by dulness more or less marked according to the extent of the lung involved (FULLER).

[Appreciable comparative lessened resonance over the engorged lobe, with a higher pitch of the percussion-note and slightly increased resistance, are generally coincident with crepitation.]

During the period of invasion in the aged, the chest continues to sound clear over the seat of the impending inflammation; and as congestion increases, the lung becoming more dense and less permeable, the sound emitted on percussion may be somewhat duller; but in general there is no appreciable alteration till the *second stage* has set in (MACLACHLAN).

The signs elicited by *percussion* depend on the consolidation of the tissues, the amount and position of the consolidated part in relation to the surface of the chest, and the amount of intervening healthy lung. If the consolidation is near the surface, the sound is decidedly dull, and the sense of resistance to the extremities of the percussing fingers is considerable in proportion to the amount of the consolidation. To emit this unequivocal dull sound of hepatization in middle age, the disease must be extensive, and without intervening healthy lung; but in some cases, if the hepatization be central, the air in the more superficial portions of healthy lung often prevents a dull sound from being returned on percussion. In such cases percussion elicits a clear but shallow resonance.

[The degree of dulness of the percussion-note, and the resistance felt by the fingers, as a general rule, measure the amount and density of the exudation. There is rarely absolute flatness (wooden sound) unless pleural effusion is present. The pitch of the percussion-note is raised, and becomes often, especially over the anterior, middle, and upper regions, quite tympanitic, and the chest over the solidified lobe or lobes may give a louder sound than over the healthy side. The degree of resistance felt by the percussing fingers will serve to correct any error from increased intensity of sound. It is most important in percussion to note the sensation of resistance felt by the striking fingers. It sometimes happens that in very muscular persons, percussion gives no shade of difference in the sound in the second and third stages of pneumonia; but on the healthy side of the chest a certain degree of elasticity is always felt, whilst over the diseased lobe the chest is hard and resisting.]

A slight diminution in the resonance of the chest, immediately over the affected portion of the lung, is usually all that percussion elicits in old age, in the second stage of pneumonia. By auscultation during the second stage, tubular breathing is found to be a most constant accompaniment alike in the adult and in old age. The respiration is of a hollow character, diffused throughout the hepatized portion—tubular towards the centre, but harsh and blowing towards the periphery of the affected part. Where the tubular

breathing prevails, râles and rhonchi are absent; while vocal resonance is intense, usually of a metallic ringing character. This is most marked in aged persons. The *ægophony* in them is more decided, the shrill, acute, tremulous voice of old age being more favorable to *ægophony* than to *bronchophony*. When fibrinous exudation is great and complete, blocking up vesicles and tubes completely, absolute dulness on percussion, with little or no vocal resonance, can alone be heard; and the heart's sounds, greatly intensified, are then frequently transmitted through the consolidated lung.

*Auscultation* during the early period of engorgement, before exudation has taken place, finds the breathing weak in the affected parts, and exaggerated in their immediate vicinity.

[Dr. Stokes, of Dublin, was the first to point out that the earliest physical sign of the morbid change in the air-cells was harsh respiration, while Dr. Grisolle holds that weak respiration is the initial sign. The opportunities to verify or disprove either opinion are so rare that the question is still an open one. In a paper read December, 1865, before the Royal Medical and Chirurgical Society of London, Dr. A. T. H. Waters has reported two cases of acute primary pneumonia in lungs previously healthy in which "a loud, harsh, peculiar respiratory murmur was heard over the lower and back part of the left lung," and nowhere else, with pain in the left side, dyspnoea, percussion-sound and movement of chest natural, and which was replaced on the next day by well-marked crepitation and slight dulness in the same region. Mr. G. Stevenson Smith (*Ed. Med. Journ.*, November, 1866), mentions two cases of acute lobar pneumonia in young children, in which a harsh respiratory murmur preceded crepitation. The writer's observations, carefully made through a series of years, incline him to believe that both weak and harsh respiration may be heard at the very beginning of pneumonic engorgement, according as the bronchial or vesicular element of the compound breath-sound predominates. This view is in accordance with what is believed to be the true pathogeny of the disorder. The air-cells are alone supplied by the pulmonary plexus, and the terminal bronchi by the bronchial arteries. If the morbid process is limited to the air-sacs, the increased vascularity, and probable dryness and congestion of the membrane, will impair their function, and the soft breezy vesicular element will be feebler, while the natural sound in the minute bronchial tubes, more or less masked in healthy respiration, will become more audible, without absolute increased intensity, and may alone reach the ear. This view would seem to be strengthened by the statement of Dr. A. Flint (*Phys. Exploration of the Chest*, 2d ed., 1866), that in cases of pneumonia where a second lobe has been gradually invaded, he has noticed first exaggerated and then weakened respiration, the first being probably supplementary harsh respiration, and the feeble respiratory murmur marking the initial morbid change in the air-sacs.\*]

In the aged, tubular breathing is sometimes audible at the root

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\* [Since the above was written, the writer has read the remarks made at the New York Academy of Medicine, November, 1865, by Dr. J. R. Leaming (*Bul. of the New York Academy of Medicine*, vol. ii, Nos. 34, 35, 36; 1866), a careful and skilled auscultator, in which, speaking of pneumonia, he says: "During the stage of engorgement there is a muffling of the true respiratory sound, with slightly exaggerated broncho-respiratory murmur." This is substantial testimony in behalf of the writer's views, as given above, views held and taught by him for many years.]

of the lung at a very early period, if the respiration is weak. But as soon as fluid exudes into the affected parts, the respiratory sounds are obscured or replaced by the small crepitation characteristic of pneumonia. This sound, called crepitation, is made up "of a multitude of minute crackles, which occur in a volley towards the end of inspiration, and are unaffected by coughing or expectoration" (FULLER). It is the true crepitating râle of Laennec, and has been variously compared "to the crackling of salt thrown upon the fire," or to "the sound produced by rubbing a lock of hair between the finger and thumb close to one's own ear" (WILLIAMS). It usually, but not invariably, accompanies the accession of pneumonia.

[In 373 cases of acute lobar pneumonia observed by Grisolle, it was absent throughout the disease in 4 only. However perfect crepitation may be, it is limited to inspiration, and may be heard during the whole time, or solely during the latter half. It is often quite feeble, and is only audible after the deep inspirations which follow coughing. Though best marked when the first stage is fully developed, it is not unfrequently present for awhile in the second stage, and may persist through the course of the disorder. It may happen irregularly, appearing and disappearing at intervals. Diffused at first over the interested lobe, it becomes, as solidification advances, more circumscribed and occasional. Its apparent nearness and remoteness to the ear of the auscultator varies. The subcrepitant rhonchus, scanty and transitory, is not infrequent in the first stage of simple pneumonia in adults; when distinct and persistent it shows bronchial complication.]

[Crepitant rhonchus] is very seldom present in advanced life. In subjects beyond the age of fifty, and particularly in still more advanced age—as between sixty and eighty—the bubbles composing the pneumonic crepitation are very generally larger, more hurried, and less numerous. The râle essentially resembles the subcrepitation of capillary bronchitis, and is very often speedily marked by copious accumulation in the larger bronchi (MACLACHLAN, GRISOLLE, CAZENEUVE, HOURMANN, DECHAMBRE). When the air-cells are completely filled by the exudation, and the minute bronchial ramifications obstructed, crepitation ceases; and when capillary bronchitis commences, small bubbling râles are heard accompanying both expiration and inspiration. When acute pneumonia arises in connection with *acute rheumatism*, Dr. Fuller has often observed that crepitation never occurs. This, he believes, is attributable to the occurrence of the exudation into the interlobular connective tissue, and consequent exclusion of the air-cells (*l. c.*, p. 221). Vocal resonance is generally intensified. The return of the respiratory murmur, and the gradual disappearance of crepitation, indicate the resolution of the disease—a process which is generally more tedious and less perfect in the aged than in the adult—often followed in elderly persons by symptoms of continued irritation of the lung or bronchi. On the other hand, in proportion as crepitation marks the respiratory murmur, and replaces the pulmonary expansion, the supervention of consolidation may be anticipated.

Beyond the limits of dulness and consolidation, the extension of



fine crepitation denotes the extension of the *hepatization*; but when resolution has commenced, crepitation again returns to the part which was dull and consolidated before;\* or moderate-sized râles are heard, denoting the passage of air to and fro amongst the fluid which occupies the air-cells and smaller bronchial ramifications; and very soon the percussion-note assumes its normal character.

[The respiratory murmur rarely becomes integrally restored in all its properties until some time after complete convalescence. The bronchial element generally predominates for awhile, the vesicular element remaining feeble. In the resolution of pneumonia, bronchial respiration usually softens into broncho-vesicular, with the appearance of the moister breath-sounds. Of 103 convalescents discharged from hospital between the twentieth and fifty-fifth days of the disorder, in 37 no morbid physical lung-sounds could be heard; in 36 the respiration was feeble; in 14 harsh or slightly tubular; and in 16 there were subcrepitant and other bronchial rhonchi (GRISOLLE).]

During the *second stage*, inspection of the thorax shows the costal movements diminished in the affected side, and they may be somewhat increased on the unaffected side; but the movement of elevation is less affected than that of expansion.

[*Measurement* shows more or less increase in size of the affected side of the chest, depending for extent and site upon the amount and bounds of the lesion of the lung-tissue.]

By *palpation* we learn that *vocal fremitus* is usually above the average.

[The exceptions to this rule are not infrequent. In some cases no comparative disparity is apparent. In other instances the fremitus is greater over the sound lung. When the fremitus is lessened over the right lung, where it is naturally more marked than in the left, it shows that the effect of solidification, under certain circumstances, is to diminish it (FLINT). Where there is much pleural fluid effusion, it is greatly impaired or absent.]

And Dr. Fuller has verified the observation by post-mortem results, which showed that when the bronchi were plugged with exudation, vocal fremitus was entirely abrogated. The *fremitus* produced by a deep-toned powerful voice is seldom observed in the aged, and even *bronchophony* is occasionally absent (MACLACHLAN).

The passage from the first to the second stage of pneumonia is generally extremely rapid in old age, sometimes within a few hours (MACLACHLAN). In the *third stage* of the disease the physical signs do not differ from those already described; and neither in the adult nor in the aged are any certain phenomena appreciable by which the passage from the second to the third stage can be determined.

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\* [The *redox crepitant rhonchus* of Laennec, and stated by him and most authors to appear during pneumonic resolution, is a myth. The driest sound heard at that period is subcrepitant, and more commonly it is still moister. The process of resolution in a solidified lung and the mechanism of true crepitation are incompatible.—EDITOR.]

A persistence of the sounds already described, in place of those denoting resolution, and especially when the expression of the phenomena diminishes with increasing prostration of the patient, and diminished energy of voice and respiration, betokens suppuration of the lung, [along with purulent expectoration, sometimes sudden and copious, and that without the existence of an abscess.] It is by the succession of the physical signs in the adult and in the aged; by the occurrence of a rhonchus composed of large humid bubbles, without crepitation, in a portion or portions of lung previously consolidated; by the coincidence of this state with increased exhaustion; by a peculiar dusky, cachectic expression of the patient; by feebleness of the pulse; and by typhoid symptoms and bloody sero-purulent sputa,—that no room is left for doubting the accession of suppuration of the affected part (MACLACHLAN).

Fine crepitation of the lung cannot always be regarded as pathognomonic of acute pneumonia. In some instances it is absent throughout the attack, alike in the adult and in the aged. "It is only," as Dr. Fuller justly observes, "by the concurrence of different signs that it is possible to arrive at a trustworthy conclusion. Fine crepitation occurring coincidently with intense heat of skin and rusty-colored expectoration, warrants the strongest suspicion of pneumonia, and justifies the adoption of active treatment; and if it be accompanied by marked alteration in the ratio of the pulse and respiration, speedily followed by dulness on percussion and tubular breathing, the existence of acute pneumonia cannot be doubted. But crepitation, however fine, if not attended by an alteration in the ratio naturally subsisting between the pulse and the respiration [and I would add, the temperature], and not speedily followed by tubular breathing, cannot be relied upon as indicative of pneumonia. If it occurs without these symptoms, it is commonly indicative of capillary bronchitis with scanty secretion; whereas, if under the same conditions, it is accompanied by dulness on percussion, it is probably due to rapid œdema, or else to congestion of the lungs, connected with some febrile hæmic disorder, cardiac disease, or the deposit of tubercle" (*l. c.*, p. 229).

Pneumonia, it has been stated, sometimes, though rarely, terminates in abscess. The physical symptoms previously to the bursting of the abscess are those of hepatization; but supposing the abscess to have burst into the bronchial tubes, the pus of course escapes, and a cavity filled with air is left, communicating with the bronchial tubes, and this new state of parts gives rise to a new series of phenomena. The air, for instance, having penetrated into the cavity, the part which returned a dull sound while the abscess was yet unbroken, will now return a sharper and clearer sound on percussion than natural, denoting a larger admission of air than takes place in health. Again, on auscultating the chest, we find some changes have taken place both in respiration and in the transmission of the voice. If the cavity, for example, be large, and the opening small, the natural respiratory murmur at that part will be superseded by a sound resembling a person blowing into a jug, and from this circumstance termed by Laennec "*râle*

amphorique," or "bottle sound." Again, if the cavity be large and its walls dense, and the abscess still contains some pus, we hear a sound as if a drop of water had fallen into a pool; and this sound is so sharp and metallic that it has still preserved the designation originally given to it by Laennec, of *metallic tinkling*. It is usually supposed that this sound is produced by a globule of pus or fluid dropping from above into the fluid below; but some are inclined to believe that it is owing to the bursting of a bubble of air, mixed with the pus of the abscess. If, on the other hand, the abscess be large, and contains some pus, on the patient coughing we actually hear a *splashing sound* of the pus against the walls of the abscess, especially if the chest of the patient be quickly moved to and fro (succussion).

In aged persons, pneumonic abscesses are generally numerous, of limited extent, and interspersed through the lung. Physical diagnosis of them is therefore scarcely possible; but if the abscess be large, the sudden evacuation of purulent sputa, with gurgling and cavernous respiration, denotes the circumscribed suppuration of an abscess cavity.

Another circumstance revealed by auscultation in the event of an abscess is *pectoriloquy*. This physical sign is heard when, the stethoscope being applied to the chest, and the patient desired to talk, we hear his voice as if he were speaking directly at the end of the stethoscope, the sound passing to the ear as through an ear-trumpet.

*Pectoriloquy*, however, does not take place in all cases of abscess of the lung: its occurrence may be considered the exception rather than the rule in this disease. The cause of this is, that many conditions are necessary to its existence; first, that the lung must be condensed so as to have some conducting power, or the voice will be destroyed, as in health, before it reaches the aperture communicating with the abscess. Again, it is necessary that the patient should have a sufficient quantity of voice to produce strong vibration; but this is often wanting. Another condition is, that the bronchial opening of the abscess be not too large, for in that case the vibrating force is diminished. It is likewise injurious to the effect that there should be not more than one opening into the abscess; for in that case not only is the vibrating force diminished, but the counter motions of sound destroy all vibration. It is plain, also, that the walls of the abscess must have a certain density, or their flaccidity will act as damper, and destroy all vibration. Many conditions, therefore, are necessary to pectoriloquy; and we cannot feel surprised that one or more of these signs of an abscess cavity may be wanting.

Besides an opening into the bronchial tube, the abscess may at the same time open into the cavity of the chest, and this new pathological state gives rise to the sound of a *metallic tinkling* of the chest, infinitely more powerful than that caused by a simple pulmonary abscess. Indeed, the intensity and sharpness of the sound quite equals that returned by a copper vessel when struck with a slight

force; for the intercostal muscles brace the walls of the chest like a drum, so that they become an excellent conductor of sound. The immediate cause of the sound is supposed to be exactly the same as when it results from an abscess; that is, either a drop of fluid falling into the pus below, or the extrication of a bubble of air from the gravitated pus. The chest in these cases always returns a remarkably clear sound on percussion beyond the precincts of the fluid.

[Skoda says that he has never been able in a single instance to recognize the presence of pneumonic abscesses by auscultation and percussion.

**Causes.**—Acute primary lobar pneumonia is rare before five years of age, acquires its maximum between twenty and thirty, is frequent up to fifty, and becomes after sixty one of the most common and fatal of the diseases of old age. The influence of sex is apparent, not real, and is in fact due to different hygienic conditions, men being more exposed to the vicissitudes of temperature, which are, unquestionably, among the chief exciting causes of the disorder. The influence of sudden chilling whilst the body is overheated, is shown in 409 cases, of which 101 could be referred distinctly to this cause (CHOMEL, BARTH, GRISOLLE). With respect to season, 296 cases show the largest number happening in February, March, April, and May, in Paris (GRISOLLE). In 456 cases of pneumonia in adults, and 254 in old persons, there were:

ADULTS.				OLD PERSONS.			
In April, . . . .	.	.	104	In January, . . . .	.	.	51
March, . . . .	.	.	82	April, . . . .	.	.	46
May, . . . .	.	.	68	March, . . . .	.	.	44
February, . . . .	.	.	64	February, . . . .	.	.	42
December, . . . .	.	.	48	May, . . . .	.	.	27
January, . . . .	.	.	46	December, . . . .	.	.	24
November, . . . .	.	.	44	November, . . . .	.	.	20
<hr/>				<hr/>			
456				254			

As an intercurrent and consecutive disorder it is met with in the so-called blood diseases; it may be traumatic in consequence of contusions of the chest, and penetrating wounds; and is often developed by foreign bodies in the bronchia, particularly if they remain any time.]

**Diagnosis.**—Pneumonia is distinguished from phthisis by the previous good health of the patient, and by the more acute nature of the disease; and, in some degree, by a difference of its seat, the lower lobes being more particularly affected in inflammation, the upper lobes in phthisis. The two diseases, however, it should be remembered, are often combined. In determining distinctly the further diagnosis of pneumonia, from the phenomena of condensation and respiratory murmurs, it is necessary to bear in mind the various conditions which may produce condensation of the lung either in the child or in the adult. The observations of Laennec, Legendre, and Baillie, Jörg, Fuchs, Barthez, Killiet, West, and Gairdner, have especially elucidated this subject, and shown its importance as an element to be attended to in diagnosis. The following is a short statement of the conditions which lead to consolida-

tion of the vesicular tissue of the lung: (1.) A partial unexpanded state of the lung in a new-born child, termed *atelectasis* (JÖRG). (2.) Consolidation connected with the accumulation of mucus in the bronchi and air-cells—*bronchitis* in the infant, followed by what has been called *lobular pneumonia* of children, the *état fœtal* of the French (FUCHS, WEST, ZENKER). (3.) Collapse of the pulmonary air-cells, causing *lobular* or *more diffuse* forms of pulmonary condensation in adults, as well as in children, due to bronchial obstruction (GAIRDNER). (4.) It is probable that the *hypostatic pneumonia* described by Piorry, and the *pérépneumonie des agonisants* of Laennec, and some of the so-called *latent pneumonias*, are forms of condensation due to *pulmonary collapse*, combined with serous effusion or vascular congestion. (5.) The consolidation of inflammatory lymph in or about the pulmonary vesicles, so that the vesicular tissue of the lung is embedded in solid material, “as the stones of a wall are in mortar.” (6.) Condensation of the lung from the pressure of pleuritic effusion. (7.) Condensation of the lung from extravasation of blood (apoplexy of the lung), or from tubercular or cancerous deposits; or enlarged bronchial lymphatic glands, which are arranged along the sides of the air-tubes in their passage through the substance of the lungs.

Seeing, therefore, that consolidation may result under such a variety of conditions, the truth of the statement so well expressed by Dr. Stokes must at once appear in the strongest possible light—namely, “That in the cases we are every day called to treat, the value of physical signs must be tested by the history and symptoms (records of temperature and the like), and these in their turn must be corrected by the physical signs.” In truth, no disease shows more forcibly than cases of pneumonia do, and especially as regards diagnosis and treatment, that *every individual case of disease requires to be made a special study as regards its individual history, progress, combination, and sequence of symptoms*. In the aged, pneumonia is extremely dangerous, and often difficult to detect.

[**Prognosis.**—Acute single lobar pneumonia, happening between the ages of 15 and 40, when uncomplicated, except by limited pleurisy, is not a perilous disorder; recovery is the rule. Intercurrent or secondary, it is much more serious. The circumstances under which it occurs and the condition of the subject must always be taken into account in estimating the probable issue. The degree and extent of the physical signs alone should not influence the prognosis; the rational symptoms are surer guides, as they betoken the condition of the vital functions. No constant or reliable relation exists between the intensity and magnitude of the local lesion, as shown by the physical signs and the vital phenomena. Where the physical signs indicate amelioration of the lung disorder, and there is no amendment in the general symptoms, or where the latter are at the same time aggravated, the prognosis becomes uncertain. Primary pneumonia of the upper lobe is more fatal than that of the lower or middle lobe; the mortality is about 1 in 4 or 5. Pneumonia of the summit is often associated with tuberculization. In double pneumonia the mortality is about 1 in 2. Delirium is a very unfavorable symptom. Sex is an important element in the prognosis of pneumonia, it being from one-third to one-half more mortal in females than in males. Pregnancy



is an unfavorable condition, especially during the first six months. Second and subsequent attacks are more dangerous than first ones. Age has a decided influence upon the issue; of those attacked with pneumonia between 40 and 50 years of age, about 1 in 5 die, and after 60, more than 1 in 2; the peril is directly in proportion to the age. Pericarditis is a very serious complication. Heart clots may form in the cavities, more frequently the right, and cause sudden death; this is more often the case in old persons. Dr. A. Flint thinks that their presence may generally be ascertained during life, by the sudden worsening of the symptoms, irregularity, frequency, and feebleness of pulse, embarrassed respiration, and haggard face, the patient soon becoming moribund. Purulent expectoration, betokening the third stage, or abscess of the lung, is of serious import. The occurrence of symptoms indicating gangrene usually precede a fatal issue. But neither the third stage, nor abscess, nor gangrene are necessarily fatal; authentic cases of recovery from each have been recorded; but it must be remembered that pleural abscess may be mistaken for pneumonic abscess.]

Treatment of pneumonia has been, and seems with some still to be, one of the most discordant topics in the Science of Medicine. At one time, especially about fifty years ago, large bleedings appear to have been demanded, and to have been well borne. Laennec and Louis seem to have been early impressed, from their experience, that large bleedings were by no means an eminently successful practice, and that in some cases they were absolutely injurious. The same difference of opinion was held with respect to large doses of the *tartrate of antimony*. These discrepancies are now to be explained by the circumstance that pneumonia, like other inflammations, not only varies in type and severity in different cases, but that "extremes in practice," whatever they may be, when adopted as a *rule absolute*, applied to all cases, can never give satisfactory results. (See also p. 722, *ante*.)

The ancients bled in pneumonia, and sometimes to delirium, and Galen appears to have adopted this practice. This was also the practice of Sydenham; and Laennec says it was common in France, at the beginning of the last century, to take twenty-four, thirty, and thirty-six ounces of blood at one bleeding. This practice was also followed throughout Europe at that time, and there can be no question of its having been proper in cases of simple inflammations like pneumonia, occurring in healthy persons. It is clear that the remedy was used with a moderation apportioned to the severity of the disease and the strength of the constitution; or it was withheld altogether when the patient was weak, or the disease assumed a low type.

Now, however, it would be an unwarrantable error to make excessive bleeding the basis of remedial measures in all cases (see vol. i, p. 154, *et seq.*); but there can be no doubt that we have it in our power materially to modify the course, and to shorten the duration of pneumonia by the judicious employment of bloodletting, leeches, tartar emetic, certain salines, and opium (PARKES).

In epidemic pneumonia, says Laennec, it is hardly possible to bleed the patient without rendering him worse. In 1814, pneu-

monia was very common among the conscripts, yet there were few indications for bleeding, and those that were bled were rendered much worse. With respect to bleeding in cases of pneumonia, much must be left to the discretion of the practitioner. That there are cases in which the patient can only be saved by general bloodletting, everybody must admit, while on the contrary, when pneumonia is epidemic, the quantity of blood drawn must be greatly limited and the case well watched. The ancients held that bleeding should not be practised after the fifth day, as it prevented *concoction*. In other words, it tended to change the type of the disease, in its natural tendency to a cure, so that the processes tending to the resolution of the inflammation by the natural *cell-therapeutics* of the part were altered for the worse. The cases of Louis appear to establish the propriety of early bleeding as a general rule; for he says those bled in the first four days of the affection are cured four or five days sooner than those who are bled later in the disease. The experience of Dr. Alison is similar. In short, as a general rule, the earlier the inflammatory state is detected (if possible, before the third day—ALISON) the more likely will bleeding be followed by well-marked beneficial results—the disease will be sooner cured, and the convalescence more rapid and perfect.

It is rare now that the cure of pneumonia is left entirely to the influence of bleeding. Antimony is a most valuable remedy; and we owe to Rasori the introduction, in modern times, of large doses of *tartar emetic* in the treatment of pneumonia. Laennec was so dissatisfied with his own results of bleeding that he adopted it, and thus describes his practice:

“As soon as the disease is determined, if the patient be in a state to bear bleeding, I take from eight to sixteen ounces of blood from the arm. I do this as momentarily arresting the inflammation, and thus giving the tartar emetic time to act, and I rarely repeat this bleeding. Immediately after it I give the first dose of tartar emetic, or a grain in two ounces and a half of orange-flower water, and I repeat this dose every two hours for six times; I then allow the patient to repose for six or seven hours. If, however, the disease be severe and the oppression great, I continue the dose every two hours till the symptoms are mitigated, increasing the dose from one to two grains, or even to two grains and a half.” The immediate effects of this practice were, that the larger number of patients vomited two or three times, and had five or six stools on the first day, but afterwards the evacuations were trifling, and when tolerance was established, they often required purgative medicines, while many bore these large doses almost without vomiting or experiencing any purgative effect. The result was, that Laennec is said to have cured 27 cases out of 28 in 1824 and in 1826. The great success obtained by Laennec appears, however, to have been of short continuance, for M. Lagarde afterwards published an account of 16 cases treated by Laennec by this method, of whom 5 died. Lecoultreux has given a list of 30 cases likewise treated by Laennec, and of whom 12 died. Neither have other physicians in other years been more fortunate. Louis treated 15 cases according

to this method, and 6 died; Chomel, 140 cases, and 40 died; and Gueneau de Mussy treated 90 cases, of whom 38 died. Andral has treated a considerable number of cases of pneumonia by tartar emetic, in quantities varying from six to thirty-two grains in the twenty-four hours; and he adds: "I have seen but two unpleasant or grave accidents result from these large doses. Sometimes the patient has not been at all affected, and has neither had nausea, vomiting, diarrhoea, or abdominal pains; at others he has suffered from nausea and distressing vomiting—effects which have subsided on omitting the medicine. Tartar emetic," he adds, "may therefore be given with impunity. But is it useful? I have not seen pneumonia ameliorated by large doses of this medicine; for neither has it appeared to do good when borne by the stomach, nor when it has excited distressing nausea and vomiting."

Great doubts are now entertained regarding the usefulness of such large doses of antimony. The Rasorian method is decidedly contraindicated in the pneumonia of old people; but when the remedy is cautiously administered as a sedative, or diaphoretic and expectorant, in doses of *the eighth or twelfth of a grain every two or three hours*, and in doses of *a sixth or a quarter of a grain*, in the more sthenic attacks, it is a most valuable remedy (MACLACHLAN). It is contraindicated in gastric or gastro-enteric irritation. There are two kinds of cases where the remedy produces severe depression and nausea—cases in which the stomach will retain *no* food, scarcely even water when given alone. It is under such circumstances that Dr. Anstie, with justice, advocates the administration of alcohol in stimulant doses often repeated. He cites one case of double pneumonia in a man aged twenty-four, who beginning with twelve ounces of brandy *per diem*, ultimately took twenty-four ounces in divided doses every half hour, and for *ten* days lived on *nothing else*, and a little water. In one month he was able to resume his work. A child fourteen months old, under similar morbid conditions, was similarly treated with port wine and water. Taking about six ounces of wine a day, in spoonful doses, with water, the child subsisted on the wine and water *alone* for twelve days. At the end of that time, the stomach still refusing food, a little cod-liver oil was substituted for the wine, and in ten days more the appetite for ordinary food began to show itself, and the child made a good recovery (*Stimulants and Narcotics*, by Dr. Anstie, p. 446).

But in the statistics which bear upon the treatment of pneumonia, little or no reliance can be placed as a guide to practice. The results revealed are so variable and contradictory as to deprive them of the slightest claim to authority. For example: *Without depletion*, Dr. Bennett's statistics show a mortality of 1 in 21.4; Dietl's, 1 in 13; the homœopathic treatment (mainly expectant), 1, in 6; Vienna, 1856, 1 in 4. *With antimony and bleeding*, Grisolle lost 1 in 8; Dr. Bell, Glasgow, 1 in 17.7; Trousseau, 1 in 26; Wossildo, 9 in 76. *Treatment by the inhalation of chloroform*, Baumgartner, 1 in 10; Varrentrapp, 1 in 23.

Former statistics in the Royal Infirmary of Edinburgh are said to show a mortality of one in three.

These results are clearly too variable a guide to any sound basis of practice. No satisfactory conclusion can be drawn from cases massed together. Each case must be studied by itself, as well as each epidemic. (See also pp. 722 and 752, *ante*.)

The numerous qualifying conditions connected with age, season, climate, epidemic and endemic influences, earliness of treatment, stage, extent, and complications, all tend to modify the line of treatment required. Dr. Lawson, Professor of Medicine in Cincinnati College, has well expressed these conditions, in a general sense, under the designation of "*the individuality*" of each case.

"Indeed," he remarks, "so great are the differences in constitutions, that no two examples will exhibit the same characteristics throughout, nor will they admit of precisely the same method of treatment. It is a due appreciation of these more minor shades of differences, as well as the broad distinctions observed in the varying *forms* of the disease, that denotes the truly skilful physician, and which enables him to meet the emergencies of each case, instead of relying on conclusions drawn from groups of cases. It has been remarked that English physicians are apt to think more of some other case they have seen than the one under treatment; while the French think more of the disease than of the patient. Hence English physicians *individualize* the disease; the French physicians *generalize* the patient.

"The true course has been indicated by Hufeland—namely, to *generalize the disease and individualize the patient*. The tendency of statistics is undoubtedly to rob each case of its individuality. Thus, one group of cases will all be bled, another will receive tartar emetic, a third will be left to the chances of Nature. In the first group some are bled who ought to have been stimulated; in the second group *tartar emetic* is administered when *bleeding* would have been preferable; in the third group some are permitted to die from over-action. Thus the individuality of a case is ignored, and the physician prescribes for a mere *name*."

A rational treatment is one which must secure to each case its own individuality. And as the shades of differences and the corresponding modifications of treatment cannot be expressed in *groups*, statistics in this sense become simply an impossibility. For example, bleeding, antimony, mercury, and blisters may be demanded in one case; quinine, opium, and wine in the next; a third may require but little interference beyond a well-regulated diet, with moderate stimulants.

The treatment of pneumonia, therefore, demands not a single, but many agents. It is the proper *combination* of remedies, not a single agent or mode of practice, which must be sought for in the treatment of pneumonia (LAWSON).

It now only remains to add the more matured opinions entertained as to the line of treatment to be adopted with pneumonia as it now occurs. In cases of sthenic pneumonia, characterized by intense heat and dryness of the skin, a full resistant pulse, rusty-colored expectoration, and *great oppression of the breathing*, blood-

letting, had recourse to at the beginning of the attack, in the stage of congestion before the *fastigium* of the pyrexia, not only affords immediate relief to the breathing, but appears to remove the extreme tension of the vascular system, and to promote secretion. The most suitable time is that during the evening exacerbation (Huss). The indications for bloodletting should be decidedly marked before it is undertaken ; and the amount of blood to be lost must vary in each case, according to the oppression of breathing, the type of the disorder, and the constitution of the patient, as well as to whether or not there is any prevalent epidemic tendency associated with the pneumonia. It is seldom necessary to draw more than from *ten* to *sixteen* ounces ; and *eight* or *ten* ounces will usually suffice. After bleeding, the pain in many instances ceases, expectoration takes place more easily, and alters in character ; the skin becomes moister, and evidence is afforded of the action of remedies which before proved inoperative (FULLER). In very many cases it is now either unnecessary or inexpedient to let blood—inexpedient chiefly because of the constitution of the patient being shattered by excesses, constitutional disease, anxiety, and mental distress ; and excessive bloodletting, under whatever circumstances practised, impairs the strength, leads to great impoverishment of the blood, arrests the actions on which the absorption of exudation-matter depends, exposes the patient to risk, and induces a tardy convalescence.

Bleeding, therefore, ought certainly not to be employed after consolidation has taken place.

A combination of *antimony* and *calomel* is believed to have saved a much larger number of cases than *antimony* alone ; a quarter of a grain to a grain of the *tartrate of antimony*, combined with one grain of *calomel*, given every four or every six hours, according to the severity of the disease, is the treatment in some cases to be adopted. Previous to its use the bowels should be well cleared out, and after the mercurial effects are indicated by the condition of the gums, the further administration of the remedy should cease. In cases of simple serous pneumonia even simpler remedies are sufficient ; and two grains of *ipæacuanha* given every four or six hours have frequently been followed by the recovery of the patient. The cases in which *tartar emetic* fails to exercise a curative action are those in which hepatization proceeds with extreme rapidity ; in which crepitation either does not exist at all, or is of very short duration, giving place after a few hours to intense tubular breathing : and those which are marked by extreme depression almost from the first.

*Salines* and *stimulants*, with *calomel* and *opium* if necessary, are found to be the most efficient means of treatment in such cases. The efficacy of *mercury*, in the experience of Dr. Fuller, is most conspicuous in those cases of pneumonia in which *tartar emetic* is of least avail ; in other words, in those instances in which the productive results of inflammation are the greatest, in cases in which crepitation does not exist at all, or is replaced in a few hours by intense tubular breathing (*On the Chest*, p. 238). In these, *calomel*



and *opium*, in combination with *salines* and small doses of *tartar emetic*, will often produce very beneficial results.

*Alkaline Treatment of Pneumonia.*—Dr. John Popham, Physician to the Cork North Infirmary, since 1865, has treated twenty-eight cases of pneumonia by alkalies. They all recovered. In some the signs of engorgement only existed at the time of admission; in others, hepatization was found. The reliable marks of pneumonia were present in all—*e. g.*, localized dulness, crepitation, tubular breathing, rusty sputa (in many), and labial herpes in about half. Six were cases of double pneumonia. *Bicarbonate* of potash was given, largely diluted in mucilaginous liquids, five grains up to thirty grains in each dose,—four, six, or even eight doses being given in the twenty-four hours. An adult took to the extent of two to three drachms of the salt per day. The evidence of its good effects appeared first in altering the character of the expectoration on the second or third day of its use. The viscid sputa became resolved; the fine bubbles became coarse and large; the rubiginous color of the expectoration was changed to white; its tenacity of adhesion was lessened, so that it was brought up easily; and the cough, instead of continuing dry, harsh, and irritative, became moist, soft, and expulsive. Thus the alkali acted as a sedative, allaying the cough and abating the congestive state on which it depends. The white pasty fur over the tongue dissolved away in an increased flow of saliva. The urine became alkaline, and the physical signs of pneumonia became resolved. A blister applied for four or six hours (not for suppuration) is a valuable auxiliary; suppuration from blistering is exhaustive and prejudicial (*Brit. Med. Jour.*, Dec. 28, 1867, p. 586).

The treatment by repeated stimulants alone is but the opposite extreme of practice to excessive bloodletting, and will no doubt soon find its level and appropriate place as a valuable aid to other remedies, especially in patients whose constitutions are depressed under the influence of life imposed upon them by the "*great town system*."

[The rational or restorative treatment of pneumonia agrees with the true pathogeny of the disorder, and takes cognizance of its mode of resolution. It has been shown that the solidified lung-tissue regains its natural state by histolysis of the granular exudation, a process of degeneration and disintegration. In healthy subjects there is a natural tendency to resolution, and the disease passes through what may be called the pneumonic cycle, in a variable period, but as definitely and methodically as an eruptive, or typhoid, fever. The expectant experiments of Balfour, Magendie, Dietl, of Vienna, Skoda, Niemeyer, Schmidt, Legendre, and others, prove it. Dr. Bourgeois (d'Estampes) for twenty-five years abstained from all active treatment in his cases of pneumonia. He found, towards the eighth day, a decided drifting towards amelioration in the symptoms—the sputa were less viscid, the breathing was easier, the tongue cleaner, the pain in the chest gone, restlessness and wakefulness were lessened or had disappeared, and there was a desire for food, and on the ninth or tenth day convalescence was fully established (*Union Médicale*, January, 1850, vol. i). Rational treatment, whilst it favors the local

vital act, at the same time recognizes the general condition ; on the other hand, spoliative and depressing treatment, hinders the one, and takes no heed of the other. The rational treatment, besides, is abundantly vindicated by large and trustworthy experience, the results of this practice being greatly more happy than those of any other, and favorable testimony in its behalf is daily accumulating.

Dr. John Turner (*Clinical Notes from the Dumfries Royal Infirmary, Ed. Med. Jour.*, October, 1866) says: "Our cases of pneumonia treated on the restorative principles advocated by Prof. Bennett, have been eminently successful. Those cases left almost entirely to the unassisted process of nature have invariably done well. In looking over the available hospital records, I find mention made of 71 cases of pneumonia that were treated in these wards from the year 1820 to 1836. Of these 12 died, or about 1 in 6. During these years venesection was freely practised." From the statistics collected by Dr. J. Hughes Bennett (*The Restorative Treatment of Pneumonia*, 3d ed., Edinburgh, 1866), it would appear that: (1.) An extreme antiphlogistic treatment has always been attended with a large mortality, amounting to 1 death in 3 cases ; but when variously modified, by lessening the amount of lowering remedies, selecting cases, or in quite young and vigorous subjects, the mortality ranges from 1.5 to 1 in 13 cases. (2.) That when one-half the cases are those of persons below twenty years of age, and the spoliative treatment moderate, the deaths are diminished to 1 in 28 cases. (3.) Treatment by large doses of tartar emetic, the mortality is from 1 in 4.5 to 1 in 9.60. (4.) Dietetic or expectant treatment, mortality from 1 in 7.25 to 1 in 10.9 in adults, and in children 0 (BARTHEZ). (5.) Mixed treatment, 1 death in 3.20. (6.) Tonic treatment (KISSEL), mortality 1 in 22 cases. (7.) Stimulants (TODD) 1 in 9. (8.) Restorative treatment (BENNETT), in the worst point of view, 1 death in 32.70 cases. (9.) 105 uncomplicated cases, occurring consecutively, during a period of 16 years, in the Royal Infirmary, Edinburgh, treated on the restorative plan, all recovered. In 189 cases treated solely by the expectant method, the mortality was 3.10 per cent. (DIETL).

Of the immediately relieving effect of general bloodletting in many cases of pneumonia there can be no doubt ; that it has no curative power over the disorder is satisfactorily established ; and that, except in quite young and vigorous subjects, it is harmful, is now very generally admitted. Trousseau, who did not absolutely reject bloodletting in the treatment of pneumonia, says : "If usually I abstain from venesection, it is not because I think it so often the cause of death in the disorder, but that experience has taught me that it rarely shortens its duration, and that it often hinders convalescence by weakening the patient" (*Clinique Médicale*, &c., 2me ed., t. i, p. 740), no doubt remembering with Kaltenbrunner that a certain amount of strength is necessary for the resolution of any inflammation. Dr. Fuller writes : "In my own practice I have had recourse to phlebotomy in pneumonia three times only within the past seven years ; but I am satisfied that in each of these cases it was the means of affording great relief, if not of saving life, and that cases do occur in which its employment is absolutely necessary to the well-being of the patient" (*On Diseases of the Lungs and Air-passages*, 2d Am. ed., p. 249). All the aid which general bloodletting may give can be got from local bleeding by leeches or cups—preferably the latter—and then it should be used discreetly, and not later than the first stage. There is no substantial evidence on the side of the treatment by tartar emetic, but much against it. The circulation can be more safely and surely controlled by the careful administration of aconite, veratrum, digitalis, and colchicum ; without

the risk of seriously, and often irreparably, lowering the vital powers, and disturbing the digestive functions. For the past twenty years, the writer has substituted the muriate of ammonia for calomel, in the second stage of the disorder, giving it in one- or two-grain doses, every hour or two. Dr. R. Cresson Stiles states, that for some time past he has used satisfactorily the carbonate of ammonia—one drachm in solution every two or three hours. By a series of carefully conducted experiments he is convinced that it possesses no stimulant property. It is converted in the stomach into the hydrochlorate of ammonia (*Medical Record*, Oct. 1, 1866). Dr. J. Hughes Bennett during the period of febrile excitement gives salines in small doses, and seems to prefer the acetate of ammonia in solution. Dr. Trousseau has for many years past relied chiefly upon Kermes mineral—antimonii oxysulphuretum. Opium is beneficial in all stages of the affection, and subcutaneous injections of morphia, or atropia, about the seat of the chest-pain, will generally give instant relief. The affected side may be kept covered with hot poultices of linseed, or cornmeal, or mashed potatoes, or with cotton batting, over which a jacket of oil-silk or -muslin should be worn. Purgatives should be used with great caution, the bowels being kept free by simple enemata. In simple acute lobar pneumonia in young subjects and previously healthy adults, alcoholic stimulants are rarely required. In the typhoid form of the disorder, and in feeble and broken-down persons, and in the aged, they form, along with tonics and food, an integral part of the treatment. In the latter part of the second stage, and during resolution, tonics are often necessary, and iron, quinine, and arsenic may be administered with advantage. The indication of nourishment, properly adjusted to the circumstances and conditions of each case, and each stage of the case, is paramount to all other indications, and the happy issue of a case of pneumonia depends quite as much upon early and properly feeding the patient, as does that of a case of continued fever. A milk diet should be permitted from the outset, to be followed, as the disease advances and the pulse softens, by beef-tea, &c. Wine or brandy, in occasional doses, may be required, but in nowise can they be regarded as the equivalent of food. Intercurrent delirium is treated by Trousseau with musk; it is often the result of inanition, and food, stimulants, and opium will generally best control it. The advantage of the so-called expectorants is doubtful, and they are apt to produce more or less gastric trouble, and interfere with nourishment.]

#### [SCLEROSIS OF THE LUNG.]

*Fibroid Degeneration of the Lung. Pulmonary Cirrhosis. Fibroid Phthisis. Granular Lung*—Syn., LAT., *Induratio Pulmonum*; FRENCH, *Cirrhose Pulmonaire*; GERMAN, *Cirrhose der Lunge*.

(DR. CLYMER.)

**Definition.**—*A chronic pulmonary affection of middle and advanced life, whose anatomical character is hyperplasy of the connective tissue, or interstitial stroma, of the lung, partial or generalized, and passing to fibroid change; there is failing health, with local symptoms of lung-trouble, and the physical signs of induration of the diseased portions of the organ, and often later the evidences of cavities; generally associated with fibroid change in other organs, and usually preceded by acute bronchial or pleuritic disease; most frequently met with in spirit-drinkers; not apparently connected with tuberculosis, but, probably, due to a specific diathesis.*

This disorder has recently attracted a good deal of attention, and its

morbid anatomy and clinical history have been studied, particularly in England, under the name of Fibroid Degeneration of the Lung, and by that name it was described by the writer in the first American edition of this work. Sclerosis of the Lung is now substituted as, provisionally, a better term, and until its true nosological position is decided upon. The characteristic morbid changes of this affection were long ago noticed by Bayle as a variety of phthisis (1810), by Laennec, Andral, Broussais, Addison, Chomel, C. J. B. Williams, Stokes, Corrigan, Rokitansky, Laycock, and Hughes, and more lately by Dr. S. Wilks, H. G. Sutton, and H. C. Bastian.\*

\* Obs. 20 of Bayle (*Recherches sur la Phthisie*, par G. L. Bayle, Paris, 1810), is "Phthisie avec Melanose" in a man æt. 69. The post-mortem pulmonary condition is thus described: The upper and middle lobes of the right lung were very hard, and when cut into grated under the scalpel, and were as black as ebony; at the base of these lobes were several empty cavities, lined by pyogenic membrane. In the inferior lobe were several small cavities surrounded with black matter. *There was no tubercle.* The upper lobe of the right lung was covered externally by fibrous membrane, very firm and adherent. The left lung presented the same alterations as the right, but in a less degree; the upper lobe was only much changed posteriorly. Obs. 22 is, "Phthisie granulaire avec melanose et quelques tubercules," in a woman with asthenic gout, æt. 72, and who died with extreme marasmus. The upper fourth of the right lung was hard, black, and grated under the knife. In cutting into the tissue, it looked like some species of granite, and as if made up of a number of round granulations of a dark slate color (*noir ardoisé*), of the size of a pea, and united by a softer substance of the same color. The lower three-fourths of the lung was healthy. In the left lung there was throughout its whole extent the same alteration, and scattered here and there were a few suppurated tubercles.[?]

Andral (*The Clinique Médicale*, by D. Spillan, London, 1836), gives a case of chronic bronchitis with melanosis, in a man, æt. 65, who had had winter cough and breathlessness for ten or twelve years, and whose lungs after death were found to be infiltrated with black matter. Also another (18) very similar. Andral describes chronic pneumonia as a slowly developed hypertrophy of the septa of the lobules and cells, attended with a gradual deposition of albuminous matter in the interstices of the pulmonary tissue, and Hope (*Path. Anat.*) adopts this description.

The late Dr. Thomas Addison (*Obs. on Pneumonia and its Consequences*, *Guy's Hospital Reports*, second series, vol. i), mentions three permanent effects of pneumonia: (1.) Uniform albuminous induration; (2.) Granular induration; (3.) Gray induration. In the first, a uniform, homogeneous, semi-transparent or opaque yellowish material is diffused through several lobules, or the greater part of a lobe, in which there is not the slightest trace of the air-cells or interlobular cellular tissue, the lung-tissues seeming blended with, or assimilated to, the permanent albuminous deposit. In the second, a solid, pale or yellowish albuminous less organizable material occupies the lobules, and apparently without having assimilated the parietes of the cells, which are still visible on the exterior, and in the interior of the lobules; the interlobular tissue being sometimes, though not invariably, distinct. The third he describes as a gray induration, composed of a mixture of dull or yellowish-white and black matter in variable proportions, the tint depending on their proportions, and says that he does not see how it can result from chronic pneumonia.

Dr. W. Stokes (*Treatise on the Diagnosis and Treatment of Diseases of the Chest*, Phil. ed., 1844), describes the lesion under the head of Senile Phthisis, and also of "Tubercle the Consequence of Chronic Bronchitis," and says that he has seen two cases of chronic abscess of the lung without any evidence of tubercles.

Speaking of the condition of lung described in the text, the late Dr. Hughes has most justly observed, that if it is to be looked upon as a consequence of unresolved pneumonia, then it is a very rare disease; but a state of the lung through which are distributed firm, dry masses, of various shades of gray, which creak under the scalpel, of size from a pin's head to a walnut or pullet's egg, is by no means rare in London.

Dr. Thomas Laycock (*Clinical Lectures on the Physiognomical Diagnosis of Disease*, *Med. Gaz. and Times*, vol. i, p. 451, 1862), speaking of the physiognomy of what he calls rheumatic tuberculosis, says: It happens chiefly among the intemperate; there is fibroid degeneration of the lung with cartilaginous thickening of the pleura, most strikingly seen at the apices, and excavations like ragged holes; the sputa is apt to be fetid; the morbid product much more plastic than in scrofulosis, and tubercle is rarely or never found; but there is fibroid degeneration of other organs, spleen, he-



**Morbid Anatomy.**—In most cases both lungs are affected, though in different degrees. When the degeneration is limited to one lung, it is most often the left. As a rule the morbid change begins in the upper lobes; in thirty-four cases recorded by Dr. Sutton, there was but one exception, and in this it was limited to the lower lobes. Fibroid pulmonary degeneration occurs in two forms: (1.) Nodular; (2.) Diffused.

(1.) On examining an indurated nodule, the lung-tissue is found to be dense, tough, solid, dry, heavy, sinking in water, and is broken down by the finger with difficulty; when cut into, it is resisting and sometimes grates under the knife. The cut surface may be granular or not, or present both appearances in the same lung. When apparently smooth, close examination will show the indurated portion nearest the sound tissue to be granular, and in parts often to such a degree as to resemble a number of minute, hard, semi-translucent tubercles, closely clustered like the eggs of some insects, each granule being in the midst of augmented interlobular tissue. Scarcely any moisture can be squeezed out. There is proliferation of the interlobular connective tissue, and it is consequently unusually distinct. On placing a small section of the nodule in water, and throwing a strong light upon it by means of a condenser, it seems to be divided and subdivided by innumerable ramifications of an overgrowth of connective tissue, within which, and surrounded by more or less dark pigment-matter, are minute polygonal spaces containing the air-cells. The whole surface is of a bluish-gray color, of different shades, from iron gray, a gray granite, to nearly black.

The microscope shows the induration to be made up of such elements as are usually regarded to denote fibroid changes. There are well-formed cells, and the cellular elements in various stages of development seem being converted into highly organized fibrous tissue, and are strikingly different from the granules and imperfectly formed cells of lowly organized scrofulous material. The lung-tissue in the neighborhood of the fibroid masses is elastic, somewhat crepitant, and floats in water. The interlobular connective tissue is increased, and contains dark pigment-matter, showing that morphological changes of the connective tissue precede solidification. In the other portions of the organ it may be quite healthy.

(2.) In some cases there are no circumscribed hard masses, but the whole lung, or the greater part, is uniformly converted into tough fibrous tissue; its natural aspect is lost, its structure destroyed, it is hard and contracted, and upon section looks striated and as if interwoven with fibrous filaments. There is great proliferation of the connective tissue, which has a granular appearance, as if studded with miliary tubercles. Nowhere are any granules to be found except in the increased connective tissue. In a less degree this has often been noticed in the upper lobes of those dying from cirrhosis of the liver, granular degeneration of the kid-

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patie peritoneum [fibrous capsule?], kidneys, and occasionally of the connective tissue of the liver.

Rokitansky (*Path. Anat.*) describes the anatomical characters under the head of Interstitial Pneumonia.

Dr. S. Wilks says (*Trans. of the Pathological Society of London*, vol. vii, 1864), that gray induration or chronic pneumonia occurs often where there is no history of previous acute inflammation, and he thinks it more allied to a growth than to an inflammation.

*Fibroid Degeneration of the Lung.* By Henry G. Sutton, M.D. *Medico-Chirurgical Transactions*, Second Series, vol. xlviii, London, 1865. A valuable paper, with several cases, and an analysis of many others, collected from the records of Guy's Hospital.

*Cirrhose Pulmonaire*, par le Dr. Feltz. *Gaz. Méd. de Strasbourg*, No. 1 and 2, 1865.]



neys, and in the summits of the lungs of old persons, and probably indicates the early stage of fibroid degeneration.

In some cases there are one or more cavities in the indurated substance at the apices; irregular and ragged, their walls are formed, partly by more or less sound lung-tissue, and partly by tissue which has undergone fibroid degeneration, and they contain an offensive dark-red fluid. Smaller cavities may coexist, varying in number, and scattered through the indurated tissue, having a very thin membranous lining, continuous with the lining of the bronchi, which may open into them, and often look like circumscribed dilatations of portions of the tubes; or the lung-tissue may soften into several small cavities. In the midst of the consolidated lung, there are frequently soft yellow cheesy masses.

Tubercular matter is not commonly found in any part of the lungs. In 30 cases analyzed by Bastian in only 4 was there any evidence of the presence of tubercle in one or other lung, and then in quantity altogether insignificant, so as to make its presence rather accidental than essential. There is generally evidence of long-existing disease of the bronchi, which are enlarged, and may be more or less dilated (JACCOUD). There is probably, however, no necessary association between bronchiectasis and pulmonary sclerosis; for although in the former disease the sclerosis is localized in the lung-tissue about the affected air-tubes, the generalized fibroid change of the lung may happen with or without alteration of the calibre of the bronchi. In the majority of cases the pleura over the diseased lung is thickened, sometimes to the extent of one-fourth to one-half of an inch, and fibrous bands have occasionally been seen to extend from the thickened pleura into the substance of the lung. At other times the pleura is slightly, or not at all, altered. The bronchial glands are generally enlarged, and are, in some instances, changed into firm, hard masses, which grate under the knife.

In the 34 cases analyzed by Dr. Sutton, the kidneys were diseased in 19, healthy in 5; in 18 there was granular degeneration, and in 1 suppurative nephritis. In 30 in which the condition of the liver was noted, it was diseased in 20, healthy in 10; it was cirrhotic in 10, fatty in 3, nutmeg in 5. In 25 cases there was no disease whatever of the intestines, in 9 not noted. In 21, the heart was diseased in 17, healthy in 4; not given, 13; there were fibroid changes in valves and muscular tissue in 2, hypertrophy and dilatation in 15. Dr. S. Ringer asserts the frequency of lardaceous change in other organs.

Microscopical examination of the indurated lung-tissue and hard nodules leads to the conclusions that: (1.) There is a production of new tissue elements, and that these are what are usually considered as representing newly formed connective or fibroid tissue; (2.) That this new formation invades and destroys in part or wholly the lung-tissue, which undergoes fibroid change. Every new formation, according to Virchow, whether homologous or heterologous, is really destructive, and destroys something of what previously existed; (3.) These fibroid elements are found most highly developed in the connective tissue surrounding the minute bronchial tubes, lobules, and lobulets, as well as in the neighborhood of the thickened pleura, and would seem to show that the new formation begins in the connective tissue of the lung, and extends in every direction amidst the elastic fibres of the air-sacs, until these become degenerate, and are obliterated. With regard to the origin and nature of the yellow or cheesy matter found amidst the gray fibrous induration, the microscope shows it to consist of a large number of granules, having bright sparkling centres, and of irregularly shaped and apparently shrivelled or wasted cells, filled

with what are commonly called fat-granules. They thus seem to be retrograde metamorphosis in the fibroid formation, and to result from an imperfect nutrition process. In fibroid growths elsewhere, as in the uterus, circumscribed fibroid deposits in the kidneys, syphilitic tumors in the lungs and liver, and in cancer of the lung (vol. ii, p. 200), yellow, cheesy centres occur.

**Symptoms.**—With our present clinical knowledge of fibroid pulmonary degeneration, it may be said to be generally preceded by an acute bronchial disorder, which has become chronic, or by acute pleurisy. Failing health, increasing weakness, constant cough with expectoration, and breathlessness, are complained of, with, in advanced cases, great lividity of the face. There is not commonly at the beginning much loss of flesh, but after awhile the waste is gradual, and when the physical signs indicate induration of the apices, or when tissue disorganization has taken place to any extent, it is very rapid. Profuse hæmoptysis may be an initial symptom. The expectoration is that of chronic bronchitis, and is often slightly streaked with blood. There does not appear to be any continuous elevation of the body-heat. The function of the digestive organs is but little impaired; the appetite is good and regular, unless there be some complicating disorder, as jaundice, from cirrhotic liver, a fibrous pylorus, etc.; there are no symptoms of intestinal affection throughout its course.

The *physical signs* are those of different degrees of pulmonary induration, combined with those of chronic bronchitis and emphysema. Over the site of the lesion there is flattening of the chest-wall, or even contraction; percussion-resonance is diminished, sometimes to absolute dulness, along with increased resistance to the striking finger. Auscultation gives feeble respiration, very prolonged expiration, tubular breathing, increased vocal resonance, and whispering bronchophony, along with distant, moist, and sometimes large, crepitation. When cavities exist they are indicated by the usual signs, as is also coexistent emphysema, and bronchiectasis.

Its progress is very slow, extending over months, and even years. When death is not caused by one of the coincident fibroid organic diseases, it is most frequent from intercurrent capillary bronchitis, acute pleurisy, or pleuro-pneumonia. In one reported case sudden and profuse hæmoptysis killed the patient; there was also circumscribed pulmonary gangrene (SUTTON).

Associated so frequently with fibroid disease of other organs, as granular kidney, fibroid degenerations of the heart, endocardium, pylorus, capsule of the spleen, bronchial glands, and skin, the symptoms must be materially modified by them, and it is impossible to give a simple history of the disorder itself. In only two out of thirty-four collected cases was it the only apparent disease.

**Causes and Pathogeny.**—Sclerosis of the lung is more frequent in males than in females. In 32 cases where the age is given, there were 31 men, and 1 woman. It is much more common in middle and advanced life. In 34 cases, under 29 years, none; between 29 and 40, 10; 40 and 50, 7; 50 and 60, 12; 60 and 70, 3; 70 and 80, 2; thus between 29 and 60, there were 29 cases, out of 34 (SUTTON). In 20 cases where the habits of the patient are mentioned, 17 were intemperate, 1 was a free liver, and in 2 there was no evidence of intemperance. In 14 cases out of 20 there was a previous history of winter cough and breathlessness, extending over several years. In a certain proportion of cases there will be found evidence of former attacks of pleurisy; and coincident fibroid degeneration in other organs is very common, and which in some cases

may have been antecedent. There is no evidence that the disease is associated with scrofula. The yellow, cheesy matter found amidst the gray indurated lung-tissue is constantly seen in new formations whose cells have undergone atrophy and fatty change. No lesions of the intestinal glands are found, nor any intestinal ulcerations. Nearly all patients with fibroid degeneration have about the same corporeal characteristics,—the physical type is good; the subjects are well built; the skeleton and muscular system well developed; the skin thick; the hair abundant, and the central teeth even, and in advanced life sound and regular. Loss of weight does not take place to any extent at the beginning, and does not become excessive until later, when the apices of the lungs are disabled, or the degree of degeneration great.

By many of the British and French authorities, Sclerosis of the Lung has been described as chronic pneumonia; but in chronic pulmonary inflammation the lung is not shrivelled or contracted, and, although rather hard and elastic, is comparatively readily broken down. The lung-tissue is firmer and drier than in true chronic hepatization, scarcely yielding a trace of moisture when scraped, and often creaking under the knife. Dr. Fuller believes that these cases form a connecting link between chronic inflammatory consolidation and tuberculous infiltration, and should be classed under the head of phthisis, as being more nearly allied to that disease than to simple inflammation of the lung (*loc. cit.*, p. 258). Dr. Wilson Fox thinks that it belongs to the tubercular constitution, and is a more frequent termination of the tubercular process than the cheesy change. Dr. C. J. B. Williams, who published two cases of the disease thirty-four years ago, looks upon it as a modification of ordinary phthisis, its distinctive character being its tendency to chronicity, to which it owes its comparative curability. Dr. J. Pollock is of opinion that it is not a specific entity, and cannot be separated from ordinary tubercular disease, of which it is a mere complication or concomitant, its peculiarity being rather one of progress and development than of nature; while Dr. Greenhow regards it as differing from pulmonary consumption in its origin, progress, and issue (see discussion on Fibroid Phthisis in the Clinical Society of London, *Lancet*, vol. i, 1868).

Dr. H. C. Bastian, who has investigated the histological affinity between tubercle and fibroid degeneration, thinks that under the microscope the gray granulations of Bayle are indistinguishable from those met with in the early stages of pulmonary sclerosis. But while there is a histological identity in these two products, there is no difficulty in distinguishing between the two; they are distinct in their etiology, their anatomical characters and distribution, and in their termination. (1.) Tubercle is the anatomical index of a serious constitutional disorder, while fibroid change seems, in a majority of cases, to be mere local change, and even in those instances where it is connected with a peculiar diathesis, and shows itself in various organs of the body at the same time, that diathesis is not tubercular. (2.) Miliary tubercle has a strong tendency to undergo fatty degeneration and disintegration, which, beginning in the more internal elements of each of the component knots, has a peripheral direction, involving the whole mass; while in fibroid change the fibro-nuclear structure has only a temporary duration, and is gradually replaced by a more purely fibre-tissue with coincident lessening of bulk in the organ. (3.) Miliary tubercle is in rounded masses, rarely greater than a hemp-seed in size, and each of these apparently formed from separate centres, so that on section the elements of each knot are more or less concentrically arranged around several centres. This concentric arrangement is frequently absent in fibroid change, and never so well marked as in the gray

granulations. When this fibroid substitution begins, the new tissue oftentimes proceeds continuously in different directions, and has no tendency to produce rounded masses like tubercle. Fibroid change may, however, be coexistent with tubercle, and then it is conservative in its nature and antagonistic to the destructive action of the primary products of disease, i. e., tubercle; while existing alone it is more frequently aggressive, and may destroy a whole organ (*Brit. Med. Jour.*, vol. ii, 1867).

According to Dr. Andrew Clarke the disease is a local expression of a constitutional affection, as rheumatism, syphilis, chronic alcoholism.

In studying the pathogeny of this disorder, the tendency to hyperplasia of the connective tissue of the lung in old persons, should not be overlooked. Dr. Stokes speaks of it as senile phthisis; Maclachlan clearly describes it, without recognizing its true character, as does also Dr. J. Pollock; and the many observations of cicatrices and puckerings, and cretaceous matter found in the lungs of the aged inmates of the Salpêtrière Hospital, and brought forward by Rogée and others as evidence of the curability of consumption, were unquestionably instances of natural senile degeneration of lung-tissue. The not unfrequent occurrence of pulmonary sclerosis in chronic alcoholism, a disorder in which the pathological changes belonging to old age are discounted (see page 432 of this volume), is another point of pathogenetic significance.

From a study of the pathogeny and clinical history of this disorder, there is reason to believe that its genesis is to be looked for in a peculiar and distinct diathesis, which may provisionally be called the *fibroid*, and which is independent of, and not allied to, the scrofulous or tuberculous constitution; and that there is generally a previous history of chronic catarrhus affection, along with excessive spirit-drinking.

**Diagnosis.**—The physical signs of fibroid degeneration of the lung are so like those of pulmonary tuberculosis, and have so constantly the same site, that a differential diagnosis based alone upon them is often very difficult. It is rather by induction, a careful study of the general symptoms, the habits and physical development of the patient, that a correct diagnosis can be made. The absence of all indications of intestinal disease, as well as of any hereditary tendency, the build, and a history of previous good health and strong constitution, the slight loss of body-weight in the early stage, and the absence of continuous elevation of temperature, as well as habits of intemperance, or spirit-drinking, should be noted. Pulmonary tuberculosis is most common between twenty and thirty, whilst fibroid degeneration is most common after thirty; females are most liable to the former, and males to the latter.

**Prognosis.**—Dr. Andrew Clarke, of the London Hospital, studied the natural history of this disorder, in 13 cases out of 65, by leaving them without any treatment (*The Lancet*, vol. ii, 1866). All symptoms and signs disappeared within five months in 4; at the end of two years they remained little altered in 6; and in 3 they went on to softening and fatal destruction of the lung, within a little more than a year.

**Treatment.**—Sclerosis of the lung is to a certain extent amenable to treatment. Under favorable circumstances some cases can be cured, and many brought to a comparative state of health. The observations of Dr. Clarke show that art is of real value in controlling and even removing the deposit. In 52 cases he made a comparative trial of three methods of treatment: (1) 16 were treated by bringing the system slowly under the effects of mercury; (2) 17 were treated by acids and tonics; and (3) 19 by alkalies and tonics. His conclusions are, that: (1) the mercurial treatment is by far the worst; (2) the alkaline tonic decidedly the best. The general



health should be raised to, and maintained at, the highest possible point; thorough moulting of the tissues must be promoted; and the physiological conditions favorable to active absorption and excretion favored. The diet should consist of meat, eggs, milk, bread, and succulent vegetables, and from three to four pints of fluid, of which one should be of some malt liquor, ought to be drank during the day. The function of the skin must be quickened by tepid and cold sponging, baths, and friction; the clothing should be warm; daily exercise in the open air taken; and whatever may bring on acute catarrhus attacks avoided. Cod-liver oil, arsenic, quinine, iron, muriate of ammonia, iodide of potassium, chlorate of potash, and bicarbonate of soda may be given; and atomized weak solutions of the chlorate or nitrate or bicarbonate of potash, or muriate of ammonia inhaled twice a day.

In a case Dr. Fuller saw with Dr. C. J. B. Williams, and which he had an opportunity of watching during three years, all trace of the disease was entirely removed at the end of that time, by a treatment of cod-liver oil, tonics, alkalies, daily oleaginous inunctions, ioduretted and counter-irritant applications to the chest, a generous diet, proper exercise, and fresh air, aided by a twelvemonth's residence in Egypt (*On Diseases of the Lungs, &c.*, Am. reprint, 1867, p. 260).]

### PHTHISIS.

LATIN Eq., *Phthisis*; FRENCH Eq., *Tuberculeux*; GERMAN Eq., *Phthisis*.—Syn., *Schwindsucht*; ITALIAN Eq., *Tisi cronica polmonale*.

**Definition.**—*The growth or exudation of a peculiar material, in the form of a tubercle, which undergoes various changes in the lungs, associated with the constitutional phenomena of scrofula.*

**Pathology.**—The nature and treatment of this disease, commonly known as *pulmonary consumption* or *phthisis pulmonalis*, have been already fully described, from p. 213 to p. 252, and it is only with reference to its diagnosis in the lungs by general symptoms and local physical signs that it requires notice here.

Tubercles may be developed in a slow and insidious manner: (1.) In the form of the granulations described (p. 217, *et seq.*), and confined to one portion of the lung, where they gradually increase in size and number; or, (2.) They suddenly and rapidly increase, and are found in many parts, of a uniform size, generally called miliary tubercles, which are regularly distributed through the pulmonary tissue. They present different stages of retrograde metamorphosis at different parts of the lung. The recent gray tubercles are soft and gelatinous, those of older date are firm; and when the tissues are extensively invaded, they are softened and infiltrated with a thick serous effusion. The course of such cases is generally acute, and hence "acute tuberculosis" as a form of scrofula. In acute phthisis they may reach the size of a pea in three or four weeks (LOUIS); and when subjects in a state of scrofulous cachexia are exposed to violent irritation of the lungs, these granulations grow so rapidly and in such numbers throughout the lungs as to give rise to most alarming dyspnoea (SIR JAMES CLARK). Tubercle also occurs—(3.) As an *infiltration of the tissues* by an albuminous fluid of a thick synovial-like character, which gradually degenerates into



a firm grayish-red granular softened mass, containing portions of tissue within it. This infiltration was first described by Dr. Baillie in his *Morbid Anatomy*. "In cutting into the lungs," he writes, "a considerable portion of their structure sometimes appears to be changed into a whitish soft matter, somewhat intermediate between a solid and a fluid, like a scrofulous gland just beginning to suppurate. This appearance is, I believe, produced by scrofulous matter being deposited in the cellular [areolar or parenchymatous] substance of a certain portion of the lungs, and advancing toward suppuration. It seems to be the same matter with that of tubercle, but only diffused uniformly over a considerable portion of the lungs, while the tubercle is circumscribed." According to the pathology of the present day, as now described, this infiltration of *scrofulous* fluid *tuberculizes*, and the *tubercles* form themselves in the first instance in the cavities of the air-vesicles.

It has been much discussed (and still is discussed), in which texture *tubercle*-matter is first deposited, whether fluid or solid, and what is the cause of the deposition or growth. The deposit in the lungs is the one most frequently described; and while we have had some most minute and elaborate descriptions in recent numbers of the *Medico-Chirurgical Review*, it appears they do no more than confirm the account which Schroeder Van der Kolk gave forty-two years ago (1826), when he fixed the seat of tubercular *deposit* in the extremities of the bronchial tubes, or in what are named the pulmonic air-vesicles. Similar views were adopted by Carswell and Andral. Dr. Sieveking has since confirmed the observation by his own researches; Schroeder Van der Kolk has again elaborately described the more minute details as to how the process takes place; and Dr. Radclyffe Hall gives a similarly minute description. One form of tubercle appears in the air-vesicles in the first instance as a degeneration of the previously existing normal epithelium; shedding off this, and continuous replacement and degeneration, till the whole of the interior of the air-vesicle is filled with tubercle. Subsequently the pulmonic fibres become inclosed and separated by the morbid growth and exudation, and free nuclei and granules are formed between and amongst them. According to Virchow, such epithelium becomes "cloudy," and the cells degenerate, and the process of tuberculization advances in the cavity of the air-vesicle. This is also consistent with the experience of Hasse, who is of opinion that the tuberculous matter is first deposited on the walls of the vesicles, and gradually presses towards their middle the epithelium cells, or other contents of the air-vesicle, and then incloses the whole as a kind of soft and sometimes dark-colored nucleus. Another fact is of interest in relation to this point. In a stone-mason who died of phthisis an earthy nucleus was found at the centre of many of the pulmonary tubercles. This earthy nucleus turned out to be of precisely the same character as the stone of Craigleith quarry, where the man had been employed (Home, *Edin. Med. and Surg. Jour.*, January, 1838).

The question of epithelium in air-cells is still the subject of discussion. Its existence is disputed by Rainy, Todd, and Bowman,

Mandl, Ecker, Deichler, Zenker, Henle, and Munk; but its existence is maintained by Kölliker, Radclyffe Hall, Williams, and Hasse. It is also asserted by Eberth that in the lungs of calves and pigs there is a very delicate unbroken epithelium (Virchow's *Archiv.*, 1862, p. 503, where figures are given). By preparing healthy human lungs from young subjects by boiling the lungs in dilute acetic acid I believe I have succeeded in demonstrating the existence of a delicate film of pavement epithelium lining the air-cells.

With regard to the origin of pulmonary miliary tubercle, an important observation is made by Deichler, which would indicate the occasional origin of tubercle in another way. He found when a section was made through the centre of one of the tubercle-masses, that it inclosed a little stem, knuckle, or portion of a minute pulmonary artery, about one-twentieth of a line in size. This knuckle or loop of artery was surrounded by tubercle. The walls soften by the new formation of cells; and the more the cells grow, the larger the tubercle-mass becomes, till it is finally lost in the larger knots. It is not improbable that the tubercle-knots are formed owing to a diseased state of the membranes of the stems of the bloodvessels. Evidence of such disease may be found in cases of *ectasis* or aneurismal distension of the walls of the pulmonary capillaries, as seen in the air-cells. Buhl and Zenker have described such cases (Virchow's *Archiv.*, 1862, p. 183). Bearing upon this point, I lately dissected a soldier who died suddenly by hemorrhage from the lungs. On opening into one of the tubercle-cavities, it was found filled with coagulated blood; and projecting from a spot on the wall of this pulmonary cavity was a round tumor about the size of a walnut. This tumor had ruptured, and the rupture held a coagulum of blood. The tumor was found to be an *ectasis* or aneurismal dilatation of the pulmonary artery; and several other tumors of a similar nature, but of variable and much smaller sizes, existed in other cavities in the lungs, projecting from the pulmonary artery. They were proved to be continuous with this vessel—(1.) By the injection of spirits into them, and their distension thereby through the pulmonary artery as it left the heart; and (2.) By microscopic examination, which showed a delicate epithelial lining to these tumors, continuous and similar to that in the artery. The preparation is preserved in the museum of the Army Medical School at Netley (see also *Lancet*, Feb. 6, 1841; and referred to in *Prov. Med. Association, Trans.*, vol. x, p. 65).

It may shortly be summed up, with regard to tubercle in the lungs (as Gluge long ago showed), that the ultimate pulmonic vesicles are often involved in serofulous infiltration, giving rise to one form of isolated tubercle (*tubercula granulata*), confined, perhaps, to a dozen alveoli, the principal part of the morbid growth being in the alveoli.

Another form of isolated tubercle in the lungs occurs as an interstitial growth, situated in those points where a certain amount of connective tissue exists. Its occurrence is in the form of little knots, or separate grains massed together, but always in their early

stage recognized as bright sparkling spots like sand (SIBSON),—the usual *miliary tubercle*.

The seat of tubercle in the lungs may therefore be the mucous membrane of the bronchial tubes entering the alveoli, the walls of the alveoli themselves, the connective-tissue of the lung, the walls of the pulmonary artery, or the surface of the pleura.

Cases are rare in which the occurrence of tubercle is not at first latent in the lung; and from the numerous cicatrices that are found after death at the apices of the lungs, it is exceedingly questionable whether any person is carried off by a first attack. Tuberculous disease of the lungs in early life frequently heals, although it generally returns (sometimes at an advanced age), and ultimately proves fatal (SIR JAMES CLARK). There are few cases, also, writes Dr. Bennett, in which the destructive and ulcerative progress of scrofulous inflammation is uniform. It is continually being checked, and for a time slumbers; and even in the worst specimens of tubercular lungs numerous cicatrices and evidences of attempts to heal may be recognized; but as one portion cicatrizes another becomes the seat of further tuberculous growth. Cicatrices of healed tubercles present different appearances, according as the cavities from which they were formed have been superficial or deep-seated. When superficial, the pleuræ are more or less adherent and thickened, frequently thus forming an external boundary to the cavity. When the walls of the cavern contract, the pleural surface of the lung is drawn inwards, and thus the irregular puckerings on its surface are produced. Occasionally no traces of scrofulous matter are discovered either within or in the vicinity of these cicatrices; but more generally the contraction and puckering of parenchyma occur round tubercle which has undergone various transformations, and sometimes a cyst incloses the mass. The inclosed growth may be found to have undergone any of the processes of transformation already noticed (p. 217, *et seq.*); and the cretaceous or calcareous concretions may remain an indefinite time in the parenchymatous substance of the lungs, or they may be evacuated through the bronchi with the sputa. Thus masses of tubercle are sometimes absorbed or thrown off, and the evidence of this may be summed up under the following points: (1.) Small puckered cicatrices with loss of substance, after subsidence of the systemic disturbance characteristic of pulmonary phthisis. Strumous growths in the lungs of children seem capable of absorption without undergoing any change such as cretification or softening. Most frequently it seems in them to affect mainly the small lymphatic glands along the outer walls of the air-tubes. (2.) The conversion of tubercles into cretaceous or horny masses, which are either expectorated or remain latent. (3.) Isolation of the growth by so-called plastic material; adhesion and thickening of the pleura; adhesions and lymph thickenings round cavities, analogous to cysts round clots of blood or musket-balls. That such appearances are really evidences of arrested tubercles is rendered apparent by the following facts:

“1. A form of indurated and circumscribed tubercle is frequently met

with gritty to the feel, which, on being dried, closely resembles cretaceous concretions.

“2. These concretions are found exactly in the same situations as tubercle—most commonly in the apex, and in both lungs. They frequently also occur in the bronchial, mesenteric, and other lymphatic glands, and in the psoas muscle, or other textures which have been the seat of tuberculous growths or scrofulous abscesses.

“3. When a lung is the seat of tubercular infiltration throughout, recent tubercle generally occupies the inferior portion, and older tubercle, and perhaps cancer, the superior, whilst cretaceous and calcareous concretions will be found at the apex.

“4. A comparison of the opposite lung will frequently show that whilst on one side there is fine encysted tubercle, partly transformed into cretaceous matter, on the other the transformation is perfect, and has occasionally even passed into a calcareous substance of stony hardness.

“5. Seeing that, according to the observations of Dr. W. T. Gairdner, cicatrices also may result from bronchial abscesses, the seat of cicatrices in the lungs may vary considerably” (BENNETT).

In the words of Carswell, “pathological anatomy has perhaps never afforded more conclusive evidence in proof of the curability of a disease than it has in that of tubercular phthisis.” Pathology, therefore, teaches most distinctly that the aim of the physician must be to correct the constitutional tendency to the further occurrence of tubercle, or even to arrest, if possible, at its origin, the development of the constitutional state which leads to such growths. The most eminent physicians are agreed that it is peculiarly a disorder of childhood and youth, “when nutrition is directed to building up the tissues of the body,” that such persons are frequently attacked with symptoms of phthisis, which under proper treatment cease, “and years elapse before there is any renewal of the disease, and that were advantage taken of the intervening period to correct the constitutional cachexia, the cure might prove complete.” During remissions of the systemic disease, there is toleration of the lesions; but the remission ceases with renewal and extension of the local lesions.

The result of the course followed by tuberculosis, when observed in patients under treatment in hospital, may be arranged, according to results obtained by Dr. Walshe, under the following heads:

1. All the symptoms were removed, and the physical signs reduced to a passive condition in about  $4\frac{1}{2}$  per cent. of the cases admitted, without reference to the stage of the disease, or to the severity of the primary or secondary morbid changes.

2. If the persistence of some active physical signs are disregarded, such as a continuance of cavernous rhonchus, all the general symptoms disappeared in nearly 8 per cent.; and complete removal of all the symptoms was more frequently effected in the *male* than in the *female*.

3. More than half the cases of phthisis undergo temporary stages of improvement more or less permanent; and the time the disease has existed, rather than the stage the disease has reached, is an important element in calculating the probable benefit a patient may derive from treatment in hospital.

“In a given mass of cases the chances of favorable influence from sojourn in the hospital (at Brompton) will be greater, in a certain (undetermined) ratio, as the duration of the disease previous to admission has been greater; in other words, natural tendency to a slow course is a more important element of success in the treatment of the disease than the fact of that treatment having been undertaken at an early period” (*Med. Chir. Review*, Jan., 1849).

There are certain circumstances which promote the chances of a remission, namely, (1.) Originally good constitution; (2.) Non-inheritance of scrofula; (3.) Direct inheritance of longevity; (4.) Limited extent of local lesion; (5.) Integrity of other organs, unimpaired digestion, absence of fever, vigorous nervous system, quiet pulse; (6.) Influence of age—the period when the young body is making its growth is the period of greater danger. The tolerance of lesions and the periods of remission are less during the growing age, and are increased just when the body has completed its process of increase.

PROVISIONAL ARRANGEMENT OF THE VARIETIES OF CHRONIC PULMONARY PHTHISIS (DR. ANDREW CLARK).

Name.	Chief Anatomical Characters.
1. Tubercular, granular, or specific phthisis.	The true gray granulation. Pigmentary tubercle. Fibrous tubercle. Cellular tubercle?
2. Scrofulous or epithelial phthisis.	Primitive yellow tubercle; accumulation, cheesy degeneration, and disintegration of epithelium-like cells.
3. Catarrhal or bronchial phthisis.	Ulceration of bronchi, with adjacent fibroid and cellular deposits, and cheesy degeneration of the same.
4. Pneumonic phthisis.	Disintegration of recent or old deposits occurring in vesicular, lobular, or lobar pneumonia, primary or secondary, common or scrofulous.
5. Fibrous phthisis (cirrhosis, chronic, or interstitial pneumonia).	Fibroid deposits, with cheesy degeneration of imprisoned portions of lung, due to (a.) Mechanical irritation (as in grinders, masons, miners, &c.); (b.) Rheumatic inflammation of interlobular tissue; (c.) Chronic pleurisy; (d.) Constitutional states, as in granular kidney and liver.
6. Amyloid phthisis.	Circumscribed or diffuse cellular formations infiltrated with amyloid material.
7. Syphilitic phthisis.	Cheesy disintegration of nodules of nucleo-fibrous tissue, and diffuse infiltrations of the same.
8. Hemorrhagic phthisis.	Cheesy degeneration and disintegration of nodules of extravasated blood.
9. Embolic phthisis, including pyæmic deposits and suppurations.	Cheesy degeneration and disintegration of gray or yellow deposits, arising, directly or indirectly, from pulmonary emboli coming from the liver, lymphatics, or veins.

**Symptoms of Tuberculosis of the Lungs, or of Phthisis.**—These correspond with the intensity of the expression of the constitutional state; they vary also with the extent of the local growth or exuda-



tion in the lung, and with the condition in which the tubercles exist, and the changes they undergo. As a general rule, it may be stated that in the ordinary chronic form the presence of tubercular matter in the substance of the lungs, whether in its semi-transparent and crude, or in its softened state, does not cause much pain to the patient beyond dull, aching, "flying" pains about the collar-bones, or under one or both shoulder-blades, occasional soreness of the throat, and tightness across the upper part of the chest.

The greater number of cases of chronic pulmonary tuberculosis are indicated in the commencement with some slight cough of a dry hacking character, most frequently induced on rising in the morning and going to bed at night. The cough at first seems simply intended to clear the throat, the irritation being mainly referred to the pharynx, which is often red, rough, and coated with tenacious mucus. As the cough becomes more fully expressed a scanty expectoration occurs, of ropy or glairy mucus, hardly discolored, or only slightly streaked or stained by a trace of blood. Dyspepsia, sick headache, biliousness, and loss of appetite prevail. The patient is feeble, easily fatigued, feels unequal to his usual work, has burning heat of the soles of the feet at night, and some perspiration in the morning; his nights are restless, so that he rises in the morning weary and unrefreshed by sleep. He is irritable, and often depressed in spirits, his appetite capricious, with dyspepsia—a most constant, important, and early symptom,—and he is convinced of a sensible loss of flesh. His muscles become flabby, the countenance pale, the conjunctiva becomes pearly-white, and the pupil of the eye dilated. These symptoms are accompanied by a permanently accelerated pulse, from 90 to 140. The cough after a time begins to recur at intervals during the day, and especially after the least exertion. He is now sensibly aware of being "short-winded," so that active exercise exhausts him, and he must rest at intervals if he walks fast or goes up a stair. The number of respirations per minute is now increased; the pulse accelerated, especially towards evening, and deficient in force. Febrile paroxysms are persistent, and of daily occurrence; and elevation of temperature, as measured by the thermometer, is a constant phenomenon capable of being appreciated for many weeks before physical signs become decided (DAVY, RINGER). There is also correspondence between the elevation of temperature and the activity of the growth of tubercles in the lungs; such that when it goes on rapidly the elevation of temperature is high; and *vice versa*. This elevation of temperature is persistent, varying from 103° to 104° Fahr., for several weeks before diminished weight or physical signs indicate the undoubted presence of tubercle (RINGER).

[The midday temperature in phthisis usually ranges from 99.2° to 102°, though sometimes it reaches a higher point. A persistent body-heat of above 99° Fahr. in a strumous person generally points to some active mischief. In such cases evening temperatures should be taken, for if there is anything serious going on the body-heat at this time would be decidedly elevated.]

This stage or state of things may last a few weeks or a few months; and even the patient often revives, and seems to an unpractised eye, for a short time to have recovered his good general health, were it not for a sense of weakness and undue exhaustion after such exercise as he has been daily accustomed to.

The disease, however, silently proceeds, and all the preceding symptoms are gradually but sensibly aggravated. A large amount of tubercle may have grown during the earlier stage; and after a time the intervening portions of lung become so congested that bronchial irritation, with intercurrent attacks of pneumonia, are of frequent occurrence. Rapid softening of the tubercles takes place in those in whom the constitutional disease is intense; and with the continued discharge of pus from the lungs, hectic fever becomes permanently established, and the sweat from the head and chest towards morning is often so profuse that the patient lies bathed in perspiration. The cough is more distressing, the sputa purulent, and the pulse more frequent, perhaps from 90 to 110. The emaciation, consequently, is now well marked and decided. The duration of this the *second stage* is very indefinite; a few weeks to many months may end in a fatal issue. During its progress, however, the disease occasionally intermits and becomes latent, so that often there is for a time a marked amendment, and the patient regains some strength—a most important interruption, to be sedulously taken advantage of, for the purpose of promoting the tendency to cure. But fresh tubercles are apt to grow during these intervals, so that the lung-tissue is still further encroached upon by the newly-formed growth, fresh local irritation is set up, all the general symptoms are aggravated, sickness and rejection of food are now often excited—the expectoration becoming more profuse, of a purulent character, and often streaked with blood.

The third and last stage of this eventful constitutional disease is that in which, after the tubercle has softened, a cavity forms. In this stage all the preceding symptoms attain their highest degree of intensity: the hectic is followed by cold clammy sweats, and great exhaustion towards noon and again at night; the appetite is lost; a colliquative diarrhœa may supervene from tuberculous ulceration of the intestines; the sputa are often pure pus, as from an abscess, but at length become little more than a rusty sanguinous mucus; the pulse rapidly increases to 110 or 150; the emaciation is excessive; the hair falls off; catamenia cease; pleurisy or pneumothorax may supervene; ulceration of the larynx; and amidst this general wreck of material existence, the Mind is often firm, collected, and even hopeful to the last.

As soon as the cavity which forms is in free communication with the bronchi, the cough is often greatly relieved. The duration of this stage is generally shorter than the former; but still, notwithstanding the existence of one or more cavities, it often lasts many months. As the fatal end approaches, the appetite fails more completely; sleep can only be obtained by narcotics; the integuments on which the patient lies are apt to become inflamed, sore, and even to die from the constant pressure; œdema of the feet and ankles

sets in; and with the approach of death, suffocative dyspnoea may render the death-struggle extremely painful. In other cases a wandering delirium enshrouds the Mind, or coma supervenes, and the prolonged and weary illness is tranquilly ended by the gentle and welcome approach of death.

Such is a short outline of the course and phenomena of this destructive disease. It sometimes terminates life within a few weeks, or extends over six or eight months, while it occasionally lasts several years, with marked intermissions in its progress.

The following is a short analysis of the principal local, constitutional, and stethoscopic signs of this remarkable affection:

Affections of the bronchial membrane and mucous membranes of the pharynx and larynx are certainly the most frequent concomitant symptoms of phthisis; but the part of the bronchial membrane affected is not always the same. Most commonly the mucous membrane of the smaller bronchial tubes is first affected; then that of the larger ones, the disease gradually ascending till it often ends in a *chronic laryngitis*, with a partial or total loss of voice. In a few cases, however, this order is inverted, and almost the first symptom is a *laryngitis* with *hoarseness*, partial loss of voice, and constriction of the throat; after which the disease descends to the larger and then to the smaller bronchi, when the patient begins to expectorate; his pulse becomes hurried; he loses flesh; and all the unerring symptoms of phthisis are established; while *pleurisy*, *pneumothorax*, *pneumonia*, *bronchitis*, and *hæmoptysis* are the more important intercurrent complications; while amongst the more chronic are *ulcerations of the bowels*, *serofulous inflammation of the epiglottis, larynx, trachea, bronchial glands, or tuberculous meningitis*.

The *expectoration* which takes place in phthisis from the bronchial membrane is usually purulent, the pus thrown up in the early stages being for the most part of good quality, and formed into "sputa," sometimes sinking, and sometimes swimming in water. It may be either of a sweet, insipid, or saltish taste, as experienced by the patient. As the disease advances, pus is often expectorated pure, as from an abscess, and without any separation into sputa, and is sometimes mixed with particles of a curdy substance.

In the last stages of consumption it is often of a rusty green, a dirty sanies, or a rusty muciform serosity. The quantity expectorated varies greatly; sometimes only a few sputa, or not more than half an ounce in the twenty-four hours, and then perhaps more than a pint in the same period, so that in a few weeks the patient has often expectorated more than his own weight of pus. If a small abscess has burst into the bronchi, the sputa, though somewhat increased in quantity, are hardly changed in character; but if the abscess be large, the quantity thrown up is proportionally great.

*Hæmoptysis*.—Hemorrhage may precede, or be contemporaneous with, or succeed to the bronchial affection. If it precedes, the patient being, as he imagines, in excellent health, is suddenly seized with *hæmoptysis*, followed perhaps by cough. This attack subsides, but a second and third follow, till the condition of pulmonary tuberculosis becomes undoubted. In this respect, however, the

hæmoptysis of phthisis must be distinguished from the recurrent and minute hæmoptysis of an aneurism opening into the air-passages. Hæmoptysis more commonly, however, occurs later in pulmonary phthisis, increasing the debility, aggravating the symptoms, and hastening the fatal issue. The quantity of blood lost is sometimes only enough to streak the sputa, at others a few teaspoonfuls, but in some instances so profuse as to amount to one, two, or more pints. In the still more advanced stages, though cases occur in which the quantity of blood thrown up is very great; yet more usually it is trifling, and more resembles a bloody sanies than pure blood.

The *dyspnœa* is generally great in phthisis, the patient being unable to make any active exertion, or even to read a few lines without pausing. The dyspnœa, however, is not always proportioned to the amount of mischief; for there are instances in which the respiration has been performed with facility, even when two-thirds of the lungs have been in a state of tuberculoma. It is doubtful whether adhesions, unless very extensive, greatly affect the respiration. Should effusion of serum, however, or of pus, from the bursting of an abscess, have taken place into the cavity of the chest, then the respiration is greatly impaired. The most common situation of the fistulous opening, caused by the bursting of an abscess into the chest, is the summit of the lung, or a little below the clavicle. It is usually very small, hidden by the lung, or so surrounded by adhesions that it is difficult to discover it.

*The Stomach* is more or less diseased in three-fifths of the cases of phthisis; yet it so seldom gives rise to any well-marked symptom that for the most part any lesion there may be said to be latent. In the worst cases the symptoms are only a capricious appetite, indigestion, some pain in the epigastrium, and vomiting after coughing. The indigestion is peculiar, as already mentioned at pp. 229, 230. With reference to fat and fat meat, the dislike of consumptive patients to such food is unquestionable; so that the fat and the lean parts have to be adjusted to the likings of the patients, and it is generally of no use giving to them any but the leanest parts of the meat. They are partial also to fish, and especially to soles.

*The Intestinal Canal* is at least as frequently affected as the stomach in phthisis; but in general the abdomen is without pain. The only marked circumstance is, that the stools are more copious than in health, the body being unable to appropriate the accustomed quantity of nutriment prepared by the stomach. As the disease advances, the patient often suffers from irritable bowels, or from diarrhœa alternating with constipation; while, towards the close of the disease, the diarrhœa often becomes colliquative, hastening the fatal result. In some few instances the peritoneum ruptures, and the patient dies of *peritonitis*; while in a somewhat larger number *ascites* is a common occurrence.

*The Liver* undergoes fatty degeneration in about one-third of the cases, and so remarkable a lesion might be expected to give rise to some particular symptoms; but this is not the case. It may occasionally be felt somewhat enlarged, but neither pain, nor altered

state of the secretions, or other circumstance, denotes its diseased condition.

*The emaciation* so remarkable in this disease is common to nearly all the tissues of the body, as the adipose tissue, the muscles, the bones, and even the intestines and skin are thinned. This emaciation often commences before the disease can be said to be well established, so that the patient has often lost one or two stones in weight before he applies for medical advice. In the more advanced stages emaciation progresses in a peculiar manner, the patient losing perhaps three pounds in one week, and gaining two pounds or more in the next; and this alternation of gain and loss goes on for many weeks or months, but generally leaving a balance against the patient. Towards the close of life the loss greatly surpasses the gain, and occasionally amounts to four, five, six, and seven pounds in a week. The total loss the patient sustains is perhaps from one-third to half his whole weight.

*The Mind*, though not capable of continued exertion, is generally perfect throughout the disease, or only wanders during the few last days of existence. It is seldom the patient dreads the future or despairs of the present, for nature, however threatening his symptoms, has imparted a singular buoyancy to his hopes, and he always says he is better; would be quite well but for his cough; feels able to take a long walk; and sometimes (in expectation merely) enjoys his meals;—yet, with all this, he may faint if he attempts to cross the room, or nauseate when his food is brought to him.

In the aged, it has been shown by Dr. MacLachlan (*op. cit.* p. 333), that “consumption may exist independent of tubercular development, and that tubercles are not the essential anatomical character of senile phthisis: and that the most extensive destruction of the lungs not unfrequently occurs, accompanied with the usual symptoms of this disease, without a trace of tubercle.” It is usually a sequence of chronic bronchitis, terminating in indolent inflammation and partial induration of the lung. These indurated parts at last break down, leaving caverns and burrowing sinuses, which are characterized by a dark and sloughy aspect of their inner surface, and by the absence of any membranous lining (ARMSTRONG, GRAVES, STOKES, MACLACHLAN). It constitutes the *ulcerous phthisis* of Bayle, the *pneumonic phthisis* of Addison; and, in the experience of Dr. MacLachlan, it “is far from unfrequent in aged persons, the victims of intemperance or of chronic visceral disease.” He believes it to be a truly scrofulous form of inflammation, disorganizing the lung. In *senile tubercular consumption* the tubercular growths are generally confined to the lungs—commonly limited to one lung only; and to the upper and back portions—tubercles existing in the very apex or apices only, the remainder of the lung being healthy (MACLACHLAN, *op. cit.*, p. 334). [See Sclerosis of the Lung by Editor.]

Such are the general symptoms of phthisis. A physical examination of the chest will yield many interesting additions to our means of diagnosis, and enable us to determine, not only that the lung is diseased, but also the particular part, and the state in which it may be. Thus the discoveries of Laennec and Avenbrugger have



rendered the diagnosis of *tuberculosis* of these organs almost as perfect as though the disease was exposed to sight. Nevertheless, there is no *single sign* by which the existence of tubercles in the lungs is clearly indicated; therefore the general symptoms must invariably be judged of in combination and comparison with the information obtained by physical signs.

Not a little has been written as to the possibility of detecting what has been called a "pretubercular stage of phthisis" (E. SMITH and others). By this expression writers mean that there exists an abnormal physical condition of the lungs and of the body preceding the deposition of tuberculous matter in the lungs, which condition is capable of demonstration by certain signs or symptoms. Some physicians find that no local physical sign of disease is indicative of the existence of phthisis *before the growth of tubercle*; but the tendency to the tuberculous state is inferred from the presence of a series of cachectic symptoms, which from experience are frequently found to terminate in tuberculous disease of the lungs. Others believe not only that such symptoms are present, but that they can detect such physical signs as are believed to indicate a peculiar local condition of the lung—a *stage anterior to the deposition of tubercle*. "The slightest subclavicular dull percussion-sound, with lessened vesicular murmur, less forcible and deep inspiration, and flattening of the apex of the lungs," are described as the physical signs of this so-called *pretubercular* state (MARKHAM). But, however much practitioners may accustom themselves to detect the physical signs of known pathological conditions, there are states of the body between cachexia and local lesions so nicely balanced that no definite local morbid state can be ascribed to them, and therefore no physical signs can be associated with such a negative position apart from those of health. The so-called "pretubercular stage of phthisis is undoubtedly an instance of this kind. The best auscultators have admitted that there are no distinct and infallible signs by which we can with certainty diagnose the early existence of tubercle; and it is also known that solitary growths of tubercle do not of themselves produce the slightest change in the percussion-sound of the lungs." On the contrary, most physicians believe that "the slightest subclavicular dull percussion-sound, the less forcible, deep, harsh, or tubular inspiration, with lessened vesicular murmur, prolonged expiration, increased vocal resonance, and flattening of the apex of the lungs, when combined with the well-known general symptoms, leave little doubt that the actual presence of tubercle is indicated." While, therefore, it is imperative on the student and practitioner to educate their ear to the utmost in the detection and appreciation of the finest thoracic murmurs, it is unjust, as already observed, to expect through the stethoscope more information than it is fitted to convey. A too exclusive study of physical signs, to the almost disregard of general symptoms, not only does injustice to the science of Medicine, but the lives of patients are endangered when treatment is solely founded on the former. It is by general symptoms, such as those which Todd, Clark, Bennett, Ancell, Hutchinson, and Ringer have so fully elucidated, that the practitioner will

be able to recognize a “pretubercular stage of phthisis,” and not by local physical phenomena alone. A very interesting illustration of this is afforded by some recent observations made by Dr. Sidney Ringer on the value of thermometric observation as indicative of a pretubercular stage of phthisis. He has been able to recognize a persistent elevation of temperature as the invariable precursor of the growth of tubercle in any organ. This persistent elevation of temperature existed for several weeks before diminished weight or other physical signs indicating the growth of tubercle in the lungs could be appreciated. After a certain time, however, growth of tubercle began to be apparent by physical signs in the apices of the lungs. His observations were made on patients in University College Hospital; and he kindly permitted me to read, in MS., a summary of his observations. (See also vol. i, p. 64.)

[The conclusions Dr. Ringer has arrived at from a more extensive series of observations, made by himself and others, of the value of the temperature of the body as a sign of tuberculosis, are embodied by him in the following propositions: (1.) There is probably a continuous elevation of the temperature of the body in all cases in which a deposition of tubercle is taking place in any of its organs. Of 24 cases, in 21 there was a continuous elevation of body-temperature, and in these 21 cases the continuous deposition of tubercle was ascertained during life by the physical signs, and proved after death by the post-mortem appearances. (2.) This elevation of temperature is probably due either to the general condition of the body (tuberculosis), or to the deposition of tubercle in its various organs (tuberculization). (3.) It is probably due to the state of the first, rather than to the second of these processes. (4.) The temperature may be taken as a measure of the amount of the tuberculosis and tuberculization, and any fluctuations in the temperature indicate corresponding fluctuations in the intensity of the disease. (5.) The temperature is a more accurate measure of the amount of tuberculosis and tuberculization than either the physical signs or rational symptoms. (6.) By means of increased body-temperature we are able to detect tuberculosis and tuberculization long before any physical sign is present, and when the symptoms are insufficient to justify such a diagnosis. (7.) By means of the body-temperature we are able to diagnosticate tuberculosis, even when throughout the course of the disorder there is no physical sign indicating the tuberculous deposition in any organ, and where the symptoms are insufficient to lead to a correct diagnosis. (8.) It is probably by means of the body-temperature that the abatement in the active general condition (tuberculosis) and the cessation of tubercular deposition (tuberculization) may be ascertained, and the conclusion reached that the persistence of the physical signs is due to obsolescent tubercles, and to chronic thickening of the lung-tissue between the deposits. Two cases (vii and viii) are given to sustain this proposition.\*]

**Physical Signs.**—In the following details a systematic examination of the patient is prescribed, after the method recommended by Dr. Fuller, otherwise the diagnosis is a matter of extreme uncertainty to those unpractised in conducting a physical examination

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\* [On the Temperature of the Body as a Means of Diagnosis in Phthisis and Tuberculosis. By Sydney Ringer, M.D., London, 1865.]

of the chest. The physical signs are obtained by—(1.) *Inspection* ; (2.) *Palpation* ; (3.) *Mensuration* ; (4.) *Percussion* ; (5.) *Auscultation* of the chest.

The physical signs of pulmonary tubercle, in the order of their relative frequency, may be stated as follows: *Dulness of percussion* (constant); *deficiency of respiratory murmur*; *bronchial voice*; *rough inspiration*; *expiration prolonged*; *mobility of chest-wall lessened*; *flattening of chest-wall*; *tubular quality of respiration*; *dry crepitation*; *wavy inspiratory sound*.

If the chest of a patient laboring under incipient phthisis be carefully examined, we may at first observe nothing remarkable unless the growth of tubercles shall be large in amount or confined to one side of the chest. Abnormally rapid respiratory movements indicate a large amount of tubercle; and there is decreased expansion of the chest in the *infra* and *supra*-clavicular regions on the affected side, or flattening of the chest-walls at the place affected; and if both sides are equally affected, this flattening is apt to escape attention. If the disease be further advanced, we find the patient emaciated, together with a singular immobility or incapacity of dilatation of that portion of the chest immediately below the clavicle, so that he breathes chiefly by his shoulders and diaphragm, and is unable to “fill the chest,” especially during forced inspiration. The respirations are now greatly increased in frequency. In the latter stages of the disease the respiratory movements are still more rapid. The clavicles appear peculiarly prominent, in consequence of the flattening, depression, or falling in of the *supra* and *infra*-clavicular regions. A fluctuating impulsive movement may be obvious in the upper intercostal spaces, generally on the left of the sternum, due to the action of the pulmonary artery or base of the heart. At a still more advanced stage, if an abscess has burst into the cavity of the pleura, and caused *pneumothorax*, the affected side is not only motionless, but distended, and as it were bulging out.

*Palpation* during the *early stage* may disclose deficient expansion in the *infra* or *supra*-clavicular regions; and if there is increased vocal fremitus at the apex of the lung, it betokens consolidation of the lung-tissue underneath.

[Palpation is a valuable aid in the diagnosis of tubercular deposition, the chest-wall over the diseased lung giving a peculiar sensation, which may be described as a want of elasticity to the ends of the fingers. The palm of the right hand should be laid upon the bare chest, beneath the clavicle, the ends of the fingers being upward towards the bone, and then a very gentle stroke from the wrist should be made on the wall, avoiding the production of all sound (see an interesting paper on this subject by Dr. G. N. Edwards, in *St. Bartholomew's Hospital Reports*, vol. ii, 1866).]

In the *advanced stages* of the disease, marked vocal fremitus betokens a large cavity, superficial, and in free communication with the air-passages. It may be rhonchial, or gurgling, or like fluctuation. If the lung has shrunk, the heart may be felt beating out of its proper place.

*Mensuration* with calipers, in the early stage may detect a slight diminution in the *antero-posterior diameter* of the *infra-clavicular* region, and a decrease in the local expansion movement; as the disease advances, the size of the side affected diminishes both in its *transverse* and *antero-posterior* diameter, probably due to atrophy and cicatricial like contraction of the lung, collapse of air-cells, or firm pleural adhesions.

*Percussion* during the earlier stages, so long as air penetrates the pulmonary tissue equally on both sides of the chest, does not convey any definite information. Dulness does not always follow even when considerable masses of tubercle exist at the apex, unless the masses are very superficial, when the sense of resistance to the fingers is also increased. But if a portion of healthy or emphysematous lung intervenes between the consolidated lung and the chest-walls, there will still be clear resonance over the affected part. Percussion should be practised during deep inspiration, and then during deep expiration. If tubercles exist, it will then be found that in percussion under a full inspiration, the resonance is *increased* on the affected side, but very *slightly* as compared with that on the healthy side; whereas, if percussion be made under a full expiration, the dulness will be far greater on the affected side than over the healthy lung (FULLER, pp. 42, 44, 365). It is differences rather than actual quality of sound on which an opinion must be based. In the more advanced stages percussion elicits evidence of dulness more intense and more widely spread. In the latter stages, where *vomicæ* exist, percussion will be absolutely dull if one or more small *vomicæ* are filled by purulent material, or surrounded by consolidated lung. If, however, the same cavities are empty and superficial, without thickening of the pleural membrane, the dulness may be slight, or the sound on percussion resonant, though shallow or amphoric. "Large empty *vomicæ*, with tense walls, yield an amphoric or cracked-pot resonance, . . . and, except in rare instances of enormous superficial *vomicæ*, with tense walls, it is almost impossible to judge of the size of a cavity by the results of percussion" (FULLER, *op. cit.*, p. 369).

A superficial empty cavity, with resilient walls, having free communication with the upper air-passages, yields a cracked-pot sound, especially when the mouth is open, and all obstacle to the egress of air removed.

*Auscultation* yields extremely variable results. Slight harshness of respiration is the first indication noticeable, with prolongation of the *expiratory* murmur, and "jerking irregularity of the respiratory sounds." When these phenomena are persistent, and confined to one side of the chest, they indicate tubercle, especially if the phenomena of "dry clicking" be added. These phenomena are earliest marked in the *supra-scapular*, *supra-clavicular*, and *infra-clavicular* regions. If the physical signs of bronchitis (namely, coarseness of respiration, with small bubbling râles and sonorous sibilant rhonchi) are persistent in these regions, and inaudible below the second intercostal space, and still more so when confined to one lung, their existence warrants the suspicion of tubercle. If

the disease is advanced, and the growth of tubercle involve bronchial tubes of considerable size, there is almost entire absence of vocal resonance and of respiratory murmur over the part affected, with the sounds of breathing exaggerated in the adjoining parts. If consolidation is extensive, the sounds of the heart and large vessels are transmitted to a greater extent than in health.

The diagnostic value of murmur in the pulmonary artery is variously estimated. It is often present in many phthisical patients at the second left sterno-costal articulation (FULLER, p. 366. See also Sieveking, in *Lancet*, Feb. 11, 1860).

[In eighty cases of incipient phthisis, observed by Dr. J. Pollock with great care, the relative frequency of the physical signs were as follows :

Physical Signs.	No. of Cases. 80
Mobility of chest-walls lessened, . . . . .	41
Flattening of chest-walls, . . . . .	38
Dulness on percussion, . . . . .	80
Deficiency of respiratory murmur, . . . . .	69
Rough inspiration, . . . . .	47
Expiration prolonged, . . . . .	47
Tubular quality of respiration, . . . . .	32
Bronchial voice, . . . . .	48
Dry crepitation, . . . . .	17
Wavy inspiratory sound, . . . . .	15 ]

As the disease advances into the second stage, auscultation indicates the presence of thin irregular-sized bubbling râles, from the passage of the inspired air through the softened and liquefied tubercles. It may also prove that coarse and hollow-sounding respiration exists over a more extensive surface than heretofore; or that the respiratory sounds are of a blowing character (bronchial breathing). In still more advanced conditions, with more or less empty cavities, the respiration is heard to be blowing or amphoric and metallic; and if the fluid contents of the cavity rise above the level of the permeable bronchi which lead into the cavity, large irregular bubbling râles or distinct gurgling may be heard. Well-marked pectoriloquy (from a mere whisper) is the form of vocal resonance most pathognomonic of pulmonary tuberculosis (FULLER, *l. c.*, pp. 363 to 370).

The physical signs denoting a few small scattered tubercles are, (a.) *Inspiration rough and dry, and its intensity increased*; (b.) *Expiration prolonged—compared with its normal intensity and duration, as eight to two*; (c.) *Bronchophony in rare cases* (POLLOCK).

Of crude tubercle in groups or masses the physical signs are,—*Pulmonary crumpling sounds*; *dry crackling rhonchus*; *sonorous sibilant rhonchus*, indicative of bronchial irritation; *inspiration rough and dry, intensity increased*; *the intensity and duration of expiration increased, and its quality blowing*; *slight bronchophony*; *diminished*



*local fremitus ; slight dulness, localized and distinct ; heart's sounds transmitted ; and in certain cases a subclavian murmur (POLLOCK).*

The following are the different modes in which phthisis pulmonalis makes its approach :

1. There is sometimes to be noticed a latent, masked, or occult form of scrofula—the real condition of the patient not being detected till the lungs are tuberculous to a considerable extent, unless recourse be had to careful thermometric observation, as described in the first volume, page 57 to 71. Although the general symptoms may be slight, this very fact ought at once to excite suspicion, especially when the general aspect or constitution of the patient denotes the tuberculous cachexia. The slight cough, the shortness of breathing, the frequent pulse, increased on the slightest exertion, the languor of the frame, and general chilliness of the body with persistent increase of temperature, morning perspirations, and progressive emaciation, betray the insidious way in which this form of consumption commences. Such a case must be closely watched, for the local symptoms often remain obscure, and friends do not always see the importance of the illness till too late. It is most usually young persons between sixteen, eighteen, and twenty-five years of age who are the victims. They begin to lose flesh, and are attacked with a short tickling cough, often regularly excited by undressing at bedtime, and again on getting out of bed in the morning. Such a cough is dry, or followed by only a small quantity of mucus. The cheeks are the seat of hot, uneasy flushings, while the feet are cold, and towards morning there is generally a little more moisture than usual on the surface of the body, especially about the head, neck, and breast. The pulse is either considerably quicker than usual, varying from 90 to 110 or 120 ; or if it is natural, it is very readily accelerated. The respiration is, in general, more frequent in a given time—usually from 24 to 28 in a minute—the inspiration being generally short, limited, and speedily checked, quickly succeeded by expiration ; and the patient cannot take a full or deep inspiration without uneasiness, and without inducing coughing (CRAIGIE).

2. There is another form of consumption, attended with a severe and sudden accession of febrile disturbance (to be measured by the thermometer), occurring in persons of a scrofulous diathesis. To this form Sir James Clark has given the name of “*febrile consumption*.” Pulmonary symptoms are not generally manifest in such cases in the first instance ; but what is commonly called biliousness more frequently prevails, or the case may present the symptoms of a common catarrh. Cough, however, generally soon appears, becomes urgent, and occurs often ; and hurried breathing is one of the most remarkable symptoms. The cough speedily becomes more frequent, accompanied by expectoration, at first colorless, or of a thin, transparent, bluish jelly-like appearance ; it subsequently assumes a yellowish or greenish hue, thick, opaque, dense, and puriform, and is occasionally streaked with blood. Fever continues unabated, and is out of all proportion to the other symptoms of pulmonary affection ; and thus the true character of the disease

may be overlooked. The pulse is seldom under 100, varying from 110 to 116, with some tension and sharpness in the beat. Gradually the fever assumes the hectic form, progressive wasting is established, and doubt can no longer be entertained regarding the nature of the case and the fatal issue. (See range of temperature in "Acute Tuberculosis.")

3. In another description of cases hæmoptysis is the first symptom that attracts attention and alarms the patient and his friends. It is followed by all those phenomena already noticed.

The cases of phthisis more usually rapid are such as arise from hereditary constitutional causes, or from the influence of exanthemata, especially measles, or of typhoid or other fevers. These run their course with implication of several organs at an early stage of the disease. In the more chronic forms of phthisis the lung in adults is the first seat of the disease, and other organs are secondarily affected. In appreciating the causes of phthisis these forms must be distinguished (Parkes's *Practical Hygiene*, [p. 463, 2d ed.])

[From the Second Medical Report (1863) of the Hospital for Consumption, Brompton, it appears that out of 1973 cases of phthisis, 275 had previously had rheumatism. In Dr. J. E. Pollock's own practice out of 67 cases of rheumatism in persons affected with tubercle, 49 had rheumatism before, and 18 after, phthisis was established (*l. c.*, p. 83). But out of a total of 4530 cases observed by him, rheumatism really preceded phthisis but in 49 cases, and taken with the 18 cases happening in the course of the disorder, it existed only in one and a half per cent. of his cases. In 1000 phthisical patients observed by Dr. Cotton, there were only 6 cases of rheumatism. (*Phthisis and the Stethoscope*, 3d ed., 1864.) These figures hardly justify Dr. Pollock's conclusion that "the two diatheses are closely allied."]

In the temperate zone, where the civilized inhabitants of the globe are located, it is calculated that *one-tenth* of the population die of this malady. Thus, in man, it is observed that in proportion as his habits of life are artificial, so is his tendency to scrofulous disease, especially of the lungs. This is strongly seen in the mining districts of Cornwall and Devonshire; for although those counties are considered among the most healthy portions of Great Britain, yet one-half of the whole number of the miners deprived of fresh air and light die of phthisis.

[Dr. Boëns-Boissau says that the strumous diathesis is almost universal among the coal-miners of Belgium, and that tubercular disease is frequent amongst their children, affecting the lungs, and still more often the brain and mesenteric glands.\*]

In connection with this statement a most significant fact, of great practical importance, has been brought to light by Dr. Walshe. He has shown that improvement in cases of pulmonary phthisis was effected by medical treatment in hospitals, in about 14 per

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\* [*Traité Pratique des Maladies des Houilleurs*. Bruxelles, 1862.]

100, more frequently in persons following "open air" and "medium" occupations than in those whose trades were "confined;" but that *death* or *deterioration* was as frequent as improvement in those who followed confined trades. Minute analysis, such as that which has been instituted by Dr. Walshe, of the numbers that die of phthisis in the different ranks and classes of life, is greatly to be desired in illustration of the remote causes of phthisis. The late Professor Coleman was of opinion that by confining the horse in a dark and dirty stable, and by feeding him on bad provender, and neglecting to clean him, he could produce phthisis on that animal at will; and similar causes will probably be found to produce similar results in man. When, however, we consider how many persons there are who carry cleanliness to excess, whose diet is most studied, and whose every exercise is directed to health, and who nevertheless die of phthisis, it is plain that more secret and hidden circumstances still remain to be discovered to account for the existence of scrofulous disease, especially of the lungs, in this country. The Reports of the Registrar-General show that, comparing the deaths from phthisis among the agriculturists and among the inhabitants of towns, the latter die in an increased ratio of 25 per cent. over the former; yet it is generally supposed that the dietary and general comforts of the townsman are greater than those of the countryman. The chances of improvement are 5 per cent. greater in persons who come from the country to a salubriously situated hospital in town than in townspeople (WALSHE). Among townsmen, also, it is determined that there are certain classes of men more predisposed to phthisis than others by the nature of their occupations. It has been observed to occur, for instance, in those workmen who suffer great vicissitudes of temperature, or who breathe an air loaded with particles of dust: as bakers, needle-grinders, stonemasons, quarrymen, cotton and wool carders, and bricklayers' laborers, and in this class of persons the disease has acquired the epithet of the "grinders' rot."

[Dust is undoubtedly a most deleterious agent, to which certain artisans are exposed, and would appear, from the investigations of Greenhow, Alison, Hannover, and others, to be a fertile cause of phthisis among those who are engaged in hackling flax, carding cotton, grinding steel; also workers in porcelain, makers of mother-of-pearl buttons and of mattresses, chaff-cutters, stonemasons, and saddlers, furriers, glovers (HANNOVER) who inhale the dust from animal tissues, and coal-miners. Dr. Greenhow shows that the potter's consumption is chiefly caused by portions of clay being allowed to be trampled into dust on the floors of the work-rooms. Few men who enter certain rooms in cotton factories ever live to 38 years; and out of 27 men in a flax factory, 23 had pulmonary disease (GREENHOW). Dr. Alison asserted, that in Edinburgh a stonemason scarcely ever reached 50 without becoming consumptive. The noxious influence of varnishes, turpentine, and drying oils, in developing phthisis, was long ago pointed out by Lombard (*Annales de Hygiène*, t. xi, 1834), and it is said to be very frequent among artisans who use solder, such as tinmen, coppersmiths, and goldsmiths.]

As far as regards their state, much has been founded upon it to show how irritant substances may induce the local growth. The mortality numerically from consumption is much higher than from any other disease in this country ; and amongst the class of society resorting to assurance offices the mortality seems to be about one-half of the whole.

The disease appears to be still more fatal to soldiers than civilians, producing nearly *one-half* of the whole mortality among the dragoon guards ; while in the foot guards it has hitherto been nearly double of what takes place in the dragoon guards in this country. The habits of a soldier's life, his liability to febrile attacks, and the frequency with which he contracts venereal diseases, have hitherto been very favorable to the induction of the more aggravated forms of phthisis. The history of phthisis in armies will at once show how materially the prevalence of such a disease influences the health, the wealth, and the military strength of a nation. In Prussia, phthisis caused 27 per cent. of the total mortality ; in Austria, 25 per cent. ; in France, 22.9 ; in Hanover, 39.4 ; in Belgium, 30 ; in Portugal, 22 per cent. In all these armies the same causes are in action, and the predominance of the disease is mainly to be sought for in the impure barrack air (Parkes's *Practical Hygiene*, p. 509, 2d ed.). Some are inclined to ascribe this excessive amount of pulmonary disease and mortality to the night duties of the soldier—a statement in some measure supported by the large amount of mortality from consumption amongst night-watchmen generally ; but when one looks to the age and height of the men enlisted for the regiments of the guards, and compares them with the physiological records regarding the stature and growth of the human frame, it will be seen how, sometimes, the combination of requirements for enlistment in the regiments of guards have been little calculated to secure a hardy and efficient body of men.\* During 1859, 1860, and 1861, it appears that the mortality in the army hospitals from phthisis was not above that of the country generally, although it is clearly above that of the healthy districts ; and there can be no doubt that there still is an excessive prevalence of tubercular disease in the army ; and the astonishing disproportionate number of cases in the foot-guards (18 to 20.6 per 1000 of strength) is still as remarkable as it was twenty years ago (PARKES, *l. c.*, p. 511, 2d ed.). The tables prepared by Dr. Parkes, and given in his most valuable work on *Practical Hygiene*, clearly show that there must be a large amount of phthisis *generated* in the army ; and in the foot guards nearly *four* times as much as among the civil male population of twenty-five to forty-five years of age.

[In the United States Armies, during the first two years of the civil war—1861–2, 1862–3—there were reported of pulmonary consumption, 2508 cases and 550 deaths in the first year, and 5599 cases and 2040 deaths during the second ; being a little more than 8 cases per 1000 of

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\* On this subject the reader is referred to a little book, by the Author, *On the Growth of the Recruit and Young Soldier*. Charles Griffin & Co., London.





ment is omitted, namely, the number of soldiers annually invalided suffering with tubercular disease, the larger part of whom die soon after their discharge, and swell the civil mortuary returns.

In the French the deaths from phthisis are rather less than in the British army.

The deaths from phthisis in the British navy, during three years, averaged 2.6 per 1000 of strength, and the invaliding 3.9 per 1000 (MILROY.)]

The science of Medicine is not unfrequently indebted to non-professional people for correcting prevailing errors of belief and establishing correct opinions. No one, perhaps, contributed more in this direction, in the discharge of his own professional duties, than the late Sir Alexander Tulloch. It was long a prevalent belief that consumption was limited by latitude, and that it never appeared in warm countries—for instance, south of the Mediterranean. But this is proved not to be the case; for the returns of the army, prepared by the above writer, have shown that phthisis is more frequent in the West Indies than even in this country—a statement first made by Sir James Clark, in his work *On Climate*, in illustration of the injurious effects of that climate on consumptive patients sent there from this country. According also to the recorded opinion of this author, great heat appears to have a powerful effect in predisposing to tuberculous diseases (probably by diminishing the exercise in the open air). That it is not the climate of the place which alone produces this result in the West Indies is shown by the fact that officers were attacked in infinitely smaller proportions than private soldiers; and in consonance with the views entertained regarding the nature of tuberculosis, it is more than probable that crowded barrack-rooms, a restriction to salt diet, and drinking spirits, may have produced the result.

It would appear that England and Wales, the Cape of Good Hope, and the Ionian Islands, are more exempt from phthisis than many countries which, from their higher temperature, have hitherto been supposed to enjoy a remarkable exemption from this complaint. The result of extended observation now entirely refutes the hypothesis that paludal districts are in an eminent degree exempted from phthisis—an opinion first promulgated by the late Dr. Wells, and advocated by M. Boudin. England and Wales, the Cape of Good Hope, Canada, and Malta—countries either the driest or the best drained, and consequently suffering the least from paludal diseases—are actually those countries the most free from phthisis.

[Dr. Lawson on this point writes: “The most reliable facts seem to me to clearly indicate some degree of antagonism between marsh miasmata and tuberculosis. . . . In the Western division of the United States, miasmatic disease has been rife from the early settlement of the country; but as the lands were cleared up and cultivated malarious affections diminished, and *apparently in the ratio of this diminution has been the increase of phthisis*. The united observations of the entire profession, together with the community generally, attest the fact that consumption has increased proportionately to the diminution of periodical fever; and it is further remarked that in special localities where the malarious poison

was present in a state of high concentration, phthisis was unknown" (*Pract. Treat.*, pp. 297, 389). Dr. Pollock says: "The concurrence of the phenomena of malarious affections, even of the minor kind, with tubercle of the lung, is most uncommon. During a residence of seven years in Rome, I never saw or heard of intermittent fever in persons affected with consumption; and although the tendency of all minor ailments there, as neuralgia, headache, &c., is to assume a remittent form, a marked immunity in this respect for the phthisical was very observable" (*Elements of Prognosis*, etc., p. 88). M. Jourdanet states that of the two neighboring provinces, Yucatan and Tobasco, the former is almost free from ague, but decimated by phthisis; whilst the latter is the seat of virulent paludal fever, but phthisis is unknown, and that the phthisical patients of Yucatan are regularly sent to the marshes of Tobasco with great benefit (*Du Mexique*, &c., Paris, 1861).]

On the other hand, the influence of climate shows that phthisis is most frequent in low and damp situations; while it is far less so in the mountainous districts of all countries. Again, in whatever climate the disease breaks out, it is the opinion of many pathologists that its course is most rapid if the patient remains in that country; and therefore it is of the utmost importance to know the physical nature of the various climates of the world most suitable for the tuberculous patient (see Appendix to the third edition of Dr. Walshe's work *On Diseases of the Lungs*; and Sir James Clark's classic work *On Climate*). The late Dr. Hennen's experience convinced him, when the disease broke out among our troops on the shores of the Mediterranean, that no other chance remained of prolonging the patient's life than by at once sending him back to this country.

[From the United States Census Tables, and other statistics—in many respects, it must be admitted, imperfect and having many elements of error, but not sufficiently so to invalidate general results—the fact is developed that phthisis in the United States progressively decreases from north to south. It originates far less commonly in the Southern than in the more Northern regions; it gradually, but perceptibly, diminishes from Maine to Florida. Dr. Lawson sets down the mortality from tubercular consumption as three times greater in the Northern than in the Southern States of the Union (*l. c.*, p. 229).]

That the tuberculous constitution is transmitted from parent to child has long been a popular belief, and regarded as one of the best established points in the etiology of the disease. Actual *proof*, however, writes Dr. Walshe, has never yet been afforded of the justness of the general conviction; and as a step towards an *accurate* settlement of the question, Dr. Walshe has analyzed and recorded (1849) the family history of 102 phthisical patients admitted into the Brompton Hospital for consumption. From these records it appears that about 26 per cent. of phthisical subjects in a given generation come of a tuberculous parent—a circumstance which may be predicated of any mass of individuals taken in hospital—namely, that 26 per cent. of them are of phthisical parents. On the other hand, while the general statement may be made, that

some cases of phthisis may be traced to hereditary influence, it is undoubted that *much phthisis is, in each generation, non-hereditary.*

It further appears that in *males* the malady exhibits itself at a mean period of about two years earlier, and in females at a mean period of about three years and a half earlier when there is a parental taint than when there is not. Phthisical persons spring from a phthisical source with a certain amount of frequency; and that freedom from taint in parentage is *probably* more rare, and the existence of such taint *probably* more common, in phthisical than in non-phthisical patients; but it is possible that, if investigation was extended to infancy, childhood, and youth, the ratio of cases of parental taint among the phthisical would be proportionably greater than it proves where inquiry is limited to adults.

It appears, also, that while about 9 per cent. more phthisical than non-phthisical persons come of a consumptive father or mother, on the other hand, there are about 10 per cent. more phthisical than non-phthisical persons free from parental taint.

The final conclusion which Dr. Walshe arrives at, after a most careful and logical analysis of 446 cases of phthisical and non-phthisical cases, is this,—That *phthisis in the adult hospital population of this country is, to a slight amount only, a disease demonstrably derived from parents*; and there is no reason to believe that the law differs among the middle and higher classes of society. But amongst the phthisical cases which form the subject of Dr. Walshe's inquiry, we have the tuberculous cachexia communicated both as regards the parents and the generation following; but there is still a class of cases to be inquired into in a similar manner—namely, such as will show how far parents laboring under *scrofulous cachexia* merely entail on their offspring *a disposition* to tuberculous affections. It is now a well-known fact, emphatically insisted upon by Sir James Clark, that in the families of consumptive parents there are constantly to be met with instances of ill-health characteristic of the scrofulous constitution; and in general such instances are much more frequent and much more strongly marked in the younger than in the elder children; nay, there are families in which the elder children are healthy, and the younger ultimately become the subjects of tuberculous disease. In such cases it has been presumed that in some instances the health of the parents has become deteriorated during the increase of their family. The mere fact of the parents being unhealthy, and not necessarily tuberculous, appears in some instances as if sufficient to entail tuberculous diseases upon their children. This statement is in some measure borne out by a result obtained by Dr. Walshe, that as far as the mere phthisical or non-phthisical condition of parents is concerned, about 24 per cent. of tuberculous patients can trace the origin of their disease to either parental source.

**Contagion of Phthisis.**—Regarding the possible contagious propagation of pulmonary phthisis, Dr. Parkes thus expresses himself (having regard to the fact that purulent and epithelial cells have now been demonstrated as floating about in the air where numbers of persons are together): “Considering that the pleuro-pneumonia

of cattle is probably propagated through the pus and epithelium cells of the sputa passing into the air-cells of other cattle; that even in man there is some evidence of a pneumonic phthisical disease being contagious (Bryson, *Cases in Mediterranean Fleet*), the floating of these cells in the air is worthy of all attention. It may explain some of those curious instances of phthisis being apparently communicated" (*Prac. Hygiene*, p. 74).

[Morgagni said *phthisicorum cadavera fugi adolescens, fugio etiam senex*. In Italy consumption has been, and still is, looked upon as a communicable disorder, a consumptive is shunned, and the vessels he may use in eating and drinking are avoided or destroyed, and his clothes burnt or buried. The opinion that long and continuous exposure to the body-effluvia of a tuberculotic patient puts in danger a previously non-tuberculous person to the risk of the disease, by a predisposition to it, has been held by Jos. Frank, Laennec, Sir James Clark, and others. Andral went so far as to say that under certain conditions, these tubercular emanations became a source of true contagion. Most medical practitioners of long experience have seen examples of the apparent contagion of consumption—a tuberculous husband infecting a wife, and the reverse. The late Dr. Leger lately communicated to Dr. Villemin some curious facts of this kind (*Gaz. Hébdomadaire*, 1868). That the tuberculous diathesis may be transmitted from the male to the female by the medium of the fœtus, and this even in such a way that children begotten by a second non-tuberculous husband, may inherit the diathesis acquired from the first or tuberculous husband, would seem likely from the many cases reported by Dr. Perroud of Bordeaux (*De la Tuberculose, &c.*, Paris, 1866), and by Dr. Alexander Harvey of Aberdeen (*Edin. Medical Journal*, 1849, 1850, 1854). Although Sir Thomas Watson explicitly states that he does not believe phthisis to be contagious, he adds: "Nevertheless I should, for obvious reasons, dissuade the occupation of the same bed, or even of the same sleeping apartment, by two persons, one of whom was known to labor under pulmonary consumption" (*Lectures on the Practice of Physic*). Dr. Fuller says: "But, though the non-infectious character of phthisis be admitted, it behooves the physician to warn the patient's friends of the dangers incident to long-continued attendance on him, especially if the disease be in an advanced stage. It would be the height of imprudence for a healthy person, and especially if young and of a scrofulous diathesis, to sleep in the same bed, or even in the same apartment, with a consumptive patient, for although the malady might not be communicated directly from one to the other, unless possibly under the condition of some tubercular matter being accidentally introduced into his air-passages or into some other part of his system, the surroundings and the air would be calculated to predispose him to the disease" (*On Diseases of the Lungs and Air-passages*, p. 431). Dr. Villemin suggests that besides the direct transmission, as by cohabitation, consumption may be contracted through indirect means, by clothes, bed-linen, water-closets, the vitiated air of rooms lived in by tuberculous persons, &c. The possible transmissibility of the disease in this manner merits, he thinks, the attention of medical officers of the army. A tuberculotic soldier dies in the hospital, and his clothes are returned to his company and worn by another: may not this, he asks, be one source of phthisis in the army? He is satisfied that the barrack is to the soldier in the production of consumption, what the regimental stable is to the horse in the

development of farcy, the contagion and transmissibility of which are at length accepted. Fournet, who in his work is a non-contagionist, still gives some weight to the possibility of infection from an atmosphere constantly breathed, and necessarily poisoned, by the consumptive.

In the recent protracted and unfruitful debates upon phthisis in the French Academy of Medicine, the subject was fully discussed; the views of two of those to whose opinion we are disposed to attach value will be quoted. Dr. Jules Guérin believed that crude tubercle can never be contagious; but, that when it is softened, and the ulcerated lung-surfaces are exposed to the air, the patient may become a source of infection to those about him, just as the pulmonary lesions he has infect his own organism by the resorption of purulent and putrid products. Dr. Bouillaud's ideas would seem to be nearly the same; he said: during the course of pulmonary tuberculosis, when pus or other septic products are formed in parts which are accessible to the atmosphere, phthisis, like so many other affections in which similar purulent foci happen, becomes indirectly a cause of septic infection. The tuberculous virus, he added, is an hypothesis, which, up to the present time, rests upon no exact or trustworthy observations, and there does not exist a single instance of tuberculosis of the lungs, or of any other part of the body, being produced in the human species by means of specific (virulent) inoculation. And this leads to the brief consideration of a subject of much present interest, and the newest wind of pathogenetic doctrine, namely:

**The Inoculation of Tubercle.**—Thirty years ago Erdt produced, what he supposed to be tubercular nodules, in the lungs of horses, by inoculating them with scrofulous matter from man, in a series of experiments which he made to ascertain the identity of scrofulosis and farcy. In 1865 Dr. Villemin published his experiments on the inoculability of tubercle from man to the lower animals, and from one animal to another. He introduced minute particles of gray, and agglomerated, tubercle, as well as cheesy tubercle from the human subject, under the skin of rabbits and guinea-pigs, and, with hardly an exception, the lungs and other viscera of the animals became filled with tubercles, usually miliary, often agglomerated, and sometimes cheesy. He inoculated a rabbit with tubercular matter from a cow, and caused a more rapid and general tuberculosis. He inoculated from rabbit to rabbit, and got an intense and widespread tuberculosis. The sputa of phthisical patients, and the blood of tubercular rabbits, produced invariably tuberculosis in rabbits. The matter of cheesy pneumonia, accompanying phthisis in man, proved quite as inoculable as gray miliary tubercle. He failed in the inoculation of cancer, hepatized pneumonic lung, and pus in its several forms, as well as the cheesy matter taken from the glands of the neck in cases in which there was some chronic eruption of the head or face. The following recent experiments of Dr. Villemin were communicated by him to Dr. Paul Spillman (*Archives Générales de Médecine*, Sept., 1868). Three rabbits were inoculated with dried sputa of consumptives; on being killed on the twentieth day, they were found tuberculotic. Some of the same sputa in powder was blown into the trachea of two other rabbits; one became tuberculotic. Finally, blisters of the size of a copper cent were placed upon the chest of two other rabbits, and when they had acted, the raw surface was dusted with some of the above powder; tuberculosis followed in one.

The first experiments of Villemin were soon afterwards repeated by Mr. John Simon, the eminent English pathologist, and he found both the yellow and gray tubercle inoculable in the same manner as asserted by the French observer, he having used not a larger quantity of tubercular



matter than is employed in vaccination. He came to the conclusion that, "whether called tubercle or not the action must be allowed to be specific;" and the committee of the London Pathological Society, on examining Simon's specimens, thought their tuberculous character to be beyond doubt. Bizzozero, Verga and Biffi of Milan, and Mantegazza satisfied themselves that the nodules in the viscera of rabbits inoculated with human tubercle are really tubercular, and, watching their evolution, inferred that they are formed by proliferation both of the connective tissue and of the epithelial elements. Dr. Hérard took seven rabbits and inoculated five of them—three with gray and semi-transparent, or yellowish, tubercle from the pleura and peritoneum of a patient who had died of phthisis, and two with the cheesy matter of caseous pneumonia. At the end of two months, all the seven rabbits were killed. The two not inoculated were healthy; the two inoculated with cheesy matter were also healthy; and two of three inoculated with miliary tubercle were decidedly tuberculous, and he concludes that the miliary tubercle is alone inoculable, and is the specific lesion of phthisis. Dr. Andrew Clarke, of London, next repeated Villemin's experiments; he divided his animals (rabbits) into four groups; he placed the first under very unfavorable hygienic conditions; the second he made breathe air in which there were various irritant substances; into the veins of the third he injected dry bone-dust, or particles of sand; and the fourth and last were inoculated with fresh tubercular matter by introducing it subcutaneously into the neck. In the first series, yellowish masses, sometimes agglomerated, appeared, and soon ended by transforming the lung into a cheesy mass. Most of the lungs of the second series showed fibroid change, with deposits similar to those found in the lungs of coal-miners, masons, steel-grinders, and others who are subjected to the inhalation of irritating dust. After the injections into the veins of fluids holding in suspension fine particles of bone-dust and sand, yellowish or grayish spherical masses, some of them the size of a pea, were found in the lungs, with some of the solid matter in their centres; secondary deposits of the same kind existed in the liver and kidneys. Finally, in the last group, inoculated with true tubercular matter, at the end of a fortnight or three weeks, gray granulations, semi-transparent, hard, and slightly prominent on the cut surface of the lung, were found uniformly disseminated in every part of the organ. Though these experiments would seem to confirm the views of Villemin, Dr. Clarke thinks that they do not establish the identity of the gray tubercle in the rabbit and in man, or that the gray granulation is the local expression of a specific virus, capable of being propagated by inoculation, as the virus of syphilis; indeed he believes that the gray tubercle of the rabbit differs in many respects from that of man; and moreover he twice produced the same results in two instances by using non-tuberculous diseased products—cancer and pus-matter. Waldenburg, as the result of his observations denies the specificity of tubercle, holding that any substance in minute division, smaller, or at least not larger, than the blood-globules, reaching and being arrested in the lung, will be followed by tuberculosis of the organ, acting either mechanically or chemically.\*

Dr. William Marcet's series of experiments of inoculating guinea-pigs with the sputa of phthisical persons in the second stage of the disorder, was perfectly successful in producing tubercle in the inoculated animal;

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\* According to a communication recently made to the French Academy of Medicine, by Dr. Colin, Waldenburg, after inoculating animals with matters blued with aniline, found them thus tinted in the artificial tubercles produced in the organs.—EDITOR.

and he, moreover, found that other substances taken from the human body in certain if not in all stages of phthisis, as blood and pus, seem to be also possessed of the power of inducing the formation of tubercle in guinea-pigs when inoculated to these animals.

All these observations have been regarded as proving conclusively the fact that tuberculosis in man is directly communicable by inoculation to the lower animals. The experiments of A. Clarke, and Waldenburg, seemed however to militate against the specific character of the inoculation. Lebert and Wyss, of Breslau, performed last year a number of varied experiments with the view of settling this question: Are the morbid appearances in animals inoculated with human tuberculous matter due to any specific or virulent property resident in the inoculated matter, or are they the secondary products of metastatic infection—capillary emboli consecutive to the carriage of the effects of the so-called inflammatory processus? Along with tubercle in its several stages, they used the cheesy products of inflammation of the lungs and of the lymphatic glands, and the matter of melanoma, cancer, cancroïd, and sarcoma: the results of these inoculations were compared with the effects brought on by the injection of simple mechanical irritants, as charcoal and mercury, into the veins of animals, and it was found that the embolism of the smaller pulmonary twigs produced by these gave rise, by propagated irritation, to changes nearly akin to those of induced tubercular disease. These views of Lebert have quite recently had strong confirmation from the experiments of Dr. Burdon Sanderson, and Dr. Wilson Fox, of London. All three of these observers worked independently of each other, and have come to the same conclusions, and unfavorable to Villemin's doctrine of specific tubercle-inoculation. While they have confirmed the fact that the inoculation of the human tubercle in some animals produces a condition apparently the same as general tuberculosis in man, they have shown that this happens, not by virtue of any specific or virulent property in the human tubercle, but simply by the mechanically irritating effect of the operation. Sanderson and Fox both found that the inoculation of tuberculous matter in rodents will not only be followed, in a large majority of cases, by a diffusion of miliary granules through the viscera, but that the same results follow any severe and prolonged subcutaneous irritation. Fox put under the skin of guinea-pigs minute particles of putrid muscle, bone, and kidney, or a thread saturated with vaccine lymph, and even a simple seton, and each gave rise to a growth not distinguishable from tubercle. Dr. Fox remarks that the analogy between the substances used by him in his experiments, and those which, under certain circumstances, already exist in the system, is very important. It shows how tubercle in the lung may be often secondary to pneumonia, and tubercle elsewhere secondary to that of the lung; how the presence of an irritant, as diseased bone, may give rise to the formation of tubercle, while if the bone is removed the danger is averted.

See Villemin: *Etudes sur la Tuberculose*. Paris, 1868. Hérard and Cornil: *De la Phthisie Pulmonaire*. Paris, 1867. Lebert: *Gazette Médicale*, 1867. A. Clarke: *Med. Times and Gazette*, 1867. Marcet: *Medico-Chir. Trans.*, 1867. Sanderson: *Brit. Med. Jour.*, 1868. W. Fox: *Brit. Med. Jour.*, 1868. Spillmann: *Archives Générales de Médecine*, vol. ii, 1868. *Brit. and For. Méd.-Chir. Rev.*, vol. ii, 1868.]

**Treatment.**—The principles of treatment having been already laid down under "Scrofula," p. 245, it only remains here to warn the student (as Dr. T. K. Chambers has so ably done in his Lectures, p.

270) against the use of so-called "cough medicines," antimony, ipecacuanha, and squill especially; also against the use of mercury, purgatives, and neutral salts. To foster and cherish an appetite for food must be the great aim of treatment. One of the best tonics is the syrup of iron, quinia, and strychnia, of which the formula by the late Professor Easton, of Glasgow, has been given at p. 95, *ante*. Let it be given in very small doses at first; and followed up by the use of cod-liver oil "of the most agreeable, clearest, sweetest, and most scentless kind." The *brown oil* ought never to be prescribed, except, perhaps, to an Esquimaux. The oil should be given in teaspoonful doses at first. The old *vinum ferri* in doses of one teaspoonful hardly ever disagrees; and equal parts of *compound squill pill* and of *opium pill* (P. B.), given in *five-grain* doses at bedtime, may procure sleep at night; and the dose may be increased to ten grains if the cough is troublesome. A mixture containing six or eight grains of *Dover's powder*, with ten drops of *tincture of squills*, ten drops of *tincture of Tolutan*, with a little mucilage and syrup, may also quiet the cough.

[The curability of consumption is becoming every day more generally owned by the profession, and yet it is, and probably always will be, one of the most fatal disorders which afflict mankind; and for the reason that the conditions, under which recovery, or continuous and substantial amendment, whether as an unassisted act of nature or a success of art, can be looked for, are precisely such as the largest number of sufferers from the disease cannot carry out. If it were limited to persons who could, or would, abidingly and earnestly follow the necessary hygienic treatment, and have their choice of climate, its arrest, with a tolerable share of relative health, might more often happen. The fashionable professional dogma of "consumption curable" is based not only on a belief in the possibility of cure founded on a better present knowledge of its history and pathogeny, and from untrustworthy statements of the results of modern treatment, but on the error of looking upon all chronic pulmonary deposits as alike, and not distinguishing between those which, under favorable circumstances, are, comparatively, susceptible of removal, and connected with less serious damage to general nutrition, and those which are the local evidence of true tuberculosis, in which organic vitality is fatally compromised. In estimating the effect of any one medication, or of any particular drug, the chances of error are so great that the most cautious, truthful, and painstaking observer may deceive himself; and, however, seemingly strong may be the proofs in favor of this method, or of that remedy, it should be borne in mind that the one may have been adopted, or the other administered, at the time of one of those natural pauses which are so marked a feature in the clinical history of phthisis, and thus be fallacious. "No one," wrote Louis years ago, "dies of a first attack of phthisis," and, excluding acute tuberculosis and rapid consumption, this is substantially true. "It is a disease" said Andral, "in which an ever-present lesion gives rise to intermitting symptoms." Do the vital statistics of Europe or of this country show any lessening in the death-rate of consumption? Do our hospital registers, and particularly those of institutions specially devoted to consumptives, furnish hopeful data? Facts and daily experience teach the truth, and warn us against admitting the exception as the rule.

Nor is the testimony sought to be drawn from post-mortem observations in favor of recovery from an advanced stage of consumption, any more convincing or encouraging. The subjects examined by Dr. Henry Bennett, and the late Dr. Rogée, in whose lungs large cretaceous deposits and puckered cartilaginous cicatrices were found, and who had died of other disorders, were all over sixty-five years of age, and at this time of life such tissue degenerations and calcareous masses are common, and have no necessary connection with tubercular consumption. Maclachlan found them in one-half of all the bodies he examined at the Chelsea Hospital, a retreat for aged soldiers mostly between sixty and eighty years of age (*Practical Treatise on Diseases of Advanced Life*, 1863). Rogée found them in fifty-one out of one hundred autopsies of old women, and amongst a class of persons in whom, from the unfavorable circumstances they are placed in, phthisis is almost surely fatal.

It must, however, be admitted by the most exacting and incredulous, that there are a certain number of well-attested instances in which all the symptoms of consumption having been present, along with the physical signs of local deposit, either stationary or undergoing change, yet apparent recovery has taken place; and others again, and a larger number, where there has been an arrest in the progress of the affection, and fair health enjoyed for many years. While weighing justly the evidence brought forward in support of the doctrine of the curability of *tuberculosis*, and finding it wanting, still much may be done towards the hindering, if not the extinction of *tuberculization*, by eradicating the vicious diathesis on which it depends,—what Rokitansky has called the fundamental tubercle-producing crasis. To do this we must secure such advantages of food, climate, and habits of life as will rally the general health, and make construction exceed waste. This subject has already been largely treated of (p. 245, of this volume). The necessity of fatty food was insisted on, and hence the value of cod-liver oil in this complaint. It is to be recollected that irritative dyspepsia is common even in the early stage of the disease, with marked dislike to grease; in such cases the gastric and hepatic disorder must be relieved before any attempt is made to push the fatty diet. Under these circumstances the *pancreatic emulsion*, proposed by Dr. Dobell, under a faulty theory, and vaunted, it is to be feared, with false statistics, has received the respectable indorsement of Dr. Fuller, who says that several of his patients who had an utter indisposition to take fatty matters, received much benefit from its use. Cream, beef-marrow, and bacon-fat may be tolerated when repugnance to the oil cannot be overcome. The oil may also be advantageously administered by inunction. There should be careful choice in meats and drinks; of the former those rich in osmazone, and roasted, are to be preferred, and raw meat, chopped fine, has been recommended by Fuster and Perroud. The full-bodied wines, and malt liquors, are better than the more alcoholic fluids.

Phosphorus, a necessary element of the tissues, has been found to be lessened in diseases accompanied by emaciation, and much has been said of good effects from the employment of the phosphates and hypophosphites in consumption. As a specific treatment it is entitled to no respect; as supplying a deficient ingredient in the economy, and hence as a true aliment, it becomes of real advantage. While therefore improved nutrition may happen under the use of the hypophosphites, chemical food, phosphoric and phosphorous acid, surer and more permanent good can be got by giving phosphorus alimentally in an organized form, in which it may be easily assimilated, using as articles of diet such organic substances



as contain it. Along with fish, eggs, and the like, the wheat-phosphates (having the organized chemical products existing in the outer layers of the wheat grain) may be used with this purpose. Perroud praises the highly phosphuretted fatty matter of the brain of the herbivora made into a sweetmeat with sugar.

After an experience of many years with it, the writer has come to regard arsenic as a most reliable improver of general nutrition and of the assimilative functions. In consumption he has been in the habit of giving one or two drops of Fowler's solution, or even less, or the one-fiftieth of a grain of arsenious acid, once, twice, or three times daily, during the meal, and continuing it for months, with occasional intermissions. Where the digestive functions are feeble, and food of all kinds, but particularly the fats, is illy borne, the effects of arsenic carefully used is constantly happy. It has recently been recommended in consumption by Dr. Perroud, and by Dr. Moutard-Martin. It will not prove disappointing just in proportion as it is rightly used, and its proper value understood.

Amongst other drugs which aid in the restorative treatment may be mentioned iron, manganese, the vegetable bitters, and the salts of iodine. The latter certainly exercise some influence on the process of retrograde tissue metamorphosis, and though all the extravagant hopes of the effects of iodine on phthisis have long since been given up, yet in many cases decided benefit seems to follow the use of iodide of potassium, which may be combined with the ammoniated citrate of iron, or tincture of the chloride, and some bitter infusion, as cascarrilla, columba, or wild-cherry bark. Perroud points out the important part of the chloride of sodium in the economy, and urges its large use in phthisis; its utility was greatly extolled thirty years ago by Latour, and afterwards by Dr. McDowell of Kentucky.

Too much stress cannot be laid upon the necessity of keeping the skin in condition; and this can best be done by a proper system of bathing, and by daily gentle hand-rubbing (grooming) of the whole surface. An occasional Turkish bath will, when the skin is harsh, dry, and inactive, and the patient is not too much reduced, be of service; or a common warm bath followed by a cold shower and moderate friction with a towel. All these means, along with moderate and frequent outdoor exercise, or suitable gymnastics, are far better to restore the skin-function, and overcome cutaneous susceptibility, than heavy clothing and coddling. An open-air life, the activity of which should be measured by the strength and endurance of the patient, is of the first importance in the treatment of consumption.

The consumptive patient suffers constantly from present conditions of discomfort, pain, and distress, and it is for the relief of these that he most frequently applies to the physician. The most common are cough, stitch-pains, and sweating. For the first, the so-called nauseating expectorants, so generally prescribed, do harm by deranging digestion, which it is so important to keep whole. It arises from so many causes that no general rules for its management can be laid down. If there is an elongated uvula, the end should be snipped off; if there is irritation about the pharynx and upper part of the larynx, it may be relieved by the inhalation of some of the atomized fluids (see writer's article on this subject, p. 821); it is often much under the will of the patient and may be controlled by an effort; opium is to be avoided when possible, and the bromide of potassium, tincture of the cimicifuga, and prussic acid, first tried. Of the preparations of opium, codeia is probably the least harmful in this complaint; it may be given in combination with prussic acid.



For the local pulmonary congestions, dry cupping, and derivatives, may be used, though the tendency to them is best hindered by restoring the skin-function, and equalizing the circulation. The attacks of intercurrent bronchitis and pneumonia may be treated with muriate of ammonia; and in the more chronic and asthenic forms some patients have found great relief from the wine of tar; it is of value when it is borne by the stomach. Counter-irritation to the chest-wall is a stereotype method in this condition, and when properly used in the earlier stages before there is much loss of strength it is undoubtedly beneficial; but later it is weakening and annoying. Croton oil liniment is the chief favorite, but a prompt and not too severe application is the following ointment recommended by Dr. Fuller.

R. Hydrarg. Chlor. Mit. gr. viij; Iodinii, ʒss.; Alcohol, fʒiss.; Unguent. Simp. ʒj. M. Rub in a portion over the affected lung morning and evening until a pustular eruption comes out.

A solution of the nitrate of silver (30 grains to the ounce of distilled water) may be painted on the skin beneath the clavicles every evening until the skin is darkened; and repeated after the cuticle peels off. Gentle and continuous irritation of the skin may be kept up, by wearing constantly on the chest a piece of flannel wet with a weak solution of iodine in glycerine and water, covering the cloth with oil-skin, to prevent too rapid evaporation and soiling of the clothes.

The night-sweats, so often annoying and profuse, better under general systemic improvement and restored cutaneous functions. Bathing with vinegar and water, alum and water, or diluted alcoholic liquors, are well-known remedies, as well as gallic and tannic acids, nitrate and oxide of silver, and oxide of zinc, and infusion of common garden sage.

Little can be done to check, or even relieve the diarrhoea when it once sets in. A flannel bandage around the abdomen should be worn; the traditional treatment is by bismuth, astringents, and opiates. When it is possible, let the stomach be spared, and drugs be given in enemata.]

### ACUTE PULMONARY CONSUMPTION.

**Definition.**—*Tuberculous growths or exudation in the lungs, expressed by febrile symptoms running an extremely rapid course, denoting the severity of the constitutional disturbance, proving fatal in from twenty days to ten or twelve weeks; and due to (a.) acute miliary tuberculosis, or (b.) acute pneumonic phthisis, or to both combined.*

**Pathology.**—Acute tuberculosis has usually been considered to be a comparatively rare disease; but it would appear that this fatal affection is unusually prevalent among the soldiers in Paris, among whom also the chronic form of phthisis is very common, probably more so than even in our own army. In 1861, M. Colin (Professeur agrégé au Val-de-Grâce) records *five* cases of *acute tuberculosis* in the soldiers of the garrison of Paris (Parkes, *Army Med. Dep. San. Report*, 1860, p. 357).

The disease occurs in two forms: (1.) One form is connected with extensive growth or infiltration of yellow or crude tubercles of the lungs, with irregular softenings in the centres of some, or with small excavations surrounded by patches of hepatization. (2.) The form most characteristic of, or most commonly found in, acute

phthisis is that in which there is general studding of both lungs with semi-transparent gray granulations, combined with pneumonia in its first stage, bright arterial injection, or hepatization (WALSHE). The two forms may coexist in the same lung.

The **Symptoms** are those of an intense febrile affection. Dr. Parkes has favored me with the notes of a case, exhibiting the correlation of the temperature, the pulse, and the respiration, during the course of a fatal case of acute phthisis. The records of temperature are as in the diagram on p. 800.

During *thirty* days the pulse ranged from 116, the lowest, to 178, the highest, and having a mean of 140, reckoned by the number of observations. The respirations *per minute* ranged from 36 to 60, having a mean of very nearly 50. In Dr. Walshe's cases the relationship of the pulse to the respiration has varied considerably. The average has been as 3 to 1.

The functions of the lungs are deeply impaired. The invasion appears to occur while the patient is in a state of health, or the fever may be remotely preceded by various depressing influences, and immediate exposure to cold and wet; after which rigors ensue, followed by acrid heat of skin. The rigors recur on several successive days, followed by perspirations, and sometimes crops of sudamina. Epistaxis, followed by coryza, may occur on the second day of seizure (WALSHE). Prostration sets in early, so that in a few days the patient may be unable to stand. There is thirst, total anorexia, epigastric tenderness, dry lips and tongue, dental sordes; all signifying great intestinal disturbance. Diarrhœa is rare, and constipation may be extreme, even with abdominal pain and ulcerated intestines. Restlessness, *insomnia*, *cephalgia*, *vertigo*, *tinnitus aurium*, diurnal wandering and nocturnal delirium, bespeak cerebral complications and the probable growth of tubercles in the arachnoid membrane. The physical signs vary with the amount of tubercular growth or *pneumonic infiltration* in the lung, and are similar to those which have been described.

[The distinguishing trait of Acute Pulmonary Consumption is the un-deviating continuousness of the morbid changes. The two anatomical forms (p. 797), have attached to each a distinct clinical history, and, probably, pathogeny.

(1.) **Acute febrile** (*Gallopig Consumption*).<sup>\*</sup> Of this form there are two varieties: (a) *Pneumonic*; (b) *Typhoid*.

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\* [Hippocrates' description of acute phthisis, as he saw it in the island of Thaso, in the Ægean Sea, is wonderfully complete. The overlapping febrile paroxysms, the gastric symptoms, the sputa, delirium, the state of the fauces, and the rapid waste, are all mentioned. "Consumption was the most considerable of the diseases which then prevailed, and the only one which proved fatal to many persons. Most of them were affected in the following manner: fever, accompanied with rigors of the continual type, acute, having no complete intermission, but of the form of the semiter-tians, being milder the one day, the next having an exacerbation, and increasing in violence; constant sweats, but not diffused over the whole body; extremities very cold and warmed with difficulty; bowels disordered, with bilious, scanty, unmixed, thin, pungent, and frequent dejections. The urine thin, colorless, unconcocted, or thick with a deficient sediment, not settling favorably, but casting down a crude but unseasonable sediment. Sputa small, dense, concocted, but brought up rarely and

Though the invasion of the *pneumonic* variety is often apparently abrupt, it is frequently preceded by loss of strength and appetite, thirst, weariness, and body-waste, some febrile movement, a slight, dry, hacking cough, and an occasional tinge of blood in the saliva. Hæmoptysis may, however, be the first symptom to attract attention, and be slight or profuse. These symptoms may last for weeks or months, when suddenly there is general illness—remitting fever with exacerbations, a dry and very hot skin, irregular and partial sweats, a quick, sharp pulse, and, sooner or later, gastric and intestinal troubles; the fauces are dry and injected, the tongue red, with anorexia, tormenting thirst, nausea, vomiting, diarrhœa, rapid and progressive wasting, great weakness, and restlessness. Breathlessness is complained of, and often becomes, at an early period, extreme; the respiration is hurried; the cough constant and tormenting, dry at first, but soon accompanied by expectoration, which may, however, be scant or wanting throughout. These symptoms rapidly worsen, delirium sets in, and the patient dies exhausted, and sometimes asphyxiated.

In the *typhoid* variety, the febrile phenomena are more intense; there is often from the outset, severe headache, insomnia, restlessness, a stupid expression, delirium, tranquil at first, but often violent, and later, subsultus; the eyes are bright and injected, the face flushed and subsequently livid, and the breathlessness excessive and increasing. In its general physiognomy, this variety of acute phthisis very closely resembles that of common continued fever, and the diagnosis is often embarrassing.

The body-heat in Acute Phthisis is commonly as high as  $102^{\circ}$  to  $104^{\circ}$ , and in some cases mounts to  $107^{\circ}$ ; though usually persistent, it sometimes falls for even a day or two to near the range in health; the pulse, however, keeps rapid, and in about twenty-four hours the temperature will be found to have again risen to its former height.

The physical signs of Acute Consumption give evidence of diffused and extensive deposits in one or both lungs, and of inflammation of their tissues, the signs of each being with difficulty distinguished. The chest-resonance may, or may not, be sensibly lessened; there are prolonged expiratory murmurs, bronchial respiration and voice, soon followed by the evidences of softening,—subcrepitation, more or less moist clicking, and mucous rhonchi. Death sometimes happens in the first stage, before softening. The physical signs rapidly change, and those of all the stages are found simultaneously in one or both lungs.

Circumscribed cavities probably never form, and the irregular excavations which may occur, give no distinctive signs, though Trousseau mentions a case in which he heard the cracked-metal sound and cavernous respiration over the clavicle.

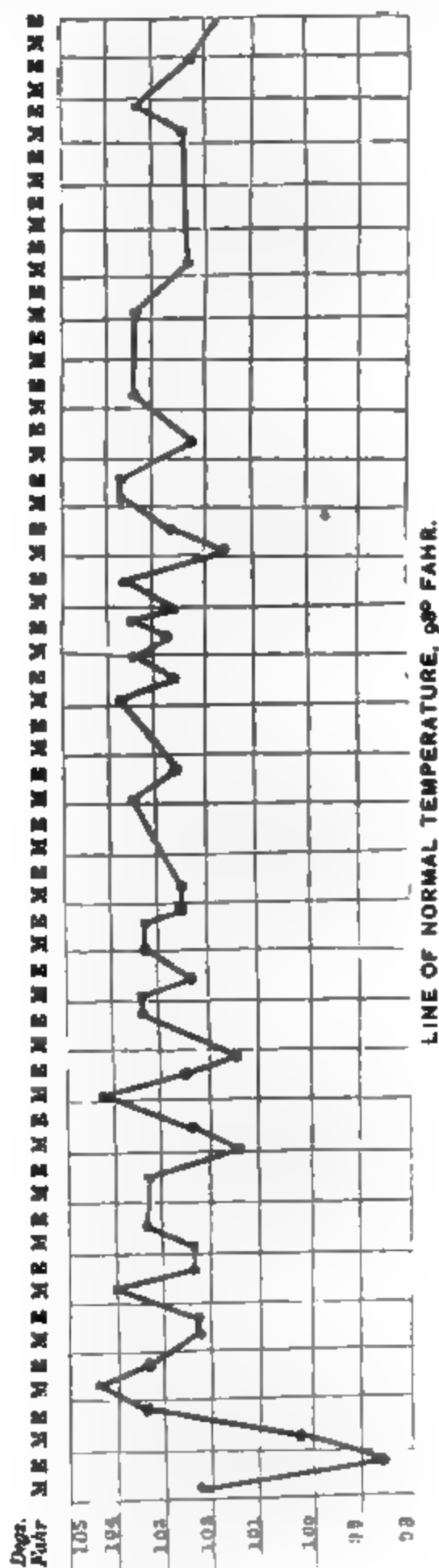
The course of this disorder may be overwhelmingly rapid. In a case reported by Dr. Law, in a boy, æt. 5, death took place on the tenth day after the general illness; Dr. Flint mentions one where the patient died eleven days after the occurrence of copious hæmoptysis, the initial symptom; and another whose duration was five weeks; and Trousseau gives

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with difficulty; and in those who encountered the most violent symptoms, there was no concoction at all, but they continued throughout spitting crude matters. The fauces, in most of them, were painful from first to last, having redness with inflammation, defluxions, thin, small, and acrid; they were soon wasted and became worse, having no appetite for any kind of food throughout; no thirst; *most persons delirious when near death*'' (*The Epidemics*, Book I. *The Genuine Works of Hippocrates*, Sydenham Society's Edition, vol. i, p. 854).—EDITOR.]

one which lasted eighteen days. The duration of acute febrile phthisis rarely extends beyond two months, though it may be prolonged to six, but, its progress is persistently continuous. It is invariably fatal, reme-

DIAGRAM SHOWING THE RECORDS OF TEMPERATURE IN A CASE OF ACUTE PHTHISIS (Parkes).



dies having no power to check or moderate its course. The prognosis respecting time, is to be determined by the intensity of the fever, and

the degree of exhaustion. Women are most liable to this form of acute phthisis. It is eminently a disease of youth, and is rarely met with after thirty-five.\*

As has been stated (p. 797), its anatomical character is the presence in the lungs of generally disseminated, semi-transparent, gray granulations, associated with products of acute inflammation of the pulmonary tissue. Pathologists differ in opinion as to the nature of these granulations: whilst many hold that they have all the essential properties of tubercle, and represent the early stage of that product, others maintain that they are histogenetically distinct (CH. ROBIN). It must be remembered, however, that the subjects of acute febrile consumption have, for the most part, decided tokens of the tuberculous cachexy for some time previous to general illness, and that the influence of hereditary predisposition is rarely wanting. Out of 179 only 34 could positively state that there was no family taint either parental or remote (POLLOCK). This form of acute phthisis must be regarded as composed of two factors,—a local inflammation of great violence, and a constitutional disorder which determines a deposit in the lung of the lowest grade of aplastic formations. The local disease is a combination of inflammatory products, in every tissue of the lung,—air-sacs, bronchi, pleura,—and of degraded morphological elements; the systemic affection reflects both disorders. Happening sometimes with no assignable exciting cause, it more frequently occurs after childbirth and during prolonged nursing, or immediately follows an attack of continued or of eruptive fever, particularly measles.

**Diagnosis.**—In ordinary pulmonary consumption, tubercular deposit limited to a portion of a lung may undergo sudden and rapid softening, followed by excavation. In such cases the amount of constitutional disturbance is commonly great, and the fever continues, with exacerbations, while the destructive changes are going on, abating or ceasing when a cavity is formed. Here the physical signs limiting the site of the deposit, and indicating the progressive changes, will prevent an incorrect diagnosis or prognosis. With some general resemblance to typhoid fever, it should not be mistaken for that disorder, if the distinctive symptoms of the latter are borne in mind, and the history of the case noted.

(2.) **Rapid Pulmonary Consumption** is characterized by the passive and universal formation of tubercle throughout both lungs in a short space of time. It is accompanied by general malnutrition, speedy and progressive waste of the body, decay of vital force, a hot and dry skin, no active fever, moderate sweating, but little or no gastric disturbance or diarrhœa, and not unfrequently there is neither cough nor expectoration, nor hæmoptysis, but constant breathlessness, increasing to distressing oppression upon the slightest exertion, or upon lying down.

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\* [Dr. Pollock says there is no positive immunity, even in advanced life. A case, æt. 63, was fatal in 3½ months, with formation of cavity. Maclachlan gives a case—male, æt. 73—death in less than 3 months, and within 6 weeks of cavities forming. Hasse gives a rapid phthisis case, æt. 89, male; and Blackiston a case, æt. 83; death in 6 weeks. The case given by Dr. Pollock (*El. of Prognosis, &c.*, p. 264), as “an interesting one,” occurring in his own practice, of acute phthisis, in advanced life (male, æt. 60), is clearly a case of sclerosis of the lung, with the same lesion of the liver. In describing the post-mortal appearance he says: “*Right lung, dark, solid, hard, non-crepitant upper two-thirds; this portion was infiltrated with granular tubercle commencing to soften; a small cavity existed at the posterior part of the apex; the deposit appeared to be a form of tubercle with an exudation of inflammatory products of white fibrinous nature; under the microscope there was a large amount of fat and tubercle corpuscles; left lung at its apex slightly infiltrated with the same; . . . liver enlarged, very hard, studded with granular deposit.*”—EDITOR.]



The deposit, often of yellow tubercle, but more generally gray granulations, are more or less solid; no softening, or only to a slight degree taking place.

The *physical signs* are, immobility of the chest-walls; diminished resonance, and increased resistance, on percussion; feeble respiratory murmur, and broncho-vesicular respiration, followed by tubal breathing and bronchial voice, and moist sounds, varying in site, degree, and intensity, and not constant. This is a rare form of pulmonary tuberculosis, and seems to happen only where the hereditary taint is well marked. Its duration is from two to four months, its course is steadily onward, and the patient fades away, dying often in the first stage.

Such cases rarely pause, and never recover.]

### SYPHILITIC DEPOSIT.

LATIN EQ., *Deposita ex syphilide*; FRENCH EQ., *Dépôt syphilitique*; GERMAN EQ., *Syphilitische Ablagerung*; ITALIAN EQ., *Deposito sifilitico*.

**Definition.**—*The development of gummatous nodules in the substance of the lungs after syphilis, with or without bronchitis or miliary tubercle.*

**Pathology.**—Morton, Sauvages, Portal, Morgagni, and more recently Graves, Stokes, Ricord, McCarthy, Walshe, Wilkes, Virchow, and Munk, have all described, with greater or less uniformity and distinctness, pulmonic lesions in cases of syphilis.

The following are the kinds of lesions which predominate:

1. *Evidences of Inflammation of the Mucous Membrane of the Bronchial Tubes.*—In such cases bronchial irritation, with fever in many cases, precedes the skin lesions, and may disappear wholly or partially when the skin lesions are established. On the other hand, if the syphilitic eruption suddenly disappears, bronchitis may ensue. Walshe records well-marked instances of this; and it is a circumstance to be looked for amongst soldiers especially, who, having recovered from a primary syphilitic sore, are apt to be exposed to the risk of bronchitis when mounting guard soon after being discharged to duty. Secondary symptoms and pulmonic lesions are then apt to date their commencement; and all the general symptoms of phthisis may supervene, and yet no tubercle in the lung may be developed; but chronic bronchitis remains persistent. On this point Dr. Walshe observes, that in the persistence of the general symptoms there “is assuredly enough to create a strong suspicion of the existence of tubercle in the lungs, taken in conjunction with the indubitable tendency of syphilis *plus* mercury to induce the outbreak of phthisis in a person having the requisite constitutional aptitude. How are the cases to be distinguished? By the total want of accordance between the physical signs and the constitutional symptoms: the patient with syphilitic bronchitis has neither consolidation signs nor, *à fortiori*, the evidences of excavation. But there is a curious source of difficulty which sometimes starts up in these cases, and renders doubt imperative—the infra-clavicular ribs and clavicle thicken from periostitis, and produce dulness under percussion, which cannot with positiveness be distinguished from

that of tubercle within the lung. Here the observer must wait for events to clear up the diagnosis" (*Diseases of the Lungs*, p. 233).

2. *The Occurrence of Gummatus Nodules in the Pulmonary Substance.*—These are, in the first instance, of the same histological constitution as the well-known node of the shin, or the subcutaneous product described by Ricord, Bärensprung, Virchow, and McCarthy. They form especially towards the periphery and bases of the lungs. In the former site they resemble nodules of lobular pneumonia. They may soften and be eliminated much in the manner of tubercle, although they may have at first a consistence like scirrhus. It is concerning those which soften in this way that Ricord gives the warning "not to confound suppuration of a few syphilitic nodules of the lungs with phthisis." Regarding those gummatus nodules Dr. Walshe observes: "I can find no positive answer to the query, Do these gummata ever form independently of other tertiary evidences of syphilis in the bones and cellular tissue? If they do, their diagnosis must be infinitely difficult—difficult, indeed, under all circumstances; for the physical signs can be none other than those of solidification, followed by softening and excavation, while the local and general symptoms closely simulate those of phthisis" (*Diseases of the Lungs*, p. 431).

3. *The Occurrence of Gummatus Nodules in various Stages of Growth and Degeneration, associated with the Miliary Deposit of Tubercle.*—In such cases the history of events in the illnesses of the patient may be found to correspond more or less closely with the appearances seen in the lungs—appearances which distinctly indicate the formation of lesions commenced at different dates—appearances which denote the occurrence of lesions in crops, or as a succession of events which may be illustrated by the history of the symptoms during life.

The minute structure of these *gummatus nodules* has been closely examined by many observers. They consist of a growth of elements which leads to the development of an elastic tumor composed of a well-defined tissue, and the elements of which are extremely minute. The tumor takes origin from the connective tissue, or the analogues of such; and hence the universality of the site of syphilitic lesions. When these are sufficiently large to attract attention—as in the form of a node on the shin-bone, or on some part of the true skin—they are small, solid, pale knots, like a hard kernel, about the size of a pea. They are generally first seen on some part of the true skin or subcutaneous or submucous tissue; and when the tissue in which they happen to grow is sufficiently lax, they grow to a considerable size, and convey to the touch a sensation as if they were filled with gum. Repeated examinations of this growth show that in its gelatinous or soft state it arises from a proliferation of nuclei amongst the elements of the connective tissue, not unlike the formation of granulations in a wound. The component cell-elements appear as round, oval, or oat-shaped particles embedded in a matrix of fine connective tissue of a granular character, and tending to fibrillation. The cell-elements are a little larger than blood-globules, and are distinctly granular in their interior when

mature. In the growing part of the node, and immediately in its vicinity, where growth is abnormally active, the minute cell-elements are seen to be developed in groups within the elongated and enlarged corpuscles of the connective tissue. In form, therefore, the node or gummatous nodule resembles a tubercle; and by fatty degeneration or tuberculization may not be capable eventually of being distinguished from tubercular deposit. How, then, are we to recognize the specific nature of such gummatous nodules? There is nothing in them so specifically and anatomically distinct that, apart from their history, they can be recognized. The history of the syphilitic case during life is the great guide. The nodes on the shin-bone or clavicles have long been recognized as the product of syphilis. It may almost be said that they have been seen to grow under the eyes of the patient and the observer; and their anatomical characters are found to be such as compose the gummatous nodules just described. In a case of inveterate syphilis, therefore, whose history is fully known, in whom the node on the shin or other bones is characteristic, and has been seen to grow, and in whom also we find similar nodules in the lungs, or in the liver, or in the testicles—symmetrically growing in these latter organs, and consisting of minute cell-elements exactly the same as the node on the shin—it is impossible to overlook the fact, or not to be impressed with the belief, that all of these lesions acknowledge one and the same cause of development—namely, the syphilitic poison—of which they are the expression. The progress of the node is also characteristic and suggestive. Growths of a similar form which result from idiopathic inflammation generally proceed to the formation of an abscess, or to the hypertrophy of fibrous tissue. Abscesses are recognized by their pus; fibrous tumors or hypertrophies, by the fibre-elements which compose them.

Growths of a form similar to the node, which result from cancer, are in general to be recognized by the juice expressed from them. In the gummatous nodule we have no juice, and the cell-elements seen in cancer are generally so diversified in their form and mode of growth as not to be easily mistaken. The gummatous nodule is uniform as to the size and form of its cell-elements, and forms a growth less highly supplied with bloodvessels than a cancer. Cancers tend to infiltrate and involve neighboring textures; the gummatous nodule remains isolated and distinct.

By way of elimination, therefore, and by duly observing the history of the case, we are generally able to recognize the nature of such growths, and to assign to them their proper place in pathology.

The gummatous nodule has now been recognized and described in almost all the solid viscera of the body. Symmetrical development is a most constant characteristic. If a node grows on one shin, it is probably also to be found advancing on the other; if found in one testicle, it is extremely probable that it will be seen in the same relative spot in the other. Numerous examples of this symmetrical development may be seen preserved in the Pathological Museum of the Army Medical Department at Netley. During the growth of the nodule, proliferation advances slowly, and a gluey-

like material forms, which constitutes the inner cell-material of the nodule. If near the surface, such a nodule is apt to melt down, soften, open, and ulcerate; and such a result seems to be associated with other evidence of active constitutional disease, such as exists with a predisposition to tubercle, or with its actual existence. The tumor, however, continues gelatinous and coherent if it is inclosed in a dense part, or is deeply seated, as in gummata of the periosteum, scalp, brain, liver, testicle, lungs, and heart, if constitutional disease remains latent or inactive. Fatty degeneration may also eventually occur in the gummatous nodule, and eventually lead to its absorption; or its absorption takes place as a natural process of cure, the changes of which are not exactly known. We know only that the node on the shin-bone not seldom disappears from view, and does not return.

For reasons already stated, phthisis must be regarded in many cases as the product of syphilis; and I would fully indorse the statement of Dr. Balfour, from what I have seen in the post-mortem rooms, when he says that a great cause of pulmonary disease among the Guards is the amount of syphilis which prevails amongst the men, which he has not the least doubt is a very fertile cause of its being called into active operation. The influence of syphilis on the health of the soldier is indeed powerful for evil throughout the whole army.

**Treatment.**—The treatment of such pulmonary lesions, when their nature is clearly established, must be guided by the rules already laid down for the treatment of syphilitic disease at p. 713, vol. i.

### GANGRENE OF THE LUNG.

LATIN Eq., *Gangræna*; FRENCH Eq., *Gangrène*; GERMAN Eq., *Brand*—  
SYN., *Gangrän*; ITALIAN Eq., *Gangrena*.

**Definition.**—*Disintegration and breaking up into fragments of the filamentous tissue of the lung. Every simple element of the tissue is so changed that neither blood-fibres nor epithelium can be recognized in a sound state. The texture becomes broken up, and shreds of fibrous tissue may here and there be distinguished, but the whole mass becomes converted into a heap of amorphous granular matter, of a yellowish-brown or black color, mingled with drops of oil. The tissue becomes soft and flaccid, in some parts perfectly liquescent, and generally emits a fetid smell.*

**Pathology.**—The comparative rarity, the almost invariable fatality, of pulmonary gangrene, obtains for it a melancholy interest whenever it occurs; and since Laennec directed attention to its peculiar characters, many pathologists have contributed to the records of this disease. But notwithstanding the researches of Andral (1822), of Lorimer, of Schroeder Van der Kolk (1826), of Bright (1827), of Cruveilhier (1833), of Guislain (1836), of Craigie (1841), of Ernest Boudet (1843), of Silfverberg (1856), of Walshe (1860), of Fuller (1862), gangrene of the lung is still a condition at all times difficult of diagnosis in the first instance; and its existence has often been

unknown until the disgusting post-mortem appearances proclaimed the condition, which general symptoms during life had failed to disclose, and which in some cases hardly even suggested a suspicion of the real nature of the affection.

The diagnostic symptoms which have been most frequently recorded are derived from the expression of the countenance, becoming small, pinched, contracted, haggard, ghastly, miserable, and death-like; eyes sunk and void of lustre; patient squeamish and languid, with occasional vomiting, and a feeling of indifference to all external objects,—some or all of which symptoms may or may not be associated with an intolerable fetor of the breath, and which, when it is present with these symptoms, may be considered conclusive of the existence of gangrene. Not one of all these symptoms, however, may manifest themselves, and yet gangrene of the lung may exist. From the recorded cases, therefore, as well as from those which I have myself seen, it may be of some use to classify the conditions of disease in which pulmonary gangrene has occurred.

The termination of acute sthenic pneumonia, in an otherwise healthy person, by gangrene of the lung, is, by all authors, considered as one of the least frequent terminations of that disease. Morgagni only records one instance; and Laennec is reported to have seen only six or eight during the whole course of his practice. But there can be no doubt that pneumonia does sometimes terminate in gangrene of the lung, as an accidental or occasional complication, as has been clearly shown by clinical and post-mortem evidence. The insidious and often sudden mode of its attack, the sudden, remarkable, and generally fatal collapse which supervenes, show, at the same time, that no local condition is suspected or considered sufficient to account for the presence of gangrene. The extent to which the death and destruction of lung-tissue takes place also varies much. Sometimes nearly the whole of a lung passes at once into a gangrenous mass. In other cases only a small portion in the centre of an exudation will die; but in every case there is the formation of a slough, its liquefaction, and the formation of a cavity. Sometimes a line of separation between dead and living parts is attempted to be formed; while in other cases no evidence exists of any limit to the extent of the destruction; and this latter diffuse form of gangrene I have generally found associated with cases in which the blood was greatly changed, as in typhus fever, or in which the nervous functions were impaired, as in maniacs, or in the paralysis of the insane. Of fifteen cases recorded by Guislain, all were maniacs. Of three cases recorded by Van der Kolk, one was a maniac and two labored under some peculiar nervous condition of the body. Of six cases recorded by Dr. Craigie, two were mentally deranged, having lesions in the brain; two suffered from *Bright's disease* and *mercurialism*; one had variola; and one had typhus. Of fifteen cases in the Royal Infirmary of Edinburgh when I was a student there, four had typhus; three had fever, the type of which is not specified; four suffered from ill-health and mercurialism; one suffered from a blow; and one had a lesion in the brain.



These cases may be classified as follows:

*Nineteen cases* were associated with, and doubtless influenced by, lesions in the great nervous centres; and under this class are included the insane.

*Nineteen cases* were associated with, and doubtless influenced by, such morbid conditions of the blood as exist in *typhus fever*, *variola*, *tuberculosis*, *Bright's disease*, *mercurialism*.

*One case* was associated with and influenced by arterial obstruction, causing pressure upon the entire mass of the affected parts, and total absence of circulation in them (Aitken, *Edin. Med. and Surg. Journal*, No. 178, 1848).

The frequency with which lesions of the lung coexist with and succeed to lesion of the brain renders it apparent that the condition of the lung is materially affected by the influence of the nervous centres. In the cases recorded by Andral and Bright, diseases of the lung, frequently going on to gangrene, were often the immediate cause of death in persons suffering from cerebral disease; and it was also observed that the tendency of inflammation of the lung to pass into gangrene was prompted by the existence of disease of the brain. Cruveilhier also directed attention to the frequency of gangrene of the lung in epileptic cases; and the insane generally are prone to the disease when the bodily health has suffered, or where, as in maniacs, a greatly depressed state of the animal functions succeeds to inordinate nervous excitement.

The following table, modified from that by Dr. Walshe, describes the conditions under which gangrene of the lung occurs:

I.	{	<i>Pneumonia</i> , acute and chronic; tuberculization; cancer; hemorrhage; hydatids (WALSHE); apoplectic foci in the lung; acute and chronic dilatation of the bronchial tubes; pleuritis; bronchitis (SILFVERBERG).
Pulmonary diseases.		
II.	{	<i>Cardiac</i> : acute endocarditis of the right side of the heart. <i>Mediastinal</i> : tumor. <i>Aortic</i> : aneurism.
Other thoracic diseases.		
III.	{	<i>Animal venoms</i> : stings of certain insects. <i>Morbid poisons</i> : { Glanders; exanthemata; typhoid fever; Bright's disease; alcoholism. Scurvy; purpura; septicæmia; pyæmia. <i>Other poisonous agents</i> : poisonous gases; mercurialism.
Diseases in which the blood is materially changed.		
IV.		
Chronic abdominal disease (SILFVERBERG).		
V.	{	Epilepsy; insanity; organic cerebral disease.
Perverted innervation.		
VI.		
Traumatic.		

The disease may occur at any age; but it appears to be more common in children than in adults; and more so in adults than in persons much advanced in life.

The symptoms vary according as the lesion is *diffused* or *circumscribed*: (1.) In the diffused form the symptoms and progress of the case are extremely rapid. Utter prostration of strength; oppressed breathing; a small, weak, and frequent pulse; pallor and anxiety of countenance; and all the general appearances of intense adynamia, are those which characterize the disease. There is also fre-

quent but feeble cough, with profuse, frothy, and diffuent expectoration, of a peculiar greenish color and intensely fetid gangrenous odor. But the power to expectorate is soon lost; and ere long the vital powers seem utterly oppressed or exhausted; the pulse fails, the features collapse, and the patient rapidly sinks, death generally occurring from apnoea. (2.) In the *circumscribed* form of gangrene the train of symptoms varies greatly at different periods of the disease. At first, signs of pulmonary congestion are coupled with an amount of prostration quite out of proportion with the apparent extent of local lesion—a prostration which is, indeed, sometimes the only prominent feature in the case. After a time expectoration commences; at first muco-purulent, rarely bloody in adults, frequently so in infants and children (if they expectorate at all), and generally emitting a disagreeable odor the moment a communication is established between the bronchial tubes and the dead tissue. The sputa then lose their muco-purulent character, and become extremely liquid, sero-purulent, and of a dirty greenish, yellowish-brown, or ash-gray color. They exhale an odor distinctly gangrenous, while the breath acquires the same offensive putrid smell, resembling somewhat that of wet mortar, or its feter is one *sui generis*. The pulse becomes feeble and rapid, with every evidence of great and increasing prostration. The patient passes rapidly into a state of collapse, and sinks in a few hours or days, without the occurrence of any other change, unless profuse hemorrhage occurs, which terminates in death (WALSHE, FULLER).

**Treatment.**—The chief reliance is to be placed in all forms of stimulants, combined with such tonics as *bark* or *quinine* in full and repeated doses. Dr. Walshe recommends that repeated doses of an ounce of yeast are deserving of investigation, as well as the influence of *chlorate of potass*. He is disposed to put more confidence in them than in ammonia, the beneficial effects of which he has never seen demonstrated. Dr. Fuller has not found ammonia so useful as the mineral acids. Inhalation of the vapor of turpentine poured upon boiling water, as recommended by Skoda, is reported to exercise a distinctly remedial power. The *mineral acids*, especially *nitro-muriatic*, combined with *quinine*, are the main remedies in the chronic state of this disease. A generous diet, easily digestible, with as much malt liquor as can be taken, are recommended as most suitable for such cases.

#### SPITTING OF BLOOD, PULMONARY HEMORRHAGE— Syn., HÆMOPTYSIS.

**Definition.**—*The discharge of blood by expectoration.*

**Pathology.**—The amount expectorated may be small—mere specks or streaks on the sputa; at other times a few ounces, or even pounds, causing great alarm to the patient or his friends, and sometimes suffocating the patient. Its occurrence is always suggestive of organic disease of the chest, such as engorgement of the pulmonary vessels, giving way of pulmonary tissue, indicating disease of the heart, or pulmonary tuberculosis or pneumonia, or thoracic aneurism,

or cancer of the lung. Cases are also recorded of its so-called idiopathic occurrence, as from variations (sudden) of atmospheric pressure, ascending high mountains, or descending in diving-bells, violent straining efforts, or from plethora ; but in such cases, according to the experience of Drs. Fuller, Walshe, and others, "there is usually some latent mischief in the chest—some local cause of pulmonary congestion—some mechanical interference with the capillary circulation through the lungs." Spitting of blood may even precede for years the fatal development of tuberculous disease, with which it seems to be most prevalently connected, and is often hereditary.

**Symptoms.**—Hæmoptysis may take place suddenly, or be preceded by a sense of heat or a feeling of weight at the chest, or the patient may suffer pain between the back and shoulders, or may labor under dyspnœa, palpitation, cough, or coldness of the extremities ; and these symptoms may last two or three days. At length a fit of coughing, or a tickling of the throat, is followed by the appearance of expectoration of arterial, but more often of venous blood. Laennec says he has seen as much as ten pints thrown up in forty-eight hours, and as much as thirty pints in a fortnight. The effort of coughing also often causes vomiting, so that the blood discharged is frequently mixed with alimentary matters.

If the quantity thrown up be inconsiderable, the patient's previous health is in no degree affected ; but if it be large, its effects are strongly marked, for the patient feels oppressed at the præcordia, breathes with difficulty, and with a gurgling sound, caused by the air passing through the viscid blood retained in the bronchi ; and this is shortly followed by increasing weakness, even to complete prostration. In still severer cases, as the blood flows, the patient turns pale, his countenance becomes œdematous, and strongly expressive of terror, or else he falls into a complete syncope, which sometimes has a curative effect. In a very few instances the effusion is so sudden and so considerable that the patient dies suffocated.

Although bronchial hemorrhage may be considerable, it often diminishes rapidly, so that at the end of some hours only a few rare isolated sputa are spat up, and at considerable intervals. Usually, however, the hæmoptysis recurs after a greater or less length of time, but not perhaps to the extent of the primary attack. After the patient has lain for a time in a state of depression, a reaction takes place. In sthenic persons the appetite becomes increased, they enjoy everything they are allowed to eat, and after some slight febrile action they rapidly recover. In the fatal cases the pulse becomes rapid, the tongue brown and dry, and the patient sinks.

**Diagnosis.**—The only disease which it is important to distinguish from *hæmoptysis* is *hæmatemesis*, and the diagnosis between them is difficult, because while the contents of the stomach are always rejected in *hæmatemesis*, they are frequently rejected in *hæmoptysis* also. The stethoscope, however, greatly assists in determining the seat of the disease ; and again, blood is generally found in the stools in cases of *hæmatemesis*, while it is for the most part wanting in *hæmoptysis*.

**Prognosis.**—The prognosis is always unfavorable eventually, so far

as freedom from organic disease is concerned, and danger is more or less directly imminent in proportion to the amount of blood lost.

**Treatment.**—The medicines most useful in *hæmoptysis* are the *bitartrate of potash*, in doses of a drachm, repeated every four or six hours, and to each dose of which may be added a quarter to half a grain of *opium*. The *mineral acids*, as the *infusion of roses* with diluted *sulphuric acid*, in doses of from three to five drops combined with *opium* or *morphia*, every four or six hours; larger doses of the *dilute sulphuric acid*, have often been tried, but are apt to be either rejected or to act injuriously on the coats of the stomach. Many practitioners use one- to three-grain doses of the *acetate of lead* every four or six hours, with *half a grain of opium* to each dose, or combined with *dilute acetic acid* and *laudanum* (A. T. THOMPSON); and, according to Andral, when the system has long been under the influence of lead, the red globules suffer a great diminution; but, nevertheless, this is certainly a less efficacious medicine than either of the preceding ones. The *nitrate of potash* has been much used in France, but Gendrin has not found it efficient, or not more so than any other diuretic. The *muriate of soda* in doses of half a drachm to a drachm is in estimation with some practitioners on the Continent.

When *hæmoptysis* is connected with *amenorrhœa*, preparations of iron often succeed when the above remedies have failed. Two grains of the *sulphate of iron*, with one-drachm doses of the *sulphate of magnesia*, three times a day, often restore the menstrual secretion and cure the *hæmoptysis*. Indeed, it is in this form of *amenorrhœa* that iron is most successful.

When *hæmoptysis* depends on disease of the heart, cupping from the chest, or moderate bleeding from the arm, is often efficacious, combined with the use of the *bitartrate of potash* or the mineral acids, to which should be added five to ten minims of the *tincture of digitalis*. It is in many cases proper to add half a drachm to a drachm of the *spirit of nitrous ether* to each dose, to give steadiness to the irregular, turbulent, or rolling action of the heart.

Dr. Fuller's experience leads him to testify most strongly in favor of repeated dry cupping, aided by the application of ice down the spine, and by the internal administration of full doses of *digitalis* (ʒj to ʒiij of the tincture; or gr. vj to gr. viij of the powder).

If these remedies fail, full and frequent doses of *gallic acid*, or *lead and opium*, may be given, if the circulation is much accelerated, and of *spirits of turpentine* in half-drachm doses, if the bleeding is unattended with vascular excitement. The *gallic acid* should be given every hour in *eight- or ten-grain doses*, until the hemorrhage is subdued, or till a dark green color in the sputa indicates its action in the system (*op. cit.*, p. 265).

Absolute bodily and mental rest must be insisted on during convalescence.

## CHAPTER XI.

DISEASES OF THE MOUTH, TONGUE, FAUCES, PHARYNX, LARYNX,  
AND OESOPHAGUS.SECTION I.—AIDS TO THE DIAGNOSIS OF DISEASES OF THE MOUTH,  
FAUCES, PHARYNX, AND LARYNX.

THE principle of the speculum in use by dentists, and first used by Robert Liston, the eminent British surgeon, more than twenty years ago, in the diagnosis of an "ulcerated glottis," has greatly

FIG. 40.\*



advanced our knowledge of the lesion of parts otherwise beyond our vision. Science is indebted especially to Garcia, Czermack, and

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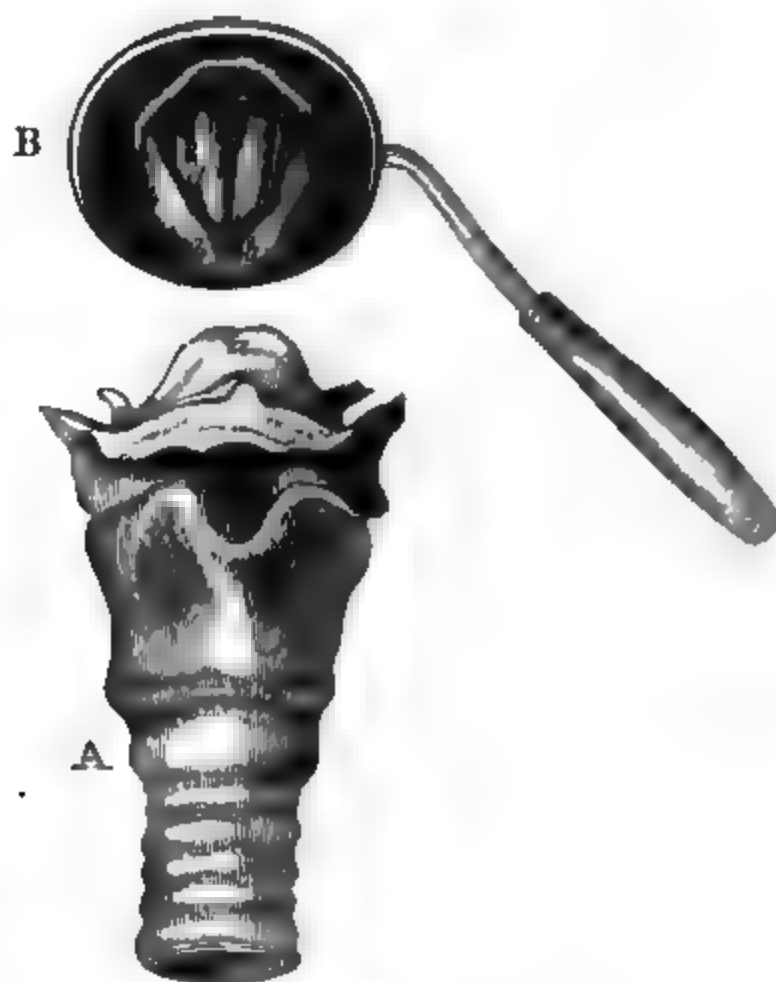
\* Laryngoscopic examination of a patient (after Dr. George Johnson).—The illumination of the pharynx is effected by light cast into the patient's mouth from a mirror over the brow of the operator. Dr. Johnson has recently suggested that the operator should also use a shade to shield his eyes from the glare of light (*Lancet*, August 26, 1865).



Turck, in Germany, and to Drs. Morell Mackenzie, Gibb, Sieveking, Johnson, and Walker, in this country, for promoting and making known the practical application of the laryngoscope.

The Laryngoscope consists of a small flat mirror with a long stem, which, being previously warmed, to prevent the breath condensing upon it, is introduced into the widely open mouth, as far back as its back part. In the application of the instrument the first great difficulty to overcome is the illumination of the pharynx; the second difficulty is to learn to manipulate the mirror in such a way as to display the larynx, or any part it is desired to see, to the best advantage (Fig. 40). It must be remembered that the image of the parts is reflected to the eye of the observer in an *inverted position*, so that the right vocal cord appears on the left side, just as

FIG. 41.\*



the right arm appears on the left in a looking-glass. Experience will soon enable the observer to correct this falsity of representation, and he will do this quite unconscious of any mental effort (Fig. 40). By means of this instrument the deep portion of the pharynx, the larynx, the passage through the glottis, the mucous membrane of the trachea, are all capable of being seen. The epiglottis and posterior aspect of the base of the tongue, as well as the

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\* A. Anterior view of the larynx.—(a.) Epiglottis. B. Opening of larynx, as seen inverted in the mirror.—(a.) Epiglottis; (b, b'.) Arytenoid cartilages; (r and l.) Right and left vocal cords (after T. J. WALKER).

posterior aspect of the pillars of the *fauces*, and the condition of the *nares*, are all capable of being seen in this way, as represented in the following wood-cuts, Figs. 42, 43, 44, 45 :\*

The instrument is absolutely necessary, not only for precision of diagnosis, but for precision in the local appliances in the treatment of diseases of these parts. The application of *glycerine*, of *nitrate*

\* A, B, C, D, E, represent the arrangement of the internal parts of the larynx of Dr. Czermack, after drawings made by Dr. Elfinger, of Vienna, from images demonstrated in the laryngoscope (*New Syden. Soc. Year-Book*, 1861). The parts situated to the right of the middle line of these figures, obtained by the aid of a mirror, are necessarily reversed from their natural position, and correspond to those on the left side of the larynx, and *vice versa*. That which is situated above in the drawing exists in front; that which is below is situated posteriorly.

THE SERIES OF WOOD-CUTS SHOW THE LARYNX FROM MODERATE CLOSURE TO FULL DILATATION.

A. The beginning of hermetic closure of the larynx—commencement of act of swallowing. The cushion of the glottis (*e, w*) applies itself over a considerable portion of the closed glottis, and of the narrowed false glottis.

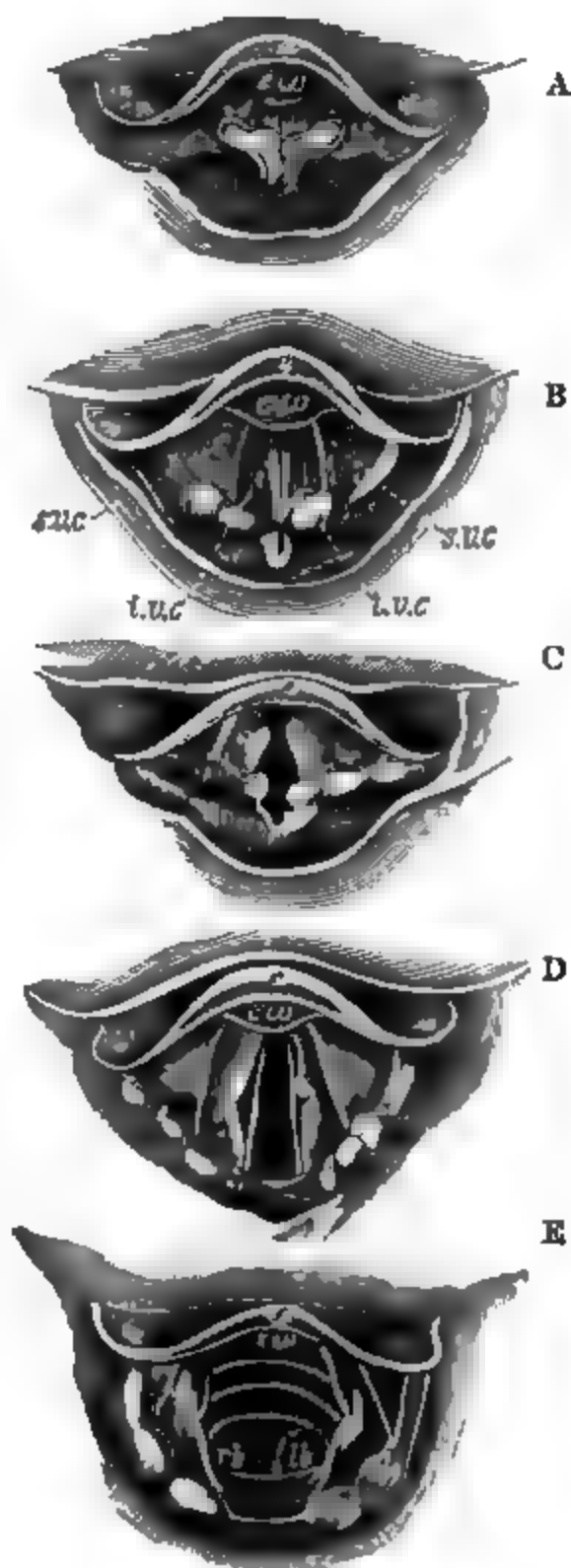
B. Condition of the interior of the larynx during the production of most acute sounds. The glottis is contracted in a linear form; and on each side are the (inferior) vocal cords (*i, v, c*), distinguished by their whitish-yellow color. A little to the outer side is a narrow groove, which indicates the ventricles of Morgagni. Farther outwards are the false or superior vocal cords (*s, v, c*).

C. Constriction of the glottis, erroneously called *respiratory glottis*, the arytenoid processes converging, and causing the posterior section of the glottis to be almost triangular.

D. Complete relaxation of all the parts, as when the glottis under ordinary circumstances is open for breathing.

E. Bifurcation of trachea and commencement of bronchial tubes, seen on widely opening the glottis, as in deep inspiration, and straightening the trachea.

FIG. 42.



of silver, and other agents, can thus be effected with perfect accuracy to a part. The reader is referred to the papers of Dr. Morell Mackenzie in the *Medical Times* for April, 1842; to those of Dr. Sieveking in the *British Medical Journal* for December 20, 1862; and to those of Dr. T. J. Walker in the same journal for February 28, 1863, *et seq.*, and to the separate monographs by these writers, for an account of the methods of using the instrument.

FIG. 48.

APPEARANCES OF THE LARYNX AS ALTERED  
BY DISEASE (after CZERMACK):

F



F. Condylomata of the larynx, believed to be the result of syphilis. Conical excrescences on inferior vocal cord, with thickening, causing hoarseness.

G



G. Polypus attached to the right vocal cord, the real cause of a supposed venous aphonia.

H



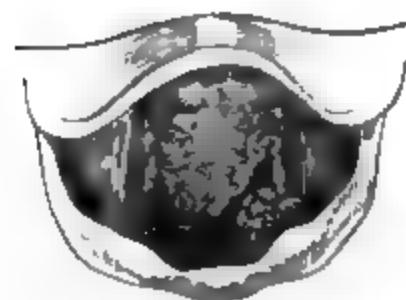
H. Transformation of the right (superior) false vocal cord into a hard, rough, and ulcerated mass.

K



K. Cicatricial contraction and loss of substance of the larynx, with destruction of epiglottis from old syphilitic disease.

L



L. Very large muciform polypus, of an epithelial character, developed within and above the glottis—aphonia of four months.

## SECTION II.—DETAILED DESCRIPTION OF THE DISEASES OF THE MOUTH, TONGUE, FAUCES, PHARYNX, LARYNX, AND ŒSOPHAGUS.

### INFLAMMATION OF THE MOUTH—*Stn*, STOMATITIS.

The mouth is specially liable to various forms of inflammation in children. They have received the name of "stomatitis." These inflammations are expressed as follow: (1.) *Simple erythema*; (2.) Diphtheritic exudation or eruption; (3.) Inflammation of the follicles of the mucous membrane—*follicular stomatitis*.

FIG 44.

#### INSPECTION OF THE NARES.—*Rhinoscopy.*

Posterior view of uvula, soft palate, and nares, as seen in the mirror (directing it upwards in place of downwards). The tumor (a.) on the left side of the figure fills up the posterior opening of the right nasal fossa; (c.) The orifice of the right Eustachian tube (after DR. GEORGE JOHNSON).

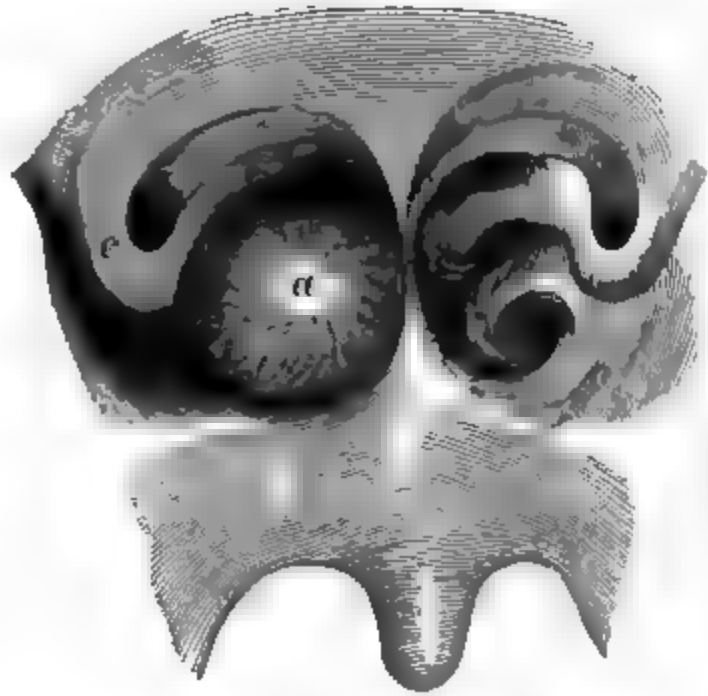


FIG. 45.

Posterior view of the parts after removal of the tumor (a.) The abraded surface from which the tumor was torn; (b.) The middle; and (c.) The inferior turbinated bones; (e.) The Eustachian tube (after DR. GEORGE JOHNSON).



[These drawings, illustrative of the larynx and laryngoscopy, were kindly made by Staff Assistant-Surgeon Dr. Humphrey C. Gillespie.]

*Simple Erythema* occurs in patches, and is generally due to hot or acrid substances taken into the mouth, to cold, or the irritation of the teeth, or of tartar upon them. It may be also due to gastric derangement. Small doses of *magnesia*, or of *rhubarb with soda*, will generally correct the stomach derangement, when the stomatitis will subside.

*Diphtheria of the Mouth.*—*Thrush*, or *Muguet*—is a pultaceous product of inflammation, in which the secretion of the mouth is greatly altered and increased. The disease chiefly attacks the new-born infant. The whole surface of the mouth exhibits unusual redness, with here and there curd-like exudation in irregular patches, preceded by vesicles, especially behind the lips and about the tip of the tongue. These patches are thrown off, to be renewed again; and the mucous membrane below is of a bright red color. The adjacent glands are apt to become tumid and tender. The skin is commonly hot and dry; thirst is considerable; swallowing seems to give pain; and diarrhœa may ensue to a degree which soon proves fatal in an infant. The disease frequently occurs as a sequela of measles.

**Treatment.**—The mouth should be frequently washed with emollient fluids, such as *linseed infusion*, *diluted glycerine*, and *biborate of soda*, or *honey mixed with biborate of soda*.

Dr. Tanner recommends the following lotion:

R. Sodæ Biboratis, ℥j; Glycerinii, ℥ij; Aquæ Rosæ, ℥iv; misce. To be painted over the lips and mucous membrane of the mouth and tongue.

*Creasote*, *vinegar*, *carbolic acid*, *glycerine*, and *alcohol* are also recommended as local applications. In severe cases, where the breath becomes fetid, with the submaxillary glands large and swollen, the lips and gums tumid, the face flushed and swollen, and the fever intense, *chlorate of potash* must be freely given, to the extent of *five grains* every four to six hours.

*Follicular Inflammation of the Mouth*, *Follicular Stomatitis*, *Aphthous Stomatitis* or *Aphthæ of the Mouth*.—This disease usually commences as a simple stomatitis; but very soon, small, round, transparent, grayish or white vesicles appear, and at the base of each is an elevated marginal ring, which is pale and firm. Fluid soon escapes from the ruptured vesicle; an ulcer forms, which spreads, bounded by a red circle and an elevated border. In some forms of the affection microscopical parasitic plants occur.

**Treatment.**—Besides the remedies already recommended, it will be found that the solution of the *pernitrate* of iron, internally, has a beneficial effect upon the sores. It is prescribed in the following formula (DUNGLISON):

R. Liq. Ferri Pernitratis, gtt. xl; Syrup. Aurant., ℥ss.; Aquæ, ℥vss.; misce. A fourth part may be given to a child three or four years of age four times a day.

In cases where parasitic vegetable productions abound, the application of a solution of *sulphite of soda* (℥j to ℥j of water) removes



the lesions in twenty-four hours (JENNER). The secretions of the mouth being acid, the salt is decomposed, and sulphurous acid is set free, which destroys the parasite. A change of air is often absolutely necessary to restore the patient to health; and arsenic and iodine are useful restorative agents in repairing, with good diet, the faulty nutrition of the child.

*Ulcerative and Gangrenous Inflammation of the Mouth, or Noma and Cancrum oris.*—These are but different grades of similar lesions. The *noma* or milder form, generally commences at the edges of the gums, opposite the incisors of the lower jaw. At these points the gums appear white, become spongy, and separate from the teeth, as if mercury had produced its specific effects. Ulceration begins, and extends along the gums until the jaws are implicated; and as the disease advances, the cheeks and lips begin to swell, so as to form a tense indurated tumefaction. The teeth are apt to fall out; and the gums assuming a gangrenous condition, the breath becomes intolerably fetid. There is generally enlargement with tenderness of the submaxillary glands.

The more severe form (*cancrum oris*) occurs in children of debilitated habits, between *two* and *five* years of age especially. A copious flow of saliva, a tumid appearance of a cheek, with fetor of breath, ought to suggest an examination of the mouth, when small vesicles of a grayish-red or even black appearance may be seen on the inside of the lips or the tumid cheek. These vesicles are surrounded by a red base; and swelling, surrounding hardness, heat, and pain increase. An ash-colored eschar may then appear in the centre of the cheek, within the cavity of the mouth, surrounded by a glossy tumefaction of the parts; and on the inside of the cheek a hard indolent swelling. So rapid may be the spread of this destructive disease, that in a few days the lips, cheeks, tonsils, palate, tongue, and even half the face, may become gangrenous, the teeth falling from their sockets, a horribly fetid saliva and fluid flowing from the parts.

**Treatment.**—Analogous to hospital gangrene, an early recognition of the nature of the disease will suggest an efficient application of *strong nitric acid* to the slough. The mouth must be frequently syringed with a solution of *carbolic acid*, in the proportion of *half a drachm dissolved in a gallon of boiling water, and allowed to become warm or tepid*. Tonics and antiseptics must be freely given. *Beef tea, wine, brandy, quinine, and chlorate of potash*, are the remedies indicated.

#### GLOSSITIS.

LATIN Eq., *Glossitis*; FRENCH Eq., *Glossite*; GERMAN Eq., *Zungenentzündung*; ITALIAN Eq., *Glossitide*.

**Definition.**—*Inflammation of the tongue.*

**Pathology.**—*The tongue is liable to acute inflammation of its substance generally, to various forms of ulcers, and to cancer.*

*Inflammation of the Tongue, or Glossitis*, may result from acrid substances taken into the mouth, or from the specific action of

*mercury*, or *scarlet fever*, or *small-pox*. There is generally great tumefaction from infiltration of serum; while fever, mental depression, and general weakness prevail, with pain and heat of the tongue, the color of which is of a deeper red than usual. The swelling may be so great as to cause the tongue to project beyond the teeth, or even to project so far back as to cause dyspnoea. Dr. Graves relates a case of inflammation affecting one half the tongue, the median line forming the boundary between the swollen and the healthy parts.

**Treatment.**—Active cathartics are generally of great service; and they are to be given as enemata. Blood must be taken directly from the tongue in such cases. Incisions along the inflamed organ, followed by the action of the vapor of hot water, may reduce the swelling and relieve congestion. In Dr. Graves's case, two or three applications of six leeches at a time to the inflamed parts produced a speedy decrease of the tumor. If suffocation is imminent, tracheotomy or laryngotomy may be performed.

*Ulcers of the Tongue* are of various kinds: (1.) Simple ulcers are usually situated about the tip, near the frænum, or at the edges; (2.) Ulcers resulting from the action of mercury are usually associated with similar ulcerations of the gums and mercurial fetor of the breath; (3.) Syphilitic ulcers and eruptions are generally superficial—often presenting (a.) A glossy *eczematous* or *psoriasis-like* patch, which creeps from place to place, and which heals and breaks out again repeatedly. (b.) Sometimes an inelastic induration or node will form on the upper back part of the tongue, often in such a position facing the epiglottis that the mirror or speculum is absolutely necessary to see the lesion. (Several such lesions are to be seen in the Museum of the Army Medical Department.) These nodes generally commence to slough in the centre, having ragged, thickened, and hard edges. (c.) Other forms of syphilitic lesion of the tongue are seen in long fissures or cracks, with enormous hypertrophy of the papillæ along the edges of the fissure, forming warty condylomatous excrescences. Primary chancres sometimes exist.

**Treatment.**—Simple ulcers soon heal under the influence of water simply, or borax solutions frequently taken into the mouth as a gargle. Ulcers from mercury heal best under the use of *sulphate of copper* or other astringent gargles. Syphilitic nodes of the tongue may yield to calomel and opium, or to opium and gray powder, in small doses every night—constitutional treatment by diet being attended to at the same time.

## TONSILLITIS.

LATIN Eq., *Inflammatio tonsillarum*; FRENCH Eq., *Amygdalite*; GERMAN Eq., *Entzündung der Mandeln*; ITALIAN Eq., *Tonsillitide*.

**Definition.**—*Inflammation of the tonsils.*

**Pathology.**—This disease is indicated by redness of the tonsils, which are loaded with blood, and moderately swollen. The *uvula* generally participates in the morbid action. It becomes elongated,

and rests on the base of the tongue, causing a most disagreeable sense of titillation. Lymph or serum may be thrown out, and in this case the tonsils are often greatly swollen, so as in some instances almost to block up the passage of the fauces. The diagnostic symptom between the effusion of serum and of lymph is that, in the latter case, the tonsil remains often permanently enlarged. In a few instances an abscess forms in the tonsils, which ultimately ruptures, and discharges a greater or less quantity of pus. It generally happens that both tonsils are affected, but occasionally the inflammation is limited to one.

In chronic inflammation of the tonsil the same phenomena are seen, but the course of the disease is slower, and the color of the parts less vivid, and in general differing little from their natural tint. The distended gland-vesicles, filled with their secretion, distend the follicles, so that the tonsils appear dotted over with little abscesses. These gland-cavities eventually rupture, and discharge more or less consistent cheesy-like pellets, having an excessive fetid, fecal-like odor.

The inflammation of the fauces, whether acute or chronic, not unfrequently extends to the pharynx. The inflammation may commence in the subcellular pharyngeal membrane, and an abscess occasionally forms in that part. In a smaller number of cases, by an extension of the original disease, the *epiglottis*, *glottis*, and even the *larynx* are affected. In bad cases, as after severe scarlatina, it may spread to the Eustachian tube, and cause suppurative or other inflammation of the mucous membrane of the internal ear. It sometimes extends up the nasal passages, by which respiration through those passages is much impeded or rendered impossible, so that the patient breathes with his mouth open.

**Symptoms.**—The different degrees of intensity which attend this affection have suggested a division of sore throat into *angina mitior* and into *angina gravior*.

*Angina gravior* is usually preceded by some shivering and fever, which having lasted a few hours, the patient has the sensation of a sore throat. [The temperature for a day or two is quite high, rising to  $102.6^{\circ}$ , with a pulse of 104 or 102, and continuing about  $99^{\circ}$  or  $100^{\circ}$  for three or four days, then falling to the natural standard, or below  $97.4^{\circ}$ .] He finds deglutition difficult and painful, and what he attempts to swallow is perhaps rejected through the nostrils; his voice is altered, being hoarse and nasal, and he can hardly breathe through his nose; his ears are also painful, and he finds it troublesome to free his throat from the viscid matters which adhere to it. Fever does not abate on the appearance of the local affections, but continues till the abscess of the tonsil bursts, or till resolution is complete. The degree of prostration which attends *tonsillitis* is usually out of all proportion to the severity of the local lesion. It generally subsides in three or four days, and rarely lasts longer than a week. To inspect the throat well, the patient should be directed to open his mouth widely. The dorsum of the tongue must then be depressed by some flat body, such as the handle of a spoon, the patient being made to inhale a

full breath. The tonsils can then generally be seen without much difficulty.

**Treatment.**—In the early stage cathartics are very useful, with very hot foot baths, containing mustard. Emollient gargles, with the inhalation of steam, should be applied to the throat. Dover's powder, to the extent of three grains every four or six hours, with warm diluents, often greatly subdue the febrile state; but the effects of the opium must be watched, so as not to be carried too far. The gum resin of *guaiacum*, in the following formula, is most efficient in subduing incipient *tonsillitis*:

R. Magnes. Sulph., ℥vj; solve in Aqua, ℥viij; adde Pulv. Guaiac., ℥iss.; Pulv. g. Tragacanth. Co., ℥ij; misce bene. A sixth part every four hours till the bowels are freely opened.

When the inflammation advances to suppuration, it may be necessary to open the abscess; but generally it bursts of itself, during some effort of the parts, in deglutition. If a puncture is necessary, its direction must be towards the middle line.

### PHARYNGITIS.

LATIN EQ., *Pharyngitis*; FRENCH EQ., *Pharyngite*; GERMAN EQ., *Schlundentzündung*; ITALIAN EQ., *Faringitide*.

**Definition.**—*Follicular inflammation of the pharynx.*

**Pathology.**—Is indicated by huskiness of the voice, with more or less coughing, hawking, or spitting. The follicles of the *isthmus faucium* and of the pharynx are unusually apparent; and the mucous membrane seems studded with granulations, probably due to an accumulation of secretion in the follicles. Occasionally the follicles burst, and discharge small masses, of an elastic consistent nature, causing great anxiety to nervous patients. Ulceration may then supervene (GREEN, DUNGLISON). The patient at first feels an uneasy sensation in the throat, with a constant inclination to swallow, as if an obstacle existed in the œsophagus. It is a disease apt to occur amongst clergymen, and men who have much speaking in public, and has been known as "clergyman's sore throat."

**Treatment.**—Tonics, such as *iron*, *quinine* and *strychnia* are mainly indicated. The local applications most useful are solutions of *nitrate of silver*, or *creasote* solution. The nitrate of silver is used in the proportion of *two to four scruples to the ounce* of water, applied by a *probang*, about ten inches long, to the affected mucous surfaces. Time is a most important element in the cure; for the disease is essentially chronic in character.

## [TREATMENT OF DISEASES OF THE PHARYNX AND OF THE RESPIRATORY ORGANS BY ATOMIZED FLUIDS.]

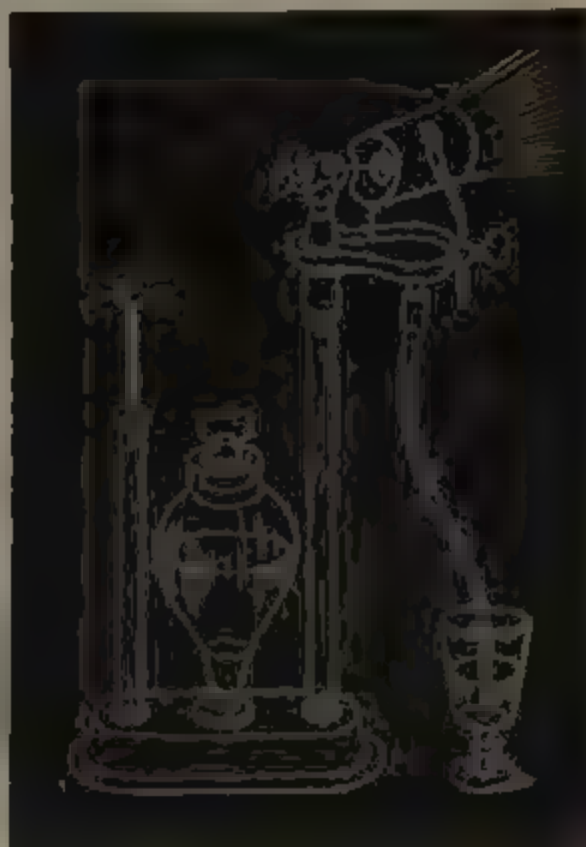
(Dr CLYMER.)

Medicated vapors and gases have been employed from very early times, at different periods, in the treatment of diseases of the pharynx and of the respiratory organs. The Grecian, Roman, and Arabian physicians seem to have recommended their inhalation in these affections. In 1849, Auphan, of Enzet-les-Bains, proposed to atomize, or pulverize, mineral water, by throwing forcibly a jet of it against the wall of a room, converted by this means into a fine spray, it was thus scattered through the atmosphere of the apartment, and inhaled by the patient. Soon afterwards Sales-Girons and Flube constructed an apparatus at Pierrefonds, by which mineral waters, simple or medicated, were converted into fine

FIG. 46 \*



FIG. 47 †



mist in a room, which was breathed by a number of persons at the same time. The water was forced through a tube, ending in several capillary openings, by an air-pump in an adjoining room, against the surface of a

\* Original Portable Apparatus of Sales-Girons and Charrière (DA COSTA).

† Sales-Girons Improved Atomizer (DA COSTA).

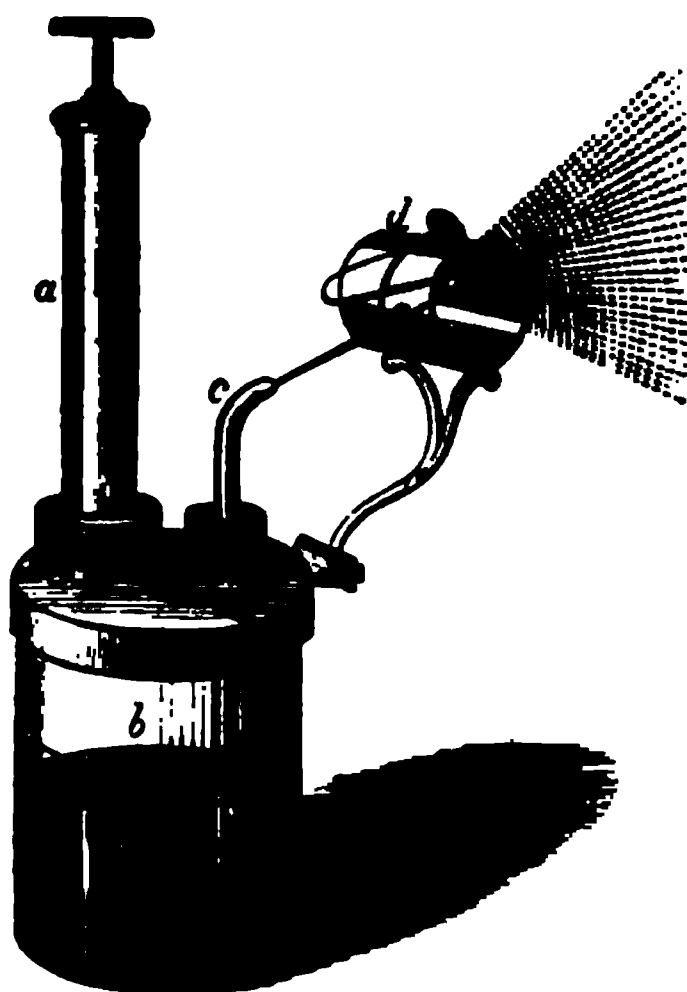


metallic disc. This method proved inconvenient from dampness. After several experiments, Sales-Girons and the late Mr. Charrière of Paris, invented a portable apparatus, by which fluids could be atomized, thus placing in the hands of physicians a means of trying this plan of treatment of the diseases of the air-passages.

The original apparatus of Sales-Girons and Charrière (1859), (Fig. 46), consists of a vessel, first made of metal and afterwards of glass, containing the fluid to be used, and an air-pump placed above it, which compresses the air on the surface of the fluid, driving it through a very fine opening, furnished with a stop-cock, against a small metallic disc, forming a minute spray or water-dust; the condensed fluid passing off through a flexible tube into a receiver. The amount of pressure, from three to five atmospheres, is shown by a manometer. This apparatus was, several years later, much simplified and improved by the inventors. As now made, there is a strong glass bottle holding the fluid, and a pump which drives it through a capillary opening in an ingeniously arranged stop-cock against a convex disc, by which it is minutely divided, producing a very fine spray, which is breathed by the patient, who sits before it (Fig. 47).

Lewin's nebulizer (Fig. 48), has a **strong glass reservoir, holding one-fourth of a gallon, and graduated in ounces.** In the metal cup there are

FIG. 48.\*



three openings: one to pour in the fluid, and to which the condensing syringe is afterwards attached; another from which projects the capillary extremity of a slender tube going to the bottom of the cylinder; and a third covered by a spring safety-valve, through which the compressed air may escape after a certain amount of pressure has been produced.

Another instrument, upon the same principle, is a modification of Sales-Girons, by Mathieu, a surgical cutler of Paris, in which the pump works by a long handle, and the fine stream impinges against the side of the cylinder, and not against a disc (Fig. 49). It has great power, and the spray is very minute.

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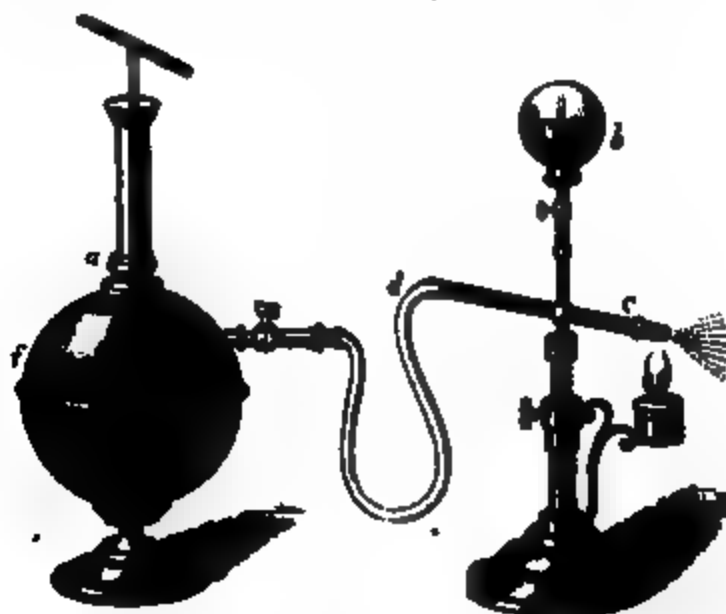
\* Lewin's Glass Nebulizer.

FIG. 49.\*



Mathieu afterwards (1859) made an apparatus which he called a Nephogène (Fig. 50). The air is compressed in a condenser to a sufficient degree, when a valve is opened, and it passes off through a flexible tube into a glass cylinder, containing the medicated fluid, and having a minute opening, with such force as to rapidly convert the liquid into fine spray. It is objected to this instrument that it has too much power, and that a quantity of atmospheric air is thrown into the air-passages.

FIG. 50.†



Dr. Bergson, utilizing the idea of Dr. Natanson, simplified the atomizing apparatus by the introduction of the tubes known by his name, and which were used by him in an instrument he called a Hydrokonion. Two glass

\* Mathieu's modification of Sales-Girons' apparatus.

† Mathieu's Nephogène.

tubes with capillary openings at one end, are so connected that the capillary extremities are at a right angle, the capillary end of the tube through which the medicated fluid is to pass being arranged so as to cover about half of the capillary opening of the other tube, when air or steam is forced through the latter, which is horizontal, a vacuum is formed in the other, which is vertical, with its other end lying in the medicated liquid, which liquid rising to the top is, by the force of air or steam, minutely divided.

FIG. 51 \*

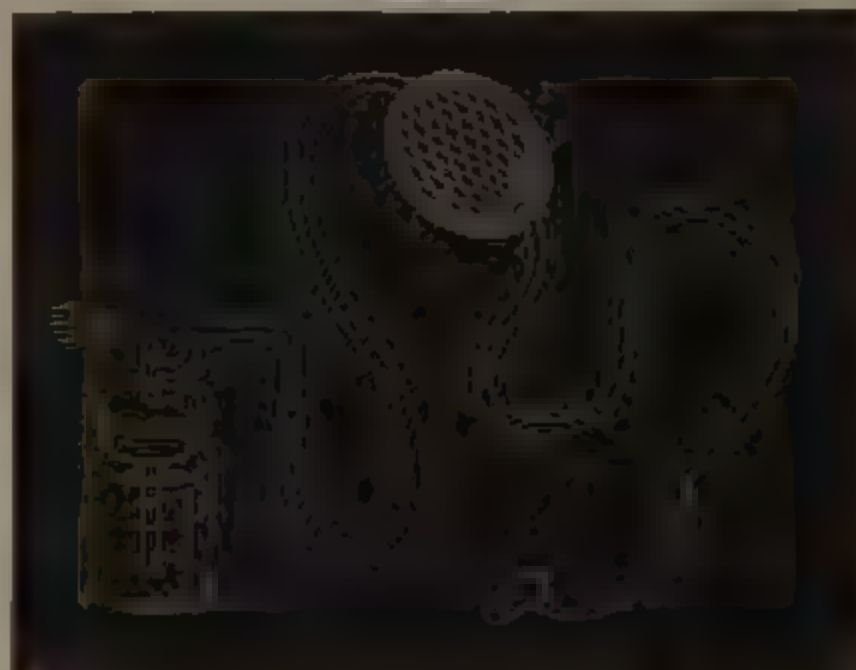


FIG. 52 †



Professor Wintrich modified these tubes by having them made separate, bent the small end of the tube to be placed in the liquid to a right angle, and bent the one through which the air or steam was to pass to a proper position, and bound them together by an India-rubber band (Fig. 51). These tubes can be made with free extremities of several inches in length, and can be passed into the different cavities, the spray generated within them, being thus brought, with a certain amount of force, into direct contact with the diseased surfaces. They can be made of all sizes and of different curves, to be passed up the nostril, as in the treatment of catarrh; to be placed in the ear, and reach the membrane of the tympanum, to be applied near the back of the throat, or immediately over the entrance of the larynx (Fig. 52), thus furnishing means for local treatment, and replacing the sponge probang.

FIG. 53 ‡



\* Wintrich, Modification of Bergson's Tubes. DA COSTA.

† Bergson's Tubes with Single India-rubber Bag.

‡ Maunder's Atomizer, with Bergson's Tubes and Dr. Andrew Clarke's Hand-ball.

The pulverizing current of air is supplied by the mouth, or by a bellows, or a Davidson's syringe, or what is still better, by one of Dr. Andrew Clarke's handballs, adapted to Maunder's Atomizer (Fig. 53).

The well-known instrument of Dr. Richardson, of London (Fig. 54), for the production of local anæsthesia, acts upon the same principle, and is an excellent atomizer. It will, by a simple arrangement,—the insertion of a slender wire in the spray-tube,—give either a fine or coarse mist.\*

FIG. 54 †



In these instruments, air being the forcing power, a certain amount of manual labor on the part of the physician, attendant, or patient, is required, and their use is apt to become irksome. This inconvenience is obviated by the ingenious apparatus of Siegle, called the Steam Atomizer (Fig. 55), in which steam replaces atmospheric air. Adopting the arrangement of the tubes of Bergson, he added a small boiler made of metal or of glass, in which steam is generated by the heat of a spirit-lamp. The steam plays the part of the compressed air, and, escaping, projects as a fine spray the liquid placed in the cup. The degree of pressure is indicated by a thermometer, marked 1 and 2. It is safe to let the mercury range between 1 and 2; above this there is some danger. By lowering the flame the steam is generated with less rapidity and force. A small lamp under the glass cup holding

FIG. 55 ‡



\* See *New York Medical Journal*, vol. iii, p. 156, and *Local Anæsthesia by Richardson's Method*, by Meredith Clymer, M.D., *New York Medical Journal*, vol. iii, p. 239, 1866.

† Richardson's Spray-Producer for Local Anæsthesia.

‡ Siegle's Steam Atomizer (De Costa).

the medicated liquid heats this, and the spray may, if necessary, be breathed warm.

A number of modifications of Siegle's apparatus, and which have materially simplified it, have been made and are in use. One by Dr. John Hart, of Boston, has been described by him in the *American Journal of the Medical Sciences*, October, 1866, p. 364. Mr. Gemrig, a surgical cutler of Philadelphia, has constructed a simple, convenient, and efficient steam atomizer, after a design given to him by Dr. Da Costa (Fig. 56). There is a copper boiler, and the thermo-barometer is replaced by a spring

FIG. 56 \*



FIG. 57 †



FIG. 58 \*



FIG. 59 ‡



FIG. 60 ‡



safety-valve. By unscrewing the safety-valve the water can be poured

FIG. 61



into the boiler; this fits into a metallic tube, at the bottom of which a spirit lamp is placed, and its flame can be raised or lowered at will. The atomizing-tubes are inserted into a cork, or a perforated piece of gutta percha, which is fastened by a metallic ring with a bayonet catch. When in use the boiler should be filled about two-thirds with water. The instrument may be kept clean by letting it pulverize pure water after it has been used with the medicated fluid.

Other modifications of Siegle's apparatus are pictured in Figs. 62 and 63. In Fig. 63, the thermo-barometer is gauged to two atmospheres, but the apparatus can be put in action with a pressure of one half atmosphere.

It is often important to protect the face from the spray, especially when liquids are used which may stain or damage the skin, for this purpose a glass or wooden funnel may be used, connected by a flexible tube with a cup (Fig. 65). Dr. William Read, of Boston, combines in a compact form the steam nebulizer and face protector. (Fig. 61).

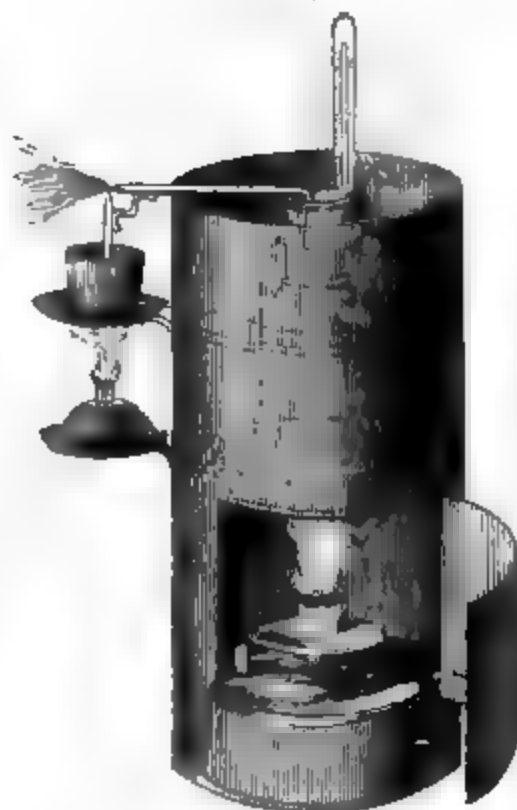
- \* Da Costa's Steam Atomizer, with duck-bill tubes.
- † Boiler, with tubes attached and spring safety-valve.
- ‡ Metal cylinder, to the upper part of which the boiler is placed, and in the part below, the lamp.
- § Lamp, with screw to raise or lower wick.
- || Cup for medicated fluid and slide to hold it.



The question may be asked, Of the several pulverizing apparatus, which is the best? As a general rule, for the diseases of the pharynx and the top of the larynx, to which this method of treatment is applicable, and particularly where it is wished to make the topical application directly

FIG. 63.†

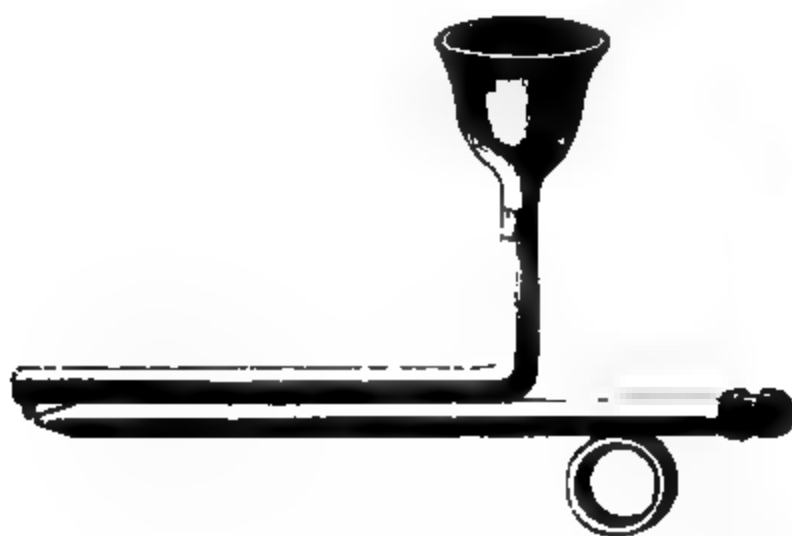
FIG. 62.\*



upon the diseased part, as in some cases it is desirable to do, and with some force, and when caustic solutions are used, the Bergson-Wintrich

FIG. 64.

FIG. 65.‡



tubes, as modified by Dr. J. S. Cohen (Fig. 64), or Richardson's Spray-Producer, furnished with Dr. Andrew Clarke's handballs, will be found good instruments; they can be used without loss of time, are handy, and the physician can apply the medicated spray upon the very spot he may desire. In diseases of the lungs and windpipe, Siegle's steam apparatus (Krohne & Seesemen's model), is, beyond all question, the best; and none of the modifications of it are improvements.

In this latter class of affections, moreover, it is generally best to use the atomized fluid warm. With regard to the temperature of the spray, Dr.

\* Siegle's apparatus, with water-gauge c and valve v, modified by Krohne.

† A modification of Siegle's apparatus (COHEN).

‡ Face-shield, with tubing and cup.

Hart remarks: "If Bergson's apparatus be used, and the temperature of the room be  $19^{\circ}$  R., and that of the liquid also  $19^{\circ}$  R., the temperature of the spray at its point of formation will be  $17^{\circ}$  R., and at a distance of eight inches  $16^{\circ}$  R. The higher the temperature of the liquid is, the greater will be the temperature of the spray. Thus, if the temperature of the liquid be  $60^{\circ}$  R., the temperature of the spray will be  $40^{\circ}$  R. at the point of its formation, and it will fall to  $17^{\circ}$  R. at a distance of eight inches. If the liquid have a temperature of  $5^{\circ}$  R., the temperature of the spray at its point of formation will be from  $9^{\circ}$  to  $10^{\circ}$  R., and at a distance of eight inches about  $14^{\circ}$  R. With Siegle's Steam Hydrokonion (or any instrument upon the same principle), the temperature of the spray will be much higher than that of the liquid. The nearer to the apparatus the patient inhales, the warmer will be the spray, and the farther, the cooler it will be" (*loc. cit.*, p. 366).

The temperature and force of the spray must be regulated by the circumstances of the case, and the judgment of the physician. At the beginning of the treatment, and particularly when there is much irritation about the throat, and cough-spells are easily brought on, the spray should be warm. The force of the current depending on the tension of the steam can be regulated by the lamp-screw and the thermo-barometer. Siegle illustrates the several relations between the strength of the current and its temperature in the following table:

- |   |   |
|---|---|
| 1. Current strong.<br>Thermo-barometer $2^{\circ}$ .<br>(In croupous laryngitis.)         | Temperature high.<br>Distance $\frac{1}{2}$ ths of an inch. |
| 2. Current strong.<br>Thermo-barometer $2^{\circ}$ .<br>(In chronic pharyngitis.)         | Temperature low.<br>Distance 6 inches.                      |
| 3. Current weak.<br>Thermo-barometer $1^{\circ}$ .<br>(Tuberculosis of larynx and lungs.) | Temperature high.<br>Distance $\frac{1}{2}$ ths of an inch. |
| 4. Current weak.<br>Thermo-barometer $1^{\circ}$ .<br>(Hæmoptysis.)                       | Temperature low.<br>Distance 6 inches.                      |

(Siegle, translated by Nickles, p. 40.)

A good many rules, to be followed by patients during the sitting, have been given; most of them are useless and embarrassing. The patient should sit in a convenient position, and so that the spray shall be formed on a level with the mouth, which is to be wide open, the head being thrown slightly backwards, and the tongue pressed against the floor of the mouth, or held down by a depressor, or protruded and held by the patient, as in laryngoscopy. A small glass speculum, from three to four inches in length, may be put into the mouth; it serves to keep the tongue out of the way, and does not interfere with the breathing; but this will be rarely necessary, and the fewer instruments used the better. A towel should be thrown around the neck, and Beigel's wooden screen with mouth-piece used, to hinder the mist-jet from wetting or soiling the face and clothes, particularly when the fluids are caustic or staining. A caution should be given to breathe naturally at first, and to afterwards learn to quicken and deepen the respirations, as may be necessary. Hurried and jerking breathing is the fault with most at their first sitting; and this not only defeats the end, but may bring on unpleasant feelings, as giddiness, tightness about the chest, and sometimes blood-spitting. Begin with about one hundred inhalations, making a pause of a few seconds after every ten or fifteen deep inspirations, and afterwards increase the number as the case may require. Dr. Beigel remarks: "Some patients after the first in-

halation of astringent fluids, have an unpleasant sensation in their throat, which disappears after a second or third repetition. The same may be said of a slight cough, which, in a sensitive larynx, shows that the spray has passed the glottis, and is therefore rather an agreeable sign than the reverse." If the spray is to be applied to the pharynx or top of the larynx, the patient is instructed to breathe naturally; if it be desired that it reach the air-tubes, he should take deeper inspirations, but not hurriedly, irregularly, nor with sufficient effort to fatigue him. A neglect of these simple rules is a very common cause of the failure of treatment by atomization. The distance of the mouth from the capillary end of the spray-tube must vary, according to the nature of the case, and the intention of the operator. At the beginning of the operation five or six inches are sufficient, and may be gradually lessened. In many pharyngeal disorders it is advisable to play the stream with some force directly upon the part, and here the tube must be introduced into the mouth; and it may be desirable in affections of the air-tubes that a large quantity of warm spray be breathed, and then the mouth should be quite close to the apparatus. Where there is much irritability of the throat and air-passages, the heated spray of water should be breathed at first. The inhalations should be taken always when the stomach is empty, and not directly after exercise; nor should the patient go out at once into the air afterwards. The first sittings should not be longer than from three to five minutes, according to individual susceptibility: subsequently, they may be extended to ten or fifteen minutes, but with frequent pauses of a few seconds, to avoid annoyance, fatigue, and feeble and irregular breathing. From half an ounce to one ounce of the medicated fluid should be atomized at each sitting. A strong current is often best in affections of the pharynx, whilst in pulmonary disorders it should be of moderate force, otherwise it is apt to condense about the fauces, and does not enter the air-passages. Except in such cases where its temporary employment only is indicated, this method of treatment demands time, persistence, and regularity. To have any measure of success it should be used daily, and sometimes two and three times daily: patients therefore should be instructed how to use the apparatus, and the physician should be present at the first sittings.

That the medicated spray not only reaches the fauces and pharynx, but also the surface of the air-tubes, has been convincingly demonstrated by the experiments of Demarquay, repeated by Poggiale, and reported to the French Academy of Medicine. Dogs and rabbits were made to breathe a pulverized solution of the perchloride of iron, and afterwards killed; on examination, the presence of the salt throughout the larynx, trachea, air-tubes, and even pulmonary structure, was shown. Experiments were also made upon the human subject by Demarquay and Fiebr, which proved confirmatory. In a case of pulmonary consumption under the care of Dr. Lewin, a pulverized solution of the chloride of iron was used to arrest hæmoptysis: the patient dying, the fluid and coagula of a cavity of the right lung were analyzed, and found to contain iron. A patient of Dr. Zedekauer, of St. Petersburg, suffering from Bright's disease, had pulmonary hemorrhage which was arrested by an atomized solution of chloride of iron; dying two days afterwards, a large quantity of iron was detected in the lung-tissue. In the discussion upon this subject before the French Academy of Medicine (1865), it was conceded that pulverized fluids do reach at least the surfaces of the larynx, trachea, and bronchi. Dr. Beigel calls attention to the fact, noticed by all who use atomized inhalations, that every patient when inhaling describes a sensation of penetration into the trachea and lungs.

The diseases which have been treated more or less successfully, by in-

halation of atomized medicated fluids, are chiefly those of the pharynx, larynx, trachea, and bronchi. In many affections of the pharynx, particularly those of chronic form, and those arising from constitutional syphilis, the results are, sometimes, prompt and happy. It is advisable often in these cases to use a coarse spray, or sort of douche. In acute pharyngeal disorders it has been less employed, but from the writer's experience of its effects it is worthy of a more extended trial. Dr. Da Costa states that even in enlargement of the tonsils, he has used pulverized fluids with success; and he mentions a case, where a cure by other topical means was impossible, from the violent resistance on the part of the little patient, and which was effected by this method. Catarrhal, tubercular, and syphilitic laryngitis, seem often greatly benefited, as well as some forms of aphonia. (Edema of the glottis has been relieved (TROUSSEAU, DA COSTA). Croup, diphtheria, and whooping-cough are alleged to have been successfully treated. Simple bronchial catarrh often improves rapidly after the inhalation of atomized fluids. Affections of the bronchial mucous membrane accompanied with profuse secretion, have been greatly bettered. Hæmoptysis would seem to be quickly arrested by it; asthma and emphysema improve; and much expectation of good results from its use in pulmonary consumption, so far as the relief of some symptoms are concerned, is entertained by many.

The writer for some time past has employed this method of treatment in diseases of the throat and respiratory organs; he has also had an opportunity of seeing it largely used in the practice of some of his medical friends; and he has carefully examined the published testimony in its behalf. Of its absolute and relative therapeutic value, he is prepared to speak more positively, from larger experience, than when this article was first published—eighteen months ago. That it is often a valuable means of treatment of many of the diseases named, there can be no doubt; but how far it may wholly replace other and established methods is yet to be determined. The chief drawbacks are uncertainty, tediousness, and incompleteness of results in many cases. The effect of atomized inhalations are often surprisingly and promptly happy, so far as the immediate relief of symptoms, but no ultimate curative results may follow, and those who rely exclusively upon them as a mode of treatment will be but too frequently disappointed, and find that much valuable time has been wasted. Dr. Beigel, whose work on the subject is an excellent one, and whose experience with this mode of medication has been large, rates its worth fairly, when he says: "He who expects wonders from this mode of treatment will soon be disappointed; and he who recommends it as an infallible one will prove a false prophet." And yet it may be truly said, with the late Dr. Trousseau, Sales-Giron has rendered a great service to the world at large by his invention of the treatment of disorders of the respiratory organs by means of atomized inhalations.

The following table gives the chief substances used, their doses, and the disorders in which they are employed.

**WATER**,—*Cold* in hæmoptysis, and pharyngeal disorders; *hot* in bronchitis, laryngitis, diphtheria, croup, asthma, aphonia.

**LIME-WATER** (1 part to 30 or 60 of water). In diphtheria, croup; (to be used tepid.)

**COMMON SALT** (3 to 20 grains in an ounce of water). In acute and chronic lung and throat disorders generally, and often very relieving in phthisis.

**NITRATE OF ALUMINUM** (2 to 5 grains to the ounce of water). Dr. Beigel speaks well of it in nervous affections of the larynx and trachea.

**TANNIN** (1 to 20 grains to the ounce of water). Diphtheria; gangrene of the tongue; croup; œdema of the glottis; hæmoptysis; paralysis of vocal muscles; chronic

disorders of fauces, pharynx, larynx, and trachea. Begin with small doses and stop if dryness and constriction are complained of; if not, alarming symptoms of pulmonary congestion may happen.

**ALUM** (5 to 30 grains). The coarse spray of a strong solution applied directly to the parts in pharyngitis, elongated uvula, and tonsillitis, is relieving; weaker solutions in hæmoptysis, diphtheria, laryngeal excrescences; and in combination with tar water in consumption.

**SOLUTION OF THE CHLORIDE OF IRON** (5 to 30 drops). In hæmoptysis; diphtheria; chronic bronchitis with excessive secretion; whooping cough; hysterical aphonia (weak solution).

**NITRATE OF SILVER** ( $\frac{1}{2}$  to 10 grains). Diphtheria; chronic pharyngeal and laryngeal disorders.

**FLUID EXTRACT OF CONIUM** (3 to 10 drops). Irritative coughs; asthma; laryngeal hyperæsthesia. May be generally substituted for laudanum.

**FOWLER'S SOLUTION** (5 to 10 minims). In asthma; phthisis.

**BROMIDE OF POTASSIUM** (1 to 10 grains). In cough from irritation about the pharynx and larynx.

**MURIATE OF AMMONIA** (2 gr. to  $\frac{1}{2}$  drachm). In beginning bronchitis; in all acute and chronic affections of the throat and lungs; capillary bronchitis; phthisis, in combination with common salt.

**TAR WATER** (1 to 2 fluid drachms). In consumption, and chronic bronchial affections, with purulent or fetid expectoration.

**CORROSIVE SUBLIMATE** ( $\frac{1}{2}$  gr. to 2 grains). Syphilitic affections of pharynx and larynx.

**GLYCERINE** (diluted about one half with water). In whooping cough; bronchitis; in inflammation of the larynx and trachea with hoarseness and aphonia; in dry cough of phthisis; in lung disorders accompanied with fetid breath or expectoration.

**SULPHUROUS ACID** ( $\frac{1}{2}$  drachm to 1 ounce of water). Syphilitic affections of the throat; chronic catarrh, with much secretion.

For more extended tables the reader may consult Da Costa On the Treatment of Diseases of the Respiratory Passages by the use of Atomized Fluids, 1867. Beigel On Inhalation as a means of Local Treatment of the Organs of Respiration by Atomized Fluids, 1866. Emil Siegle's Treatment of Diseases of the Throat and Lungs by Inhalations, translated by Dr. Nickles of Cincinnati, 1868. Inhalation: its Therapeutics and Practice, by Dr. J. Solis Cohen; (an excellent abstract.)]

## LARYNGITIS.

**LATIN** Eq., *Laryngitis*; **FRENCH** Eq., *Laryngite*; **GERMAN** Eq., *Entzündung des Kehlkopfs*; **ITALIAN** Eq., *Laringitide*.

**Definition.**—*Inflammation of the larynx.*

**Pathology.**—It is from the situation of the lesion that affections of the larynx derive their importance, their gravity, and their great danger. *Acute laryngitis* is not only dangerous to life, but there are few diseases which can kill quicker. Death approaches by suffocation. The width of an eighth of an inch or so in the chink of the *glottis* may be all the passage left to breathe by; and the œdema which attends the disease may so completely close the chink, that death may ensue in two or three minutes.

It is the œdema which attends the inflammation which makes laryngitis so dangerous a lesion. The disease may supervene with rigors like a common cold; but very soon a sense of constriction and strangulation occurs about the throat; and the voice becomes affected, or completely disappears. The most striking phenomena are expressed by modifications of the natural functions of the part. Vocalization, cough, and respiration are all more or less modified.



While the patient breathes he makes a noise in his throat, both during expiration and inspiration. The noise is like the sound of a whisper, and may be heard at a considerable distance. Inspiration is labored and tedious, and starts sharp from the conclusion of the previous expiration; and both are accompanied by a rasping, hissing sound. Voice is gone, which shows that the *larynx* is the part affected, because there, and there only, voice is formed. The cough of disease of the larynx is also peculiar. It is brassy in its tone, terminates in a hissing noise, and begins similarly by a hissing inspiration in a muffled manner, because the lips of the glottis being thickened, irregular, and rough, cannot be sufficiently closed to begin a sharp sound (HYDE SALTER). Laryngitis is one of the legitimate results of a catarrh, and if once it occurs is liable to occur again.

**Treatment.**—If there be time, commence by the inhalation of the steam of boiling water, and continue to breathe hot moist air, as much by the nostrils as possible. Leeches may also be applied to the throat, followed by hot fomentations. If benefit do not follow these remedial measures, tracheotomy ought not to be delayed. The air of respiration ought then to be warm, moist, and plenty of it, through a large-sized canula, the orifice of which must be kept free of secretion.

#### LOSS OF VOICE—*SYN.*, APHONIA.

**Definition.**—*When the larynx is so affected that the voice is wholly or partially lost, the patient is unable to speak except in a whisper.*

**Pathology.**—Whatever affects the muscles of the larynx, as any overstraining of the voice in singing or speaking, cold, or sudden changes of the weather, is apt to produce a more or less temporary loss of voice. Rheumatic affection of those parts is also a cause, as likewise all that impairs the nervous energy of the laryngeal muscles. Thus the voice is often lost after a paroxysm of hysteria, or a severe mental affection. It is well known, also, that at the period of menstruation, two or more of the upper notes of the voice are apt to be lost for the time. Cases of dilated aorta, or of aneurism of the aorta about its arch, are apt to be associated with temporary aphonia.

Complete aphonia is common to all ages, but is most common in early adult age, and more particularly in the female. In advanced age the change of the voice, and the general impairment of its tone and volume, are well known. Many singers who have compassed two octaves in their prime hardly, perhaps, preserve four feeble notes in old age. This, perhaps, among other changes, may be owing to ossification of the cartilages.

Tumors and warty-like growths have been recognized by the laryngoscope (see p. 812, *ante*, Fig. 37); but an entire loss of the voice often takes place without any congestion, inflammation, or other structural lesion of the tissues of the larynx and glottis. When aphonia is secondary or symptomatic, tubercular or other structural diseases of the lungs are often found.

**Symptoms.**—In primary aphonia there is no tenderness or sore-

ness of the larynx, no pain on pressure, and no expectoration, and the general health of the patient is good. It often comes on suddenly, and only in a few instances is the attack gradual. It often also disappears in a few hours, but in other cases it continues for some weeks, and even months.

**Diagnosis.**—Aphonia is so marked a symptom, that though some doubt may exist as to the cause, none can exist as to the disease. It is distinguished from the aphonia in phthisis, or other malignant diseases, by the general good health of the patient; and the laryngoscope ought to be used to see the condition of the vocal cords.

**Prognosis.**—Primary aphonia is seldom of any moment. When it results from phthisis, it is one of those symptoms which mark the tendency of the disease to a speedy fatal termination.

**Treatment.**—Aphonia is often very difficult to cure. Sometimes attention to the general health will remove it. In other cases it yields to some local application, as blisters, mustard poultices, or the camphor liniment, or other similar stimulating applications. Tonic regimen, the fundamental basis of which may be summed up in the three words, "air exercise, and diet," of the most sanitary kind, is generally sufficient to bring about the cure. The shower-bath has often been of service; and it should be remembered that this disease can be easily feigned.

## [NERVOUS AFFECTIONS OF THE LARYNX.

(DR. CLYMER.)

The Laryngeal neuroses may be classed under two heads (*A.*) Diseases of the Motor System, and (*B.*) Diseases of the Sensory System.

### (*A.*) DISEASES OF THE MOTOR SYSTEM.

These may be subdivided into (*a.*) Paralysis of the Vocal Cords, and (*b.*) Spasm of the Vocal Cords.

#### (*a.*) PARALYSIS OF THE VOCAL CORDS.

**1. Bilateral Paralysis of the Adductors of the Vocal Cords** (*Crico-arytænoidæi laterales, and arytænoidæus proprius*).—A condition in which, from non-approximation of the vocal cords on attempted phonation, there is loss of voice. The characteristic symptom is aphonia. Though voluntary muscular power is lost, the reflex function is not generally affected; the cough and the sneeze are usually accompanied with a distinctly laryngeal sound; the laugh being a feeble expiratory sound, is not always phonetic. On examination with the laryngoscope, an attempt to say *a*, *e*, or *o*, does not approximate the vocal cords; they may approach one another slightly, or they may be immovable, leaving a large triangular space between them. Though both vocal cords are paralyzed one may be more affected than the other. The laryngeal mucous membrane is nearly always pale, though occasionally congested.

This disorder may be confounded with loss of voice from feeble respiratory action—expiration not being powerful enough to set the cords in proper vibration. The approximative action of the cords may be mechanically interfered with by the presence of outgrowths, or cicatrices, or disease of the crico-arytenoid joints, conditions detected by the laryngoscope.

**Pathogeny and Causes.**—As a rule there is no appreciable structural impairment, either in the larynx, nerves, or nerve-centres. Nerve-force seems to be feebly produced, or wrongly directed. Most commonly the disorder follows catarrhal congestion; hoarseness and cough disappear, and the voice is gone. It is met with in the feeble and anæmic; women are more subject to it than men, and young women more so than old. Frequent in the second and third stages of consumption, it should be distinguished from the aphonia due to organic changes in that affection. Emotional influences, especially sudden fright, frequently give rise to this disorder. It occurs in hysteria. A case is recorded where it was produced by pressure on the recurrent nerves (BÄUMLER); and Dr. Mackenzie saw an instance where it was caused by a cancerous tumor of the brain. Sometimes it seems caused by malarial toxæmia, and is then intermitting (GERHARDT).

**Prognosis** is favorable, for though often obstinate it commonly yields to treatment, even when of six, eight, or even ten years' duration; atrophy of these muscles rarely happening even when the disorder is of long standing. Sudden restoration of the voice may take place spontaneously, particularly under the influence of emotion.

**Treatment.**—Stimulant inhalations may be used, as the vapor of ammonia, or of creasote (MACKENZIE), or chlorine gas (PANCOAST). (See section on Atomized Fluids.) Solutions of the nitrate of silver (3j to f3j of water), or of the chloride of iron (f3ij to f3j of water), or a saturated solution of tannin in glycerine, may be applied with a brush to the interior of the larynx. The one remedy which, according to Dr. Mackenzie, is almost invariably successful, is the direct application of electricity to the vocal cords. One pole is introduced into the glottis (the other being placed externally on the neck between the cricoid and thyroid cartilages), and kept there for three or four seconds, and a succession of short shocks passed; at each sitting this is repeated three or four times. In two hundred cases this treatment failed in four (MACKENZIE). Mackenzie's laryngeal electrode is the most convenient instrument to use.

**2. Unilateral Paralysis of the Adductors of a Vocal Cord.**—In these cases there is loss of voice or hoarseness, from one of the vocal cords not being adducted to the median line. The condition is at once seen with the laryngoscope. On attempted phonation, the affected vocal cord remains at the side of the larynx, whilst the healthy one is adducted well to the median line. The mucous membrane over the affected cord is often congested. There is aphonia, or dysphonia, and usually no constitutional symptoms. If due to disease of a nerve-centre there is usually paralysis of the tongue or palate, or hemiplegia. When the paralysis of the vocal cord is complete, or even much marked, coughing, sneezing, and laughing are always altered in character, and often without sound. A modification of the natural sneeze, or cough, is often one of the earliest symptoms. This disorder is frequently associated with slight dysphagia, from imperfect action of the epiglottis, or, more probably, from paresis of the superior and middle constrictors.

Swelling of the ventricular band (false vocal cord), which, when excessive, hides more or less the true vocal cord on the same side, and whilst the other is seen well adducted towards the median line, may lead to an error of diagnosis; but a careful examination should show the true nature of the case. Ossification, or other changes, in one of the crico-arytenoid joints may hinder the action of the muscles; but this condition can be usually detected.

**Pathogeny and Causes.**—In a case of seven years' standing, examined

after death, there was considerable atrophy of the crico-arytænoideus lateralis muscle of the affected side (MACKENZIE). The disease is probably due to simple or dyscrasic deposit into the muscle-tissue. When accompanied with paresis of the same side of the tongue or palate, it indicates lesion of the nerve-centre about the nucleus of the spinal accessory. It is met with, in toxæmia from lead, arsenic, diphtheria, &c., after small-pox, in constitutional syphilis, and may be caused by cold, or muscular strain. Sometimes it results from pressure of an aneurism or other tumor on one of the recurrent nerves; the left nerve is affected through the arch of the aorta, and the right through the subclavian or carotid of the same side. When it happens in phthisis, as it sometimes does, it has been thought by some to be due to pressure on the right recurrent nerve (MANDL, OGLE); in two cases observed by Mackenzie the lung and vocal cord were affected on opposite sides.

**Prognosis.**—This condition is not relievable when it depends on cerebral disease, or thoracic tumors, or phthisis, or tissue-change in the muscles, or is of many years' duration. If due to chronic toxæmia, or cold, it is curable.

**Treatment.**—Direct electrization of the muscles at fault.

**3 Bilateral Paralysis of the Abductors of the Vocal Cords** (*Crico-arytænoidæi postici*).—The vocal cords not being drawn aside, or abducted, from the median line in inspiration, there is dyspnœa and stridulous breathing; the voice is generally only slightly affected, being a little thick, or hoarse. The laryngoscope shows that the vocal cords on inspiration, instead of being abducted from the median line, remain nearly approximate, the opening of the glottis being proportionate to the degree of paralysis, and varying from one line to two lines or more. In forced inspiration the opening generally becomes smaller, and in forced expiration larger. The vocal cords are often slightly congested. When the patient is quiet, respiration may be but little affected, but any exertion brings on breathlessness. During sleep the breathing is stridulous; and there is croupy cough. In children the symptoms are not unlike those of laryngismus.

Spasms of the adductors produce symptoms closely resembling those of paralysis of the abductors. In cases of spasm, the vocal cords are constantly varying in the degree of adduction, while in paralysis, the cords are immovable.

**Pathogeny and Causes.**—This disorder consists in a loss of power of the crico-arytænoidæi postici, the abductors of the vocal cords, caused by the interception or non-production of the nerve current, which should be supplied to these muscles through the pneumogastric and its branches. In one case examined after death, the muscles were greatly atrophied. Though most frequently connected with cerebral disease, conditions affecting both pneumogastric, or both recurrent, nerves may produce it. It has been met, where there have been scrofulous deposits in the bronchial or cervical glands, in exophthalmic goitre, in cancer of the œsophagus, &c.

**Prognosis.**—Is very serious, and there is constant risk of immediate suffocation, from congestion or œdema of the glottis.

**Treatment.**—The only relief is from tracheotomy, which should be done at once.

**4. Unilateral Paralysis of the Abductor of a Vocal Cord.**—Owing to the non-abduction of one of the vocal cords in inspiration there is dyspnœa, stridulous breathing on exertion, and slight alteration of the voice. On laryngoscopic examination, when the patient inspires the affected cord is not drawn aside from the median line; it is generally congested.

The voice is shrill. The constitutional symptoms vary with the conditions giving rise to the paralysis.

**Pathogeny and Causes.**—This variety has the same origin as the bilateral, only peripheral influences, as pressure on one pneumogastric or one recurrent, are more frequent. The diseased muscle is much wasted.

**Prognosis**—Is unfavorable, as the condition is indicative of serious organic disease.

**Treatment.**—When the symptoms are threatening tracheotomy should be performed.

**Paralysis of the Tensors of the Vocal Cords (*Crico-thyroides*).**—This condition may be bi- or uni-lateral, and is one in which, owing to the vocal cords not being properly stretched, the voice loses power and clearness, especially in the high notes, and it may be suppressed. Fatigue after or during speaking is commonly felt; sometimes the talking voice is not affected, whilst the singing, or preaching, voice is faulty. There are three things to note in a laryngoscopic exploration. (1.) The surface of the vocal cords is not perfectly horizontal; in quiet respiration there is a slight depression or elevation near the centre, according as the breath is inspired or expired. This is best shown by making the patient whisper the letter e. (2.) When the disease is marked, the edges of the vocal cord can sometimes be seen to be waved in such a manner, that, taking the anterior insertion of the vocal cord as one point, and its posterior extremities as the other, the edge of the vocal cord does not pass in a direct line between them, but is more or less sinuous. (3.) The processus vocalis can never be seen with the aid of the laryngoscope, when the tensors are paralyzed. This though a confirmative symptom is not diagnostic, as the extent to which it is apparent depends on its natural development, and on the thickness of the mucous membrane. The abductive action of the cord is sometimes at fault. The mucous membrane is of a dirty gray color, though not generally congested.

**Pathogeny and Causes.**—This disorder is functional, depending on a strain or a weakness of the crico-thyroid and posterior crico-arytænoid muscles, for though the former are the chief tensors, the latter are auxiliary and necessary to complete action. The most common cause is too long or too violent use of the voice. Its subjects are clergymen, auctioneers, singers, actors, orators, military men who give words of command. Young girls learning to sing and choir-boys have been its subjects. Dr. Mackenzie says that he has never known a barrister to be affected.

**Prognosis** is favorable especially when the complaint is unilateral, it usually quickly yielding to proper treatment by blistering over the affected muscle, and the application of the electro-magnetic or galvanic current to the posterior surface of the cricoid cartilages, as well as to the crico-thyroid muscles.

**Paralysis of the Laxors of the Vocal Cords (*Thyro-arytænoidæ*).**—In this variety of laryngeal paralysis the voice is unnaturally high, shrill or grating, or there is difficulty or fatigue in using the lower notes, from imperfect relaxation of the vocal cords. It may be uni- or bi-lateral. With the laryngoscope the vocal cords may sometimes be seen unusually elongated. A very minute elliptical opening between the vocal cords, corresponding to their middle third, is more frequently to be noticed in phonation; and the tense condition of the cords often throws a shadow towards the ventricular orifice, which makes this opening seem larger than in the natural state. This elliptical aperture is only to be seen when both ends are affected; when the laxor of one end is involved, only one-half the ellipse is visible, and the arytenoid cartilage on the same side appears



rather in advance of its fellow. There is generally slight congestion of the mucous membrane of the cord.

It is important to note whether the elliptical opening varies much in form. If evident one moment and at the next not to be seen, it is due to spasm of the tensors, and belongs to a different class of disorders. When due to paralysis of the laxors, the shape in phonation varies very slightly or not at all.

**Pathogeny and Causes.**—This complaint is probably more muscular than nervous, and due to some change in the ultimate sarcous elements. It is caused more often from over-fatigued than from strained muscles. It is sometimes a congenital condition, in which the relative power of the tensors is too great.

**Prognosis.**—When congenital incurable; always obstinate.

**Treatment.**—Absolute rest of the organ—silence,—which alone is adequate to the cure in slight cases; in severer ones local electrization, continued for over a longer period than is necessary in paralysis of the tensors.

### (b.) SPASM OF THE VOCAL CORDS.

This condition is characterized by sudden, and temporary approximation of the cords; when complete, there is arrest of respiration with apnoea; when incomplete, stridulous inspiration and breathlessness. It is known as *laryngismus stridulus*, *spasmodic croup*, &c.

**Pathogeny and Causes.**—It is most commonly met with in children between the ages of six months and two years, and more often in males than in females, and then is due either to (1), centric nervous disorder, probably congestion or molecular tissue-change about the origin of the pneumogastric or spinal accessory nerves, which is often dependent on some dyscrasic state; it is met with in chronic hydrocephalus (West); or (2), to peripheral causes, which may be (a), *direct*, as pressure on the recurrent or pneumogastric nerves by tuberculous, cervical, or bronchial glands, or (b), *reflex*, as the irritation of teething, indigestible food, worms in the alimentary canal, or cold currents of air on the skin. Attacks come on, also, in the infant when sucking, caused by the passage of liquid into the glottis.

In adults the causes are obscure; there is no evidence of cerebral irritation, and it happens either as an hysterical phenomenon, or follows catarrhal congestion of the larynx.

**Symptoms.**—A first attack happens in this wise. A child put to bed, apparently in good health, wakes up suddenly about midnight with breathlessness, inspiration being accompanied by a peculiar crowing sound, similar to that heard in croup. After two or three of these stridulous inspirations, the frightened child bursts out crying, and in a few minutes is fast asleep again. It will be found, however, that in many, and the writer believes, in the majority of instances, some feverish catarrhal symptoms, or sighing breathing, and slight hoarseness generally pre-exist; this was the case in the greater part of the cases collected by Jurine, and in nearly all of those analyzed by Rilliet and Barthez. The next day the child is seemingly well, or at least free from any croupy symptoms, but most often is attacked again about the same hour, eleven o'clock in the night, and the second attack being more severe than the first, both in character and duration. In a severe fit there is sudden great embarrassment of breathing, each indraught of air being prolonged, with harsh stridor; then all sound ceases, the glottis is completely closed, and

the respiratory movements of the chest suspended; the flushed face becomes pale, and then livid; the eyeballs roll, the veins of the neck are turgid, the hands are flexed on the wrist, and the fingers are closed on the thumb, which is bent in the palm; the foot is flexed, and rotated slightly outwards, and the great toe is forcibly abducted. The infant will then throw its head back, struggle for breath, recovering it with a noisy inspiration; or there may be partial relaxation, and then a return of the seizures, in one of which death may happen.

In adults, the breathlessness is very great, with distended nostrils, fixed and protruded eyeballs, livid face, &c.; but there is no muscular spasm of the hands and feet. The remission is less complete than in children, catarrhus symptoms remaining.

The laryngoscope shows the vocal cords during the seizure to be spasmodically approximated. They may separate widely, but instead of remaining apart for a few seconds, they are instantly and spasmodically brought towards the median line, or even beyond it and against one another. Often there is great tension of the vocal cords. The larynx may appear perfectly healthy, or there may be more or less congestion; sometimes the vocal cords seem perfectly healthy, whilst the rest of the membrane is hyperæmic.

**Diagnosis.**—The diacritic signs between spasmodic and true croup are distinctive. The absence of severe febrile symptoms, the suddenness of the attack, the complete remissions after the first seizures, serve to make the diagnosis of laryngismus easy, and prevent error. In the adult we have the laryngoscopic signs.

**Prognosis** in children varies with the cause: when there is evidence of cerebral disorder it is always serious; if there is enlargement of the thymus gland, once thought to be so common in this complaint, it is dangerous. The intensity and length of the seizures are to be considered,—the longer the interval the greater the chances of recovery. In adults it is not so threatening as in children, and in them tracheotomy, if necessary for immediate relief, is more apt to be successful.

**Treatment.**—The first object during the fit is to produce violent inspiratory action: this may be accomplished by dashing cold water on the face; blowing forcibly in the ear; putting a piece of ice for a moment on the upper part of the spine; placing the little sufferer in a half hot bath; the application of a sponge wrung out of hot water, and applied to the front of the throat has brought relief. In Germany they tickle the fauces with a feather or the finger until vomiting comes on. Chloroform stops the spasm, but should be used with great care. No remedies by the mouth can be given, for the child cannot swallow. Turpentine, or assafoetida, enemata, have been recommended. In the adult, inhalations of hot water, simple, or to which conium, stramonium, or chloroform (¼ 40 to half a pint of fluid) have been added, are recommended. Dr. Mackenzie says that two of his patients got relief by smoking the *datura tatula*.

Subsequent treatment should be directed to the removal of the cause, if it can be ascertained, and to the improvement of the general health. Careful attention should be paid to the diet.

**Spasm of the Tensors of the Vocal Cords.**—The vocal cords are spasmodically stretched, giving rise to a feeble straining voice, so peculiar in its tone, as to be diagnostic. Some notes of the voice, either natural or slightly muffled, are produced, and then there is partial interruption, the sound giving the idea of arrested action of the expiratory muscles. Mackenzie describes it as much like the suppressed voice of a person doing a straining act, as defecation, parturition. After speaking several words

or even sentences in this peculiar tone, a few words may be uttered in a comparatively healthy voice, and then there is an immediate relapse into the diagnostic intonation. Exertion may diminish the spasm, or it may increase it.

The tense condition of the vocal cords can generally be seen with the laryngoscope, and they are usually congested, along with the mucous membrane of the larynx. Diagnosis between this affection and paralysis of the larynx has been already pointed out. The *varying* voice of the spasmodic larynx is distinctively different from the constantly *high-pitched* or *suppressed* voice of the paralytic disorder.

**Pathogeny.**—Probably due to some morbid condition of the sympathetic ganglia. There is not simple spasm of the crico-thyroid muscle, but spasm of the expiratory act, in which the thoracic and abdominal muscles participate.

**Prognosis.**—Unless recognized and treated early, it is incurable.

**Treatment.**—The patient should be forbidden to speak above a whisper, even for a second. Mackenzie's treatment is to apply extract of belladonna over the crico-thyroid muscles by constantly wearing a strip of lint covered with it on the throat. He has seen good effects from breathing the fumes of nitrated paper.

#### (B.) DISEASES OF THE SENSORY SYSTEM OF THE LARYNX.

**Hyperæsthesia.**—Increased sensation, occurring without inflammatory disease or chronic tissue-change. Cases have been reported by Gerhardt, Mackenzie, and Handfield Jones. It may be periodic or not.

Inhalation of hot sedative vapors and anæsthetics do good in non-intermittent cases, with internal use of narcotics.

**Anæsthesia.**—This rarely happens as a distinct disorder. Disease at the origin, or in the course, of the pneumogastric nerves, or their superior laryngeal branches, may diminish the sensibility of the larynx. Romberg asserts that it is impaired in cholera.

#### ATROPHY OF THE VOCAL CORDS.

This is very infrequent, owing to the dense structure of the vocal cords, and the slowness of their nutritive changes, and the rarity of complete paralysis. Even when the loss of voluntary power is total, the cords move in respiration and various reflex respiratory acts. Mackenzie reports four cases, three of which followed syphilis, and the fourth was suffering from chronic lead-poisoning. In three the aphonia came on suddenly, showing paralysis to have been the starting-point. The voice in this affection goes at once, or is reduced to a low whisper, and the sufferer cannot cough. The laryngoscope showed only a trace of one of the cords.

The literature of this subject is limited, and scattered, and the writer acknowledges his obligations to Dr. Morrell Mackenzie's article on Hoarseness and Loss of Voice in *The London Hospital Reports*, vol. iv, 1868, in which he has collated what has been written, and added his own experience, illustrating it by thirty cases. See also Türck's *Klinik der Krankheiten des Kehlkopfes*, 1866. Gerhardt, *Virchow's Archiv.*, vol. xxi. Ziemssen, *Electricität in der Medicin*, 1866.]

## INFLAMMATION OF THE MUCOUS MEMBRANE OF THE ŒSOPHAGUS.

*Œsophagitis* is a rare disease, for morbid poisons seem to have little influence over this portion of the alimentary canal, and atmospheric vicissitudes are in like manner seldom followed by inflammatory affections of this part. The most frequent causes of inflammation of the Œsophagus are, accidentally drinking boiling water; swallowing corrosive liquids, as the mineral acids; and wounds, most commonly inflicted in the act of committing suicide. Children a few days old are sometimes affected with slight inflammatory affections of the Œsophagus.

Inflammation of the mucous membrane of the Œsophagus is characterized by a deep redness of the part, generally terminating by resolution, but occasionally followed by separation of the cuticle. Lymph may be thrown out. In new-born children points of lymph are often found lying on the mucous membrane of the Œsophagus, being apparently an extension of the thrush affecting the mouth and pharynx. Andral has seen, in a girl twelve years old, lymph thrown out after the manner of broad bands, in the pharynx, Œsophagus, and stomach. After puberty this form of inflammation is still more rare, but there are some few instances. Cruveilhier says that he found among the preparations of Dupuytren a very remarkable example of inflammation of the Œsophagus, terminating in the formation of a false membrane, which coated this canal throughout its whole length. Dr. Abercrombie also gives the case of a gentleman, aged twenty-six, who caught cold, and died in about three weeks. The whole of the pharynx was covered by a loose adventitious membrane, which extended over the epiglottis, and portions of it were found lying in small irregular masses within the larynx at the upper part. A similar membrane was traced through the whole extent of the inner surface of the Œsophagus quite to the cardiac orifice.

Besides lymph being thrown out, the mucous membrane of the Œsophagus may also ulcerate, especially as a result of irritant poisons. These ulcers in general form on the anterior portion of the Œsophagus; and by continued extension they at last penetrate the posterior surface of the larynx, so that the patient often dies suffocated from the escape of food into the lungs. Occasionally the ulceration takes place from without inwards. The cicatrices are very apt to induce stricture. In cases of poisoning with the mineral acids, the whole Œsophageal canal may become constricted and narrowed, and the mucous membrane puckered up and contracted, so as greatly to diminish the calibre of the canal generally. More commonly the stricture is partial, one circular muscular fibre perhaps having been abnormally contracted, and in this state bound down by adhesive inflammation, diminishing the diameter of the canal at that part to at least one-half. Dr. Baillie mentions a case in which, from this cause, the diameter of the Œsophagus was so

reduced as hardly to allow a garden pea to pass; yet in all other respects the œsophagus was healthy.

**Symptoms.**—The symptoms of *œsophagitis* are almost entirely local, and consist principally of pain, of dysphagia, of the expectoration of a thick viscid mucus, and perhaps of vomiting. Emaciation follows the loss of nutrition, and the patient ultimately dies from inanition. Stricture may be induced by the careless introduction of a probang.

**Diagnosis.**—The diseases with which it may be confounded are similar states of the stomach; and the diagnosis in these cases is often difficult and perplexing. Stricture may be confounded with the spasmodic affections caused by an irritated state of the lung or trachea.

**Prognosis.**—Simple *œsophagitis* is probably often recovered from, as is seen after wounds of the throat partially dividing the œsophagus; but the chronic forms of inflammation probably often lay the foundation of disease leading to the ultimate death of the patient. Ulceration extending into the thoracic or pericardial cavity has been in all cases fatal.

**Treatment.**—The treatment of *œsophagitis* is by small local bleedings, by warm cataplasms to the neck, and by moderately acting on the bowels. In the treatment of the more chronic forms some sedative is essential. The use of the probang must be left to the discretion of the practitioner. There is always some danger in its use, of rupturing the canal, or of causing an ulcer. When the case is hopeless, from the small quantity of aliment which reaches the stomach, life may yet be prolonged by enemata of soups, milk, egg wine, or other nutritious fluid matters.

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## CHAPTER XII.

### ON THE DIAGNOSIS OF BOWEL DISEASES.

#### SECTION I.—RELATION OF THE ABDOMINAL VISCERA TO THE WALLS OF THE ABDOMEN.

THE regions into which the abdomen is usually divided by lines are, like those of the thorax, already described, quite arbitrary; and Figs. 5, 6, 7, already given, are referred to here, likewise the description given at p. 534, as to the fixed skeleton points which determine the direction of these lines, and the regions mapped out by them.

The vertical lines which have reference to the abdomen are five in number, and run as follows: (1.) From the insertion of Poupart's ligament in the external tubercle of the pubes to the acromial extremity of the clavicle (right and left side); (2.) From the posterior boundary of the axilla (the inferior edge of *latissimus dorsi*) to that



point of the crest of the ilium on which it falls vertically, Fig. 6 (right and left side); (3.) Along the spinous processes of the vertebræ from the sacrum to the nape of the neck.

The transverse lines are four in number, and indicate horizontal planes as follow: (1.) On the point of the xiphoid cartilage; (2.) On the last short ribs; (3.) On the anterior superior spinous process of the ilium on each side; (4.) On the upper margin of the *os pubis*. These three horizontal and five vertical bands map out the abdominal walls into *thirteen* regions, of which five are anterior, four are lateral (two on each side), and four are posterior. They are named as follows: *Anterior regions* are *epigastric* (No. 4 on the figures); *umbilical* (No. 5); *hypogastric* (No. 6); right and left *inguinal* (No. 11). The *lateral regions* comprehend the right and left *hypochondriac* (No. 9); the right and left *iliac* (No. 10). The *posterior regions* embrace the *inferior dorsal* on the right and left (No. 15); the right and left *lumbar* (No. 16).

The best idea of the contents of these regions is obtained by defining first the limits of the *liver*. It fills the right *hypochondriac* region (No. 9, Figs. 5, 6), filling up the concavity of the diaphragm; and it is almost completely concealed by the arch of the ribs. A part of the left lobe projects into the *epigastric* region (No. 4, Figs. 5, 6) and *left hypochondriac*. It also projects upwards into the *infra-axillary* region (No. 8, Figs. 5, 6) of the thorax, where it is separated from the thoracic wall by the thin lower margin of the right lung. Its upper margin in this space is on a line nearly with the level of the nipple, about the fifth intercostal space—less frequently beneath the fifth rib. In the perpendicular axillary line its margin is about the *seventh* intercostal space—more seldom under the seventh rib: close to the vertebral column its margin is in the tenth intercostal space—less frequently in the ninth (FRERICHS). At the median line the upper boundary of the liver cannot usually be distinguished from the lower margin of the heart. It is best made out by drawing a straight line from the point of contact of the right margin of the cardiac dulness with the upper boundary of the liver to the apex of the cardiac dulness on the left (CONRADI, FRERICHS).

Percussion of the liver after a meal is to be avoided; and any obstinate constipation which may be present must be removed before percussion, by means of free purgation, and any accumulation of gas must be got rid of.

In the epigastric and left hypochondriac regions lies the stomach. The umbilical region is crossed by the transverse colon, passing from right to left a little above the umbilicus.

The convolutions of the jejunum and ileum occupy the umbilical and hypogastric regions. The large intestine surrounding the convolutions of the lesser intestines occupies the iliac and lumbar regions on each side. The kidneys are equally shared between the *infra-scapular* (No. 14, Fig. 9) and the inferior dorsal regions (No. 15, Fig. 9). The spleen in its greater bulk is in the same region on the left side.

## SECTION II.—METHODS OF EXPLORING THE ABDOMEN.

These are principally three—namely, *inspection*, *manual examination* (*palpation*), and *percussion*.

**Inspection** furnishes information relative to *size*, *form*, and *movement*; and such information ought always to be acquired when the chest, as well as the abdomen, are both exposed simultaneously in a good light, the patient being protected from cold by a previous regulation of temperature in a room suited for the purpose of such an examination. The eyes of the patient ought to be directed away from the examiner.

**Palpation** furnishes information relative to position, size, consistence, elasticity, spontaneous movement, or mobility, and the presence of vibrations which may reach the surface. Care should be taken that the hand, when applied, should not be cold.

**Percussion** furnishes information relative to the comparative solidity of regions, and thereby indicates the kind of organ immediately below the seat of percussion.

The chief objects to be held in view in exploration of the abdomen are the following:

To ascertain—(1.) Its form and size; (2.) Its degree of tension or solidity; (3.) Its temperature; (4.) Sensibility or tenderness over any part; (5.) The presence or absence of tumor in or amongst the viscera; (6.) The presence or absence of fluids in the peritoneal sac; (7.) The nature and extent of the intestinal contents.

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## CHAPTER XIII.

### DISEASES DURING WHICH LESIONS TEND TO BE LOCALIZED IN THE ABDOMINAL CAVITY AND CONTAINED VISCERA.

#### SECTION I.—DISEASES ASSOCIATED WITH ORGANIC LESION. OR FUNCTIONAL DISORDER OF THE PERITONEUM.

##### PERITONITIS.

**LATIN** Eq., *Peritonitis*; **FRENCH** Eq., *Péritonite*; **GERMAN** Eq., *Bauchfellentzündung*; **ITALIAN** Eq., *Peritonitide*.

**Definition.**—An inflammation of the serous membrane lining the cavity of the abdomen, and covering the viscera contained in that cavity.

**Pathology.**—The peritoneum, like the pleura, is liable to inflammatory processes, either acute or chronic in their progress.

Acute inflammation of the peritoneum (as of all serous membranes) begins in the connecting fibrous tissue, which becomes red and injected, and at length the same phenomena pervade the serous

membrane itself. Its color when inflamed is a bright arterial scarlet hue—being first dotted with a number of small red points, which become confluent, and form streaks and patches, which in their turn coalesce; or a small central nucleus of inflammation may form, and spread till the whole extent of the peritoneum is one entire bright red color. In addition to the redness, some interstitial growth or exudation accompanies diffuse inflammation of the peritoneum, so that the membrane loses its transparency, and is thickened. When redness does not exist, opacity is often the only evidence of the previous existence of the inflammatory state. The consistence also of the subperitoneal tissue is greatly impaired, and rendered easily lacerable, so that the peritoneum is capable of being detached in considerable portions. This inflammation may terminate by resolution, or it may advance to the effusion of serum. The quantity may be trifling, not exceeding a few ounces, but occasionally it is large, fills the cavity of the abdomen, and constitutes inflammatory dropsy.

The exudation may be of the fibrinous type, when coagulation of the effused fluid tends to occur, and the opposed surfaces to be glued together. In some cases, however, the fluid predominates, and the fibrinous coagulated masses are loose, so as to float unattached in the serum, or of such consistence as to unite opposite parts together, and of such extent as sometimes to form an adventitious membrane, covering the entire surface of the abdominal walls as well as the whole of the intestines. The period at which organization of the lymph thus effused may begin, Mr. Hunter determined to be about twenty-four hours. If the disease proceeds, pus forms, sometimes not to a greater amount than a few ounces; but in other cases it amounts to many pints, or even fills the whole of the abdominal cavity. Ulceration of the peritoneum is not frequent, and generally takes place from without inwards, as from a perforating ulcer of the small or large intestines, or from the rupturing of an abscess or other tumor. The different acute inflammations described have been mentioned as though succeeding each other; but in many instances all these different forms coexist in different parts of the peritoneum at the same time, and perhaps have been irregularly set up.

Experience has also shown that although the structure of the peritoneum appears to be uniformly the same, yet certain parts of it are more liable to inflammation than others, as the convex surface of the liver or spleen, the right iliac fossa, the surface of the small intestine, and in females the broad ligaments, the Fallopian tubes, and the parts immediately adjoining them, as also the space covering the rectum and bladder. The parts the most rarely affected are those covering the stomach, bladder, omentum, and the mesentery. It will be seen that the liability of different parts of the peritoneum to inflammation is in proportion to the liability of the organs they cover to become diseased, and that these partial inflammations are for the most part the result of contiguous irritation.

The forms of peritonitis to be distinguished are,—(a.) *Puerperal peritonitis*; (b.) *Chronic peritonitis*; (c.) *Suppurative peritonitis*; (d.)

*Tubercular peritonitis*; (e.) *Adhesive peritonitis*, and local adhesions of the parts opposed to each other.

**Symptoms.**—Peritonitis may be acute or chronic, partial or general. It is occasionally ushered in by some previous shivering and fever, but in many cases there are no preliminary symptoms. The symptoms of peritonitis from perforation of the intestine or stomach are characterized by the *suddenness* and *intensity* of pain, often referred to a particular region of the abdomen; but the whole abdomen soon becomes painful to pressure. The pain is constant, exquisite, and leads to such lowering of the heart's action that death rapidly follows by *asthenia*, preceded by the most marked symptoms of collapse.

If acute peritonitis should not terminate by resolution, but by effusion of serum or of lymph, the patient complains of a severe pain in the abdomen, which is increased on pressure. He lies on his back, fearing to move. His pulse is from 90 to 120, and peculiar, as an inflammatory pulse. In proportion as it is frequent, so is it smaller. The tongue is coated, and the bowels constipated, or regular. If serum be effused, that event can often be determined by fluctuation, or by percussion in some parts; or if lymph, by a rubbing sound heard under the stethoscope when the abdominal movements of respiration are not suppressed. The course of these forms of acute peritonitis varies from a few hours to ten or fourteen days.

When acute peritonitis terminates in effusion of pus, the symptoms are infinitely more formidable. The pain in the abdomen is often the severest that human nature can suffer. The patient lies on his back, but his legs are drawn up and bent so as to relax as much as possible the abdominal muscles. By fixing his pelvis he endeavors to keep the abdomen still; he is restless, and unable to bear the slightest pressure, not even the weight of a sheet, and is incessantly tossing his arms about in every direction. The state of his tongue and bowels is similar, perhaps, to what has been described; but his pulse is excessively small and rapid, varying from 130 to 150, while the patient is often distressingly affected by retching and vomiting. These symptoms perhaps continue without intermission for twenty-four, forty-eight, seventy-two, or more hours; when, with or without some previous shivering, pus is effused, and the pain, from being agonizing, is now bearable. The subsidence of the pain, however, is not followed by any amendment; on the contrary, a most alarming collapse succeeds,—a cold, clammy sweat breaks out over the body, while hiccup, and a pulse hourly increasing in frequency, proclaim the entire hopelessness of the patient's surviving beyond a few hours.

When acute peritonitis is local—confined, for instance, to the surface of the liver or other organ—the pain is often limited to that part, while the other symptoms vary according to the severity of the affection and the organ whose covering is affected.

Chronic peritonitis often takes place to a great extent, and yet without any great amount of suffering. The symptoms are rather those of abdominal soreness and uneasiness than of pain, together

with a full but sometimes rapid pulse. The intestines, indeed, may be glued together, and sometimes pus has been found effused, without the patient suffering more than in ascites. When chronic peritonitis is partial, as of the liver or spleen, the patient often experiences a dragging pain, which is increased by a change of position, and arises from the parts being suspended by adhesion.

**Causes.**—Inflammation of the peritoneum often manifests itself during the course of some specific disease, such as paludal fevers, scarlet fever, and the like. Mechanical violence, as the kick of a horse, the operation for hernia, ovariectomy, or the stone, or of paracentesis, are occasional causes. Rupture of the intestine from ulceration, or the bursting of an abscess, or of an aneurismal tumor into the abdominal cavity, are examples of another class of causes. Errors of diet, and especially frequent intoxication, are also occasional causes. The disease termed *gin colic* is a chronic inflammation of the peritoneum. Sudden and great changes of temperature are also causes, especially in women at the period of menstruation. Intussusception of the intestine, or strangulation of the intestine from hernia, or other accidents, are also occasional causes. As a secondary disease it is frequently produced by hepatitis, splenitis, enteritis, and by *cancerous typhoid*, and *tubercular* deposits in the subperitoneal tissue.

Children sometimes die of this affection after fevers. Peritonitis, however, is most common between the ages of twenty and forty. Women appear to die more frequently from it than men; this greater liability to peritonitis in the female arising, perhaps, from the great sympathy between the uterus and the peritoneum—a sympathy which is strongly marked, not only at the period of menstruation, but also at the time of parturition. At the latter period, indeed, puerperal peritonitis often becomes contagious among parturient females.

**Diagnosis.**—The pain being greatly increased on pressure, and the pulse rapid, together with the general uneasiness and evident danger of the patient, readily distinguish peritonitis from colic. Its salient points of difference from enteritis will be noticed under that head.

**Prognosis.**—Partial peritonitis often terminates without in any sensible degree impairing the general health. Thus we often find extensive adhesions of the liver without any marked symptoms, as well as limited opacities of the membrane. In every case, however, in which the structure of the peritoneum is thickened or otherwise impaired, the patient may recover, but generally he relapses and dies of dropsy; for the peritoneum, like all other serous tissues, appears to possess little power of restoration after disease. Every attack of acute inflammation is of grave import, and when pus is effused it is very generally fatal; neither will the patient likely recover if the peritonitis is caused by subperitoneal tubercles, typhoid or cancerous lesions.

**Treatment.**—The treatment of acute peritonitis must be active—the activity of the treatment being proportioned to the amount of pain, the rapidity of the pulse, and intensity of the inflammatory fever,



which is frequently marked by the peculiar depressing influence of the inflammation on the heart's action. In the milder forms of the disease, when the pain is bearable, and the pulse steady and under 100, twenty leeches over the abdomen, followed by warm fomentations, together with the administration of opium in frequently repeated grain doses, ought to be the basis of treatment. In the severer forms of disease the first indication is to relieve pain. The stomach may be unable to retain food; and vomiting may be present. Under such circumstances, Dr. Anstie has found great benefit from the injection *per rectum* of a pint of strong meat soup slowly thrown up in three successive portions. About three hours' relief of pain was obtained, when a new injection of soup being given, the same relief to pain followed, and the pulse fell from 124 to 104. Small quantities of broth and wine could then be borne by the stomach, which were administered every two hours (*op. cit.*, p. 117). All action of the bowels should be prevented for several days. Opium should be given in doses of one to two grains, repeated as often as its effects subside—generally every two, three, or four hours. Morphia may be given in doses of a quarter to half a grain, and similarly repeated. Morphia combined with chloroform, as in the formula for chloromorphine or chlorodyne, recommended in vol. i, p. 453, will be found useful in allaying pain.

Leeches applied to the abdomen is the only method of bloodletting likely to be useful, and a poultice afterwards may be required to encourage the bleeding in sthenic cases. It is not proven that calomel, given for the purpose of inducing mercurialism, has any curative tendency in peritonitis. It has been prescribed traditionally; but the experience of Dr. Taylor regarding mercury in pericarditis, as well as of other physicians regarding the influence of mercury generally in the cure of these inflammations, tends to discard it now from our methods of cure in such affections. Fomentations are to be diligently employed, on the same principle as described under typhoid fever, vol. i, p. 387. Acute peritonitis has also been treated by quinine alone, in large and repeated doses, by M. Beau, at the Hôpital de la Charité. The remedy was given in the proportion of twenty to thirty grains of the drug in the twenty-four hours. When the abdomen can bear pressure, a flannel roller should be firmly applied round the body.

The treatment of chronic peritonitis must be directed by the same principles; but we should be content with effecting a present alleviation of symptoms, and without attempting the removal of the mischief which has already occurred; for in patients that have labored under chronic peritonitis, and survived many years, the peritoneum has still been found opaque, thickened, and silvery, so that in all probability these alterations are permanent, and not attended with danger.

The diet of the patient in the acute forms of peritonitis should be of the mildest and least stimulant kind.

## ASCITES.

LATIN Eq., *Ascites*; FRENCH Eq., *Ascite*; GERMAN Eq., *Ascites*—Syn., *Bauchwassersucht*; ITALIAN Eq., *Ascite*.

**Definition.**—*A collection of serum or inflammatory fluid effused into the cavity of the peritoneum.*

**Pathology and Morbid Anatomy.**—Cases of ascites are often examined after death, in which no affection of the peritoneum, or of any organ or tissue, can be discovered. More commonly, however, the peritoneum shows evidence of having been either acutely or chronically inflamed, some viscus diseased, such as the liver or the heart, or some tumor pressing on the large vessels, and causing the effusion which constitutes the ascites, in consequence of hyper-distension of small bloodvessels.

The most frequent concomitant affection with ascites is disease of the heart and large bloodvessels, to which it is believed that at least one-fourth of all the cases of ascites is owing. In these cases the cavities of the heart are often enlarged, and their walls either hypertrophied or atrophied, or the valves are ossified, or their action otherwise impeded, and the aorta may be pouchy—its elasticity and contractility being impaired by calcareous or other degeneration.

Morbid states of the liver and spleen are the next most frequently associated affections. These organs may be found in every possible state and stage of disease.

In general, *anasarca* accompanies *ascites*. It is an infiltration of serous fluid amongst the elements of the general connecting or areolar tissue of the body, passing, therefore, up through and amongst the more loosely connected parts of the body generally, such as between the skin and the muscles. In these cases the areolar tissue is found in various states: in some cases the interspaces or areolæ are greatly enlarged, while the tissue itself, generally thickened, tears most readily in some cases, while in others it is not only greatly thickened, but also greatly indurated. The fluid which it contains is generally limpid and watery, composed merely of the serous part of the blood; while in other instances the fluid is viscid, contains lymph, and the organizable elements characteristic of inflammatory origin.

The quantity of fluid contained in the abdomen in cases of ascites varies from a few ounces to many gallons; three to four gallons are by no means unusual, and as much as eighteen gallons are said to have been drawn off at one time by the operation of *paracentesis*. The quality of this fluid is very various. In color it is generally green or yellow; in consistence viscid, often containing so much coagulated matter as to be incapable of flowing through the canula.

**Symptoms.**—The symptoms of ascites are extremely well marked, but vary in some degree according to the cause, so that it is better to give, first, a general outline of its more prominent features, and

afterwards to point out those particular symptoms which indicate the cause from which it springs.

In ascites, if the quantity of fluid effused be considerable, the abdomen is distended and shining, with a number of large superficial veins creeping over its surface. From the weight of the abdomen, the gait of the patient is upright, like that of a pregnant woman; and if anasarca be present, he generally walks with his legs widely apart. In bed he is unable to lie down, on account of the fluid in the abdomen gravitating towards the chest and compressing the lungs, so that he is obliged to be raised towards the head and shoulders. If the anasarca be limited to the lower extremities, the upper portion of the body is in general greatly emaciated, the sharp and pinched features and the withered arms forming a striking contrast to the protuberant abdomen and swollen legs. On the contrary, if the *anasarca* be general, as the true signification of the word implies, the trunk, the arms, the hands, the eyelids, and face generally, are tumid, and swollen to a most unsightly degree. The urine is often defective in quantity, but is sometimes natural and sometimes in excess. The skin is dry, and the patient thirsty; his appetite greatly impaired, and his spirits generally greatly depressed.

The progress of the disease is seldom accompanied by any severe constitutional symptoms; but at length the legs and scrotum become greatly distended, and often inflame, so that the patient sometimes ultimately dies from gangrene of these parts. Again, bronchitis may take place, or the urine may become nearly suppressed, and similar effusions may occur in the cavities of the pleuræ.

The favorable circumstances are, the secretion of urine being re-established and becoming natural, the subsidence of the anasarca and of the ascites, and then a gradual return to health.

The presence of water in the abdomen may be determined by percussion of that cavity; and the best mode is to place one hand on the abdomen, and to give a sharp but gentle tap on the opposite side with the fingers of the other, when, if water be present a fluctuation will be felt. If, however, the quantity of fluid be small, the fluctuation is best felt by percussing the side of the abdomen from before backwards. The existence of fluid in the areolar tissue of the trunk or extremities is determined by the finger leaving a mark or "pit;" and the fluid being thus displaced, the part does not recover its original form and fulness for some seconds, and is said "*to pit on pressure.*"

The ascites may form suddenly, and the abdomen of the patient may be distended in a few hours, or the fluid may take weeks or months to accumulate. The duration of the disease is indefinite. If the effusion be general, the patient's life may terminate in a few days; but more commonly the affection is chronic, and the patient survives many weeks or months. Such are the more general phenomena of *ascites* and *anasarca*; but it is now necessary to pass to those particular forms which constitute its varieties.

Ascites sometimes results from the large effusion of serum which is poured out constantly into the cavity after undue exposure to

cold and wet. To this form the name of *active ascites* has been given, and although such cases have not the marked symptoms of inflammation, such as pain, yet the febrile reaction which generally accompanies such cases, and the fact that they yield to those remedies which subdue the inflammatory tendency, indicate such a connection. There are cases of a similar kind in which the *ascites* obviously results from *chronic peritonitis*; and now, although the patient sometimes suffers much pain, more commonly this symptom is wanting, or only occurs in occasional paroxysms. In this latter form the patient generally appears to die from the conjoint effects of *anasarca* and of *ascites*. The urine is scanty, but for the most part free from albumen in both these forms of disease.

The forms of *ascites* resulting from the following causes are examples of *passive ascites*, because they are all explicable by the fact that the cause tends ultimately to retard the flow of blood through the system of the *vena portæ*.

A *diseased heart*, or diseased state of the aorta, is often the primary cause of *ascites*. The heart's sounds, its impulsion, together with the character of the pulse, will indicate the particular lesion under which the patient labors, as explained under cardiac diseases. Dropsy from this cause may first show itself either by effusion into the abdomen, or into the areolar tissue of the lower extremities causing *anasarca*. When effusion has taken place, it is often remarked that the action of the heart becomes more regular, its impulse more natural, the pulse slower and steadier, while perhaps the murmur also may disappear. This apparent amendment, however, is fallacious: the dropsical symptoms increase, effusion takes place, first into one cavity and then into another, so that the patient seldom long survives this fatal symptom. The urine in this form of dropsy is generally deep in color, small in quantity, and of a healthy density.

*Diseases of the liver* offer the best illustration of how morbid states obstructing the *portal* circulation are the main causes of *passive* or *mechanical ascites*. When *ascites* arises from a *diseased liver*, that viscus is generally enlarged in the left lobe, when it is enlarged; but mere enlargement is not a common condition giving rise to *ascites*. The liver in most instances is smaller than usual; it is contracted and condensed, so that its shrunk and diminished bulk compresses the *portal* circulation. The condensation is also of a peculiar kind. It generally results from a compression of the proper hepatic substance, by a contracting tendency in the connecting fibrous tissue which accompanies the *portal* vessels—namely, the *capsule of Glisson*, and giving rise to the condition known as *cirrhosis* or *hob-nail liver*. The *ascites* in this case has no new feature, except that the patient may or may not be jaundiced. In the former case all the fluids effused are of a yellowish or greenish-yellow color. The urine also is loaded with bile, which is generally turned green by the addition of nitric acid; while in a smaller number of cases the bile appears to be in a peculiar state of combination with the urine, so that the acid has now no effect on it; the urine likewise is always small in quantity, much loaded with the usual salts, and

of a high density. The bowels are difficult to act upon, and the patient is liable to severe abdominal pains, simulating chronic peritonitis. The pulse continues throughout the disease for the most part natural, but the patient usually falls into a typhoid state, from which there is no recovery.

In ascites associated with disease of the *spleen*, the viscus is uniformly enlarged, and can readily be felt occupying the left hypochondriac region, and thus the cause, though not its exact nature, may be determined; for we have no diagnostic symptoms denoting whether the spleen be simply hypertrophied or in a cancerous or tuberculated state. The early symptoms are similar to those which occur in dropsy from disease of the liver, and, for the most part, are secondary to hepatic obstruction; and the termination of the disease, if the patient dies, is generally by hemorrhage from the stomach and bowels, often so profuse as to amount to many pints in a few hours, greatly exhausting the patient, and hastening the fatal issue.

In dropsy from disease of the *kidney* the urine may or may not contain *albumen*; but in the great majority of cases it does so. When albumen is absent (as the chronic forms of diseased kidney are all devoid of pain), we are unable to determine either the seat or the nature of the disease with which ascites is associated, and the ascites is consequently in general attributed to an affection of the peritoneum, of the liver, the spleen, the kidneys, the heart, or other viscus.

**Causes.**—These have been already sufficiently indicated, and may be summed up as comprehending undue exposure to sudden changes of temperature, loss of blood, obstruction of the portal circulation from morbid states of the liver, or spleen, or heart, and especially from dilatation of the chambers of that organ. The lesions of the kidney associated with ascites generally exercise their pernicious influence through the diseases of the heart which supervene during their course. Such cases prevail most between the ages of twenty and forty-five, while cases from disease of the heart and liver are most common from the ages of forty to sixty.

**Diagnosis.**—Ascites is readily distinguished in the male from every other intumescence of the abdomen by the fluctuation on percussion. In the female it can only be confounded with pregnancy, or with *ovarian dropsy*, which consists in the accumulation of fluid in one or more cysts, generally multilocular, within the substance of the ovary, or in a serous cyst connected with the uterine appendages.

The grounds of diagnosis may be thus shortly stated:

1. The uniform and symmetrical appearance, and general increased breadth across the flanks, possessed by the abdomen in *ascites* from the first, contrasted with the general *one-sided* growth of an ovarian tumor.

2. Percussion gives useful diagnostic results when performed in the different positions in which a patient may be placed. In ovarian dropsy dull sounds are fixed and invariable in one place, whatever



position is assumed by the patient. In ascites the dull sound follows the gravitating fluid in all positions.

The development of hydatids in the abdominal cavity may also simulate ascites, and so may an over-distended bladder.

**Prognosis.**—The *prognosis* in anasarca, in young persons not laboring under any organic disease, is always favorable. If, however, it be consecutive to organic disease, a fatal termination is ultimately to be feared.

Ascites arising from indeterminate causes is often recovered from, but no case is free from danger, the peritoneum often being so much impaired that the function of absorption ceases to be effected through it, and so the disease resists the action of all remedies.

Ascites depending on moderate inflammation of the peritoneum is often recovered from, and especially if the inflammation depends on the action of exposure to cold or to paludal poison.

Ascites with albuminous urine, arising from mere functional disorder of the kidney, is generally recovered from; but if the structure of the kidney be impaired, the disease is always grave, and generally fatal. In a few cases, however, the disease subsides, and the patient may continue well for two, three, or four years, when he generally relapses and dies.

Ascites from disordered function of the heart is often recovered from; but if it depends on diseased structure, either of the heart or large vessels, some temporary amendment may take place, while the patient quickly relapses and finally sinks.

Ascites depending on diseased structure of the liver or of the spleen is rarely recovered from unless the primary disease be cured.

**Treatment.**—When ascites occurs without any obvious organic cause, and without albumen in the urine, the best remedy is the *bitartrate of potash*, administered in divided doses, as one drachm three times a day, or every six hours, or in one large dose, as half an ounce, combined, if the patient's bowels be confined, with ten to fifteen grains of *jalap*. When the smaller doses are used, it is often exceedingly useful to add ten grains of the *citrate* or *tartrate of iron* to each dose. If these remedies should fail, one-sixth to half a grain of the *extract of elaterium* every night, or every other night, may be given.

There is a form of ascites, without any obvious organic cause, in which there is accompanying anasarca. Under these circumstances *squills* appear to afford most relief; and by giving five to eight grains of the *pulvis scillæ* three times a day, the dropsy is generally relieved, and the patient sometimes cured. If the stomach be irritable, *half a grain of opium* should be added to each dose so that the remedy may be retained.

Should the ascites arise from simple inflammation of the peritoneum, this form of dropsy in general yields to leeches and fomentations to the abdomen, together with the administration of a mild saline aperient every six hours.

When the ascites arises from disease of the heart, the kidney being sound, and the urine free from albumen, the treatment must

have reference to the nature of that disease. If the valves of the heart are diseased, the patient, though he cannot recover, may be greatly relieved by the administration of tonics, stimulants, and saline or drastic purgatives. An ounce and a half of camphor mixture with a drachm of the spirit of nitrous ether, fifteen minims of the tincture of hyoscyamus and a drachm of the sulphate of magnesia, will form a draught which, taken three times a day, will often greatly reduce the dropsy. When the stomach will bear it, the tincture of squills (℞x to ℞xx), with a drachm of the acetate of potash, has occasionally succeeded. Small doses of *elaterium*, as one-eighth to one-fourth of a grain three times a day, is a medicine that is also sometimes useful.

The ascites may be caused by disease of the liver; and should that organ be merely inflamed or hypertrophied, without other alterations of structure, the dropsy may disappear with the cure of the hepatic disease. The treatment is by bleeding, if the hepatic lesion be the congestion of inflammation, and the neutral salts, as the sulphates of magnesia or of soda; or should they fail, by moderate doses of calomel. When, however, its structure is otherwise altered the patient is seldom cured; but the disease may still be alleviated and life prolonged. In this form of ascites the patient suffers greatly from abdominal pains, which are relieved most effectually by fomentations. In these cases, also, the bowels are often greatly constipated, and require the most powerful drastic purgatives, as the black draught, castor or croton oil, or *elaterium*. In this form of dropsy, however, the peritoneum becomes more impaired in its power to absorb the fluid than in most of the others; the fluid is therefore seldom reduced, and the patient generally requires the last imperfect resource of art—namely, tapping, or *paracentesis*.

Ascites depending on enlarged spleen is also difficult of cure. If the spleen be simply hypertrophied, the *bromide of potash* and the *iodide of potassium*, in doses of five to eight grains three times a day, have been recommended. The patient, however, often dies from hemorrhage from the stomach after all the more prominent symptoms have been relieved. My friend and colleague, Dr. Maclean, informs me that rubbing the *biniodide* of mercury, in the form of an ointment, on the skin over the region of the enlarged spleen, has a very marked beneficial effect in reducing the enlargement. The ointment is to be rubbed into the skin while the patient sits before a strong fire, or in the rays of an Indian sun; and, as a further evidence of efficacy, the invalid soldiers invariably ask to be furnished with a supply of the remedy when they go from the hospital. The *biniodide of mercury*, similarly used, has been noticed as of great service in reducing the swelling of a goitre (vol. i, p. 795).

The dropsy which often occurs in young chlorotic women, in whom the urine contains *albumen*, the kidney being healthy in structure though disordered in function, is generally curable,—the most efficient remedy being the *bitartrate of potash* in drachm doses three times a day. It acts as a diuretic and as a purgative in these cases.

## SECTION II.—DISEASES OF THE HOLLOW VISCERA OF THE ABDOMEN.

## STOMACH DISEASES.

**Definition.**—*Under this indefinite heading it is proposed to notice some morbid states of the stomach, expressed by functional disorders, lesions of texture, or both combined.*

**Pathology and Morbid Anatomy.**—The organic diseases and functional disorders of the stomach have been recently elucidated in this country by the researches especially of Drs. Budd, Handfield Jones, Chambers, Leared, and Brinton. Nevertheless, the morbid changes which are known to occur in it are still but very imperfectly connected with the expression of clinical phenomena. “There is no part of the body,” says Dr. Chambers, “of which we hear so much from our patients, and are able to communicate so little knowledge in return, as about the stomach; and truly,” he also observes, “it is an ill-used viscus—flattered in metaphor and insulted in fact.” It has been regarded as a mere bag, a mere mill, or a mere chemical laboratory for the solution of substances; yet still all such similes and metaphors, as Hunter remarked, explain nothing; and he wisely insisted that it is a viscus *sui generis*, with definite functions to perform; in short, a “stomach is a stomach.” Its morbid states are similarly peculiar to itself and to its functions.

Instead of describing a series of disorders one by one, and by names such as *gastritis*, *dyspepsia*, *hæmatemesis*, and the like, it may perhaps be more instructive to write a condensed statement of that positive knowledge so satisfactorily established by the prolonged and well-directed labors of those men just mentioned. Such a method will better furnish those grounds for diagnosis by which clinical phenomena, often of the most painful, prolonged, and harassing kind, may be connected with some understood morbid state, and so furnish some fixed and definite principles by which the treatment of stomach diseases may be directed.

The morbid states of the stomach, ascertained after death to have existed during life, and in some measure to have been expressed by certain symptoms, may be enumerated as follows: (1.) *Softening of tissue*; (2.) *Glandular degeneration of the proper mucous substance*; (3.) *Congestion*; (4.) *Forms of inflammation tending to exudations and destruction of parts, or condensations of tissue, especially about the pyloric orifice*; (5.) *Ulceration*; (6.) *Carcinoma*.

Abnormal functional states are expressed by—(1.) *Vomiting associated with lesions of other organs*. (2.) *Deficient secretion of gastric juice*. (3.) *Fermentive processes (alcoholic, butyric, or lactic), tending to the development of entophytes, such as sarcinae*. (4.) *Indigestion, associated with and depending upon—(a.) Morbid states of those viscera which are conjoined with the stomach in the processes of digestion, such as the liver, pancreas, and small intestines; (b.) Imperfect action of the kidneys, as in Bright's disease; (c.) Defective or diminished morphological changes during the processes of nutrition in the tissues, generally expressed by altered secretions and excretions, as in many constitutional*

diseases ; (d.) *Indigestion, associated with pyrosis and increased secretion of the juices of the stomach and salivary glands, and with cutaneous disorders, such as urticaria* ; (e.) *Indigestion, associated with drunken habits.* The disease of the stomach with which one or more of these organic or functional states may be associated are,—*gastritis, chronic ulcer, hæmatemesis, perforation, dilatation, stricture, gastric fistula, hernia, cancer, colloid, tumors (non-malignant), sarcinæ, injuries, laceration, dyspepsia, gastrodynia, pyrosis.*

The essential juices of the stomach and of the intestines play a most prominent part in determining the nature of these diseases ; and some of the most important principles of treatment are based upon the physiological relations of those juices, and upon the fact that while the process of digestion of food is only commenced in the stomach, it is completed in the alimentary canal by the influence of the intestinal fluids. There is a *stomachal digestion*, in which the gastric juices, with the saliva and mucus of the stomach, play the most prominent part ; and there is an *intestinal digestion*, in which the intestinal juices, composed of the *biliary, pancreatic, and intestinal secreted fluid*, play the most prominent part. It is also an important fact, especially insisted upon by Dr. Chambers, having been proved by the experiments of Bidder, Schmidt, and Handfield Jones, that the intestinal digestion may be made to do more or less of the work of stomachal digestion, so that the exercise of the function of the stomach may be spared when necessary, and the food encouraged to pass from it into the bowels, to be digested entirely by the intestinal juices.

The reader is referred for information regarding the properties and constituents of the gastric juice to the text-books on physiology ; but it is necessary here to advert to the fact, recently established by the experiments of Schmidt and Bidder upon animals, and the observations of Grünewaldt upon an Esthonian peasant who suffered from a stomach fistula, that there is a constant circulation of an immense quantity of fluid through the mucous membrane of the alimentary canal, necessary to the solution and absorption of food from its interior. It is now also well ascertained that from the gastric glands the principal part of the solid animal matter of the gastric juice is derived, and which is the exciting cause of the solution of albuminoid substances in the stomach. The solid matter is regarded as a *gastric ferment*, and has been variously named as such, and also as *rennet*. But water constitutes the chief bulk of the juice ; and from the experiments and observations alluded to, it is known to perform a most important function. It is continually poured forth from the surface of the mucous membrane in vast quantities, to the extent of between a *fifth* or a *quarter* of the *weight* of the *body*, and as constantly returns to mix again with the sanguineous fluid. This secretion of fluid is constantly going on from the internal alimentary mucous surface. It mixes with the dissolving food in the alimentary canal, and takes up those particles of it which it is calculated to hold in solution, loaded with which it returns again to the sanguiferous, chyloferous, and lymphatic circulations. Thus a “poor” watery fluid is constantly being sent forth, to return “*laden with wealth*,” and so to minister to the nutrition of the body. This has been described as an internal mucous

circulation of fluid within the body; and the arrest of this interchange, with the great retention of water, has been shown by Virchow and Parkes to be a constant condition in the febrile state (see vol. i, p. 77).

**I. Softening of the Stomach.**—It was first announced by Hunter and subsequently confirmed by the experiments of Spallanzani, Wilson Philip, and Carswell, that the stomach under certain conditions as to temperature and properties of the gastric juice, especially at death, and immediately after that event, may be dissolved or digested by the secretions poured forth from its own secreting glands.[\*] In certain diseases, also, of a catarrhal kind, it has since been ascertained that potent gastric juice is sometimes secreted by the empty stomach, or that lactic acid being freely generated from the saccharine principles of food, forms with the mucous membrane an efficient digesting mixture; and not unfrequently a softening of the stomach may be predicted with tolerable certainty by a peculiar train of symptoms, and resulting from the presence of free gastric juice, or of a digesting acid, in the otherwise healthy stomach (BUDD). Dr. John Gairdner, of Edinburgh, however, so long ago as 1824, inculcated a similar doctrine. He observed, in a numerous series of cases of children, that a peculiar action of the gastric and intestinal mucous membrane, analogous to inflammation, weakened the texture of the organ, and rendered it morbidly susceptible to the action of the gastric juice during life (*Edin. Med.-Chir. Trans.*, vol. i, p. 311). It was in opposition to this view that Carswell performed his experiments, an account of which he published in 1838; so that the testimony they undoubtedly afford regarding post-mortem *softening*, as the only kind of softening, must be received with qualifications. The observations of Dr. John Gairdner and Dr. Budd evidently show that the probability of softening may be predicted during life, although Dr. Budd is of opinion that the softening does not take place till after death. A similar opinion is expressed by Andral. The diseases in which it is so apt to occur are those in which there is much cerebral disturbance and increasing debility and emaciation before death, and from such diseases

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\* [John Hunter's doctrine, that the escape of the stomach from being digested by its own secretions is due to what he called the "living principle" (*Philosoph. Trans.*, 1772), was upset by Cl. Bernard's experiment of introducing the hind legs of a living frog, through a fistulous opening, into the stomach of a dog, and showing that they were digested just as any other articles of food. This experiment was repeated by Pavy, and with a like result; he also substituted the ear of a rabbit for the frog's legs, and found at the end of four hours and a half that it was almost completely dissolved. Bernard suggested that the immunity of the stomach's coats from self-digestion and perforation, during life, might be due to its power of renewing its epithelial coat; this power ceasing on death. Pavy has proved this view to be erroneous by removing patches of mucous membrane from the stomachs of dogs, and showing that these denuded portions show no signs of destruction. Dr. Pavy concludes, from his own experiments, that the alkaline blood, which permeates the coats of the stomach, is the true agent which counteracts the effects of the gastric juice and saves the stomach from self-digestion. As the result of tying the vessels of the stomach of a rabbit, he has seen digestion of the walls proceed during life to the extent of perforation (*Philosophical Transactions*, 1863; *A Treatise on the Function of Digestion; its Disorders, and their Treatment*. By F. W. Pavy, M.D., London, 1867).—EDITOR.]



as *typhoid fever*, *cancer of the uterus*, or *peritonitis*,—in infants who die of *tubercular hydrocephalus*; in deaths from exhaustion from inflammatory diseases of the brain, when vomiting is a constant symptom; in persons who die from phthisis and from ulcer of the stomach. The symptoms from which the softening may be predicted are, that when, along with any of these diseases, there is much disorder of the stomach, such as pain and tenderness at the epigastrium, loss of appetite, thirst, frequent vomiting of acid fluids, and nausea, the lesion may be expected to be found after death. In the common form, as Hunter described it, the mucous membrane towards the splenic end is thin, and for the most part stained by hæmatine, very slippery, and appearing as a dark film gliding over the submucous tissue. The tubes appear under the microscope to be a good deal altered, chiefly by solution of their epithelium, while dark grains of melanic matter are deposited between the tubes. The amount of probable knowledge we possess respecting softening of the stomach is thus expressed by Dr. Handfield Jones:

1. There are two forms of softening: one, the commonest by far, which is simply the result of the action of the acid contents of the stomach upon its own dead tissue; the other, the consequence of a peculiar change taking place in its glandular structure, which generates a powerful acid, dissolving, corroding, or destroying the surrounding tissue.

2. The latter form may occur either with an empty or a full state of the bloodvessels of the stomach, the softening part of which will accordingly be either quite pale or of a dark blackish tint.

3. This same form occurs in a great variety of morbid states, which seem to have only this in common,—that they are attended with great depression of the vital powers.

4. It is more common in children than in adults, on account [probably] of the greater delicacy and less resisting power of the system.

**II. Glandular Degeneration of the Proper Mucous Substance** is by far the most common of the organic lesions—existing in 72 out of 100 cases examined by Dr. Jones. It is expressed in a variety of forms, the most marked of which are—(1.) Peculiar morbid changes in the tubes, probably analogous to the process which occurs in the tubes of the kidney in Bright's disease, where the molecular contents of enlarged epithelial cells increase, ultimately leading to complete destruction, so that the tubes become filled with the debris of this destruction; (2.) Melanic and fatty deposits in the epithelium; (3.) Interstitial deposit of nuclear and fibroid exudation at the expense of the gland-substance, so that hypertrophy of tissue may exist, along with—(4.) Atrophy of the tubular epithelium of the tubes themselves, and of the solitary glands—a form of cirrhosis generally associated with alcoholism, and described by Dr. Wilson Fox.

There is thus, on the one hand, in some instances, an utter destruction of the tubular glands—actual loss of substance without replacement; on the other hand, there is no actual loss of substance, but a replacement of elements by the deposition of granular matter within

the tubes, without diminution or alteration of their form. That these partial degenerations, even when they extend over a considerable portion of the stomach, do not materially interfere with the ultimate digestion of food, seems to be established by the cases described by Dr. Jones, deficiency of stomachal digestion being compensated for by increased vigor of intestinal digestion. Such loss of the gastric glands appear thus to have little influence over the vital acts, so that it is rare to find any evidence of the existence of such lesion during life, even when the degeneration is very extensive. I believe, however, from observations upon the intestinal mucous membrane in analogous glandular degeneration, that in cases characteristic of intestinal wasting, associated with anæmic states, as described at p. 110 of this volume, the mucous membrane throughout the whole alimentary tract is similarly affected, and that an examination of the mucous membrane of the lips, and buccal membrane of the mouth, indicates with great probability the change below, just as the condition of the tongue is the index from which we judge generally of the functional state of the alimentary canal. The general symptoms of such cases are undoubtedly anæmia and debility, without any obvious organic cause, often associated with vomiting or nausea in the morning, no desire for food, and a sensation as if it never was effectually swallowed, but stuck at the diaphragmatic entrance of the stomach, causing the peculiar feeling of weight which attends indigestion, and the abundant generation of gaseous fluids. It has also been well remarked by Dr. Chambers, that such symptoms indicate a participation of the whole alimentary tract in such lesions, and that where the lesion is merely confined to the stomach, the absence of such symptoms are sufficiently accounted for by the compensation to stomachal digestion afforded by the healthy intestinal tract.

**III. Forms of Congestion.**—These may be described as *passive congestions* ; or as *active congestions* associated with inflammatory lesions.

The *passive* form of *congestion*, like the passive form of *ascites*, is explained by the physiological phenomenon now recognized as perfectly familiar—namely, that the freedom of the transit of blood through any part is in a great measure dependent upon the healthy performance of its functions, and of the function of those parts immediately associated with it—so that, if function is arrested, the circulating current is arrested also, and the blood reverts, or is thrown back, to those intervening parts in the course of the circulation between the site of obstruction and the force propelling the blood. Thus blood ceasing to flow freely through the lungs reverts, or is thrown back, upon the right side of the heart ; blood ceasing to flow freely through the liver is thrown back upon the stomach, upon the one hand, or upon the spleen by the gastro-epiploic vessels, expressed by congestions of these viscera ; or, on the other hand, the portal system being obstructed, the blood reverts to the membranes of the intestines, and expresses such an occurrence by ascites, as well as by gastric venous congestion. Any mere mechanical impediment, therefore, which prevents the blood returning from the stomach towards the heart will induce congestion of the stomach.

The immediate effects of such congestion upon the functions of the stomach are sufficiently expressed by symptoms such as *hæmatemesis*, or the vomiting of blood; the secretion of the gastric juice also is diminished, the stomach can digest less food, and requires longer intervals of rest between the meals.

The persistence of such passive forms of congestion further leads to the deposition of melanic matter, and to disintegration of the tubular glands of the stomach, the formation of ulcers in its mucous membrane, just as varicose ulcers are established in the limbs from the passive congestion which attends varicose veins in the lower extremities, by impairing the nutrition of the part, as explained by Simon, Budd, and Jones.

Besides congestion from mere mechanical impediments, there are congestions of the stomach which have been described as *vicarious*—that is, as taking the place of congestions which ought to occur in other parts—such, for example, as when it occurs along with hemorrhage in women from stoppage of the catamenia; and which has been observed to occur in yellow fever, in malignant cholera, and in typhoid fevers. Congestion also arises during the development of—

**IV. Forms of Inflammation tending to Exudations and Destruction of Parts, or Condensations of Tissue, especially about the Pyloric Opening.**—*Idiopathic gastritis* is an exceedingly rare disease. When inflammation of the stomach does occur, it is generally the consequence of direct injury from irritant or corrosive poisons. Dr. Jones has never met with a case of acute idiopathic gastritis. Louis examined five hundred bodies without finding a single instance. Andral, however, relates several cases in his *Clinique Medicale*; and it is stated by Dr. Robert Williams, that during the whole of the Peninsular war not more than six cases were reported among the troops, although exposed to every species of privation, and addicted as they were to its most efficient exciting cause—the use of spirits in every form in which alcohol could be got to drink.

The difficulty of exciting acute inflammation in the stomach is well shown by experiments upon animals, and by the often long escape of the polyphagist, who swallows knives and watches, and all sorts of heterogeneous things; and of the Indian who passed many times daily a blunt sword into his stomach with impunity, till at last its coats were pierced, and he died. The stomach, also, we find will bear tea or coffee of an almost boiling temperature, followed perhaps shortly afterwards by a quantity of ice. One of the persons resident at the Eddystone Lighthouse at the time it was burnt, in 1755, swallowed a quantity of molten lead, which accidentally dropped into his mouth when looking from below upwards, to observe the progress of the fire. But even after this intensely hot substance had passed into his stomach, he lived several days, having been taken to the Plymouth Hospital, where he was attended by Dr. Sprey, who describes the case in the *Transactions* of the Royal Society. His attendants hardly believed his story possible; but on examining him after death, a lump of lead, weighing 7 oz. 5 drs. 8 grs., was taken from the stomach.

The simplest and most frequent form of inflammation of the stomach is that which is brought about by excess of food, especially of alcoholic drinks. It is characterized by a distension of the stomach and an excessive secretion of mucus in the stomach, a condition which Dr. Jones describes under the name of *catarrh*. Dr. H. Jones thus describes its phenomena:

“It occurs under the same influences as catarrh of the conjunctivitis, and often either coexists with these or follows them. Its anatomical characters are, distension of the stomach, abundant secretion of an extremely tenacious, clear, watery mucus. At the commencement of the disease, the hyperæmia is a marked feature; but as the disease advances, the watery mucus is a more constantly observed and characteristic feature. The secretion is of a passive kind powerfully predisposed to, and commonly conjoined with it. Watery, slightly acid, neutral fluids are secreted, and often ejected, and the disease does not tend to the destruction of the glands.”

*and Clinical Observations respecting the Stomach*

There is a comparatively rare form of inflammation of the stomach described by Dr. Budd, in which coagulable lymph is effused over the mucous areolar coat, and, hardening and contracting, forms a firm, fibrous mass, binding the mucous membrane to the underlying parts. Round the pyloric orifice such a morbid state may form a permanent stricture. It is a condition almost invariably the result of spirit drinking, and seldom occurs before the age of thirty.

**V. Ulceration.**—The observations of Drs. Habershon, Jones, and Brinton, have shown that gastric ulcer is a means uncommon. *Simple, chronic, and perforating* are the terms used to describe it. The ulcer is generally solitary and is usually smaller than a shilling, circular or oval in shape, its edges are well defined, as if punched out. It is generally situated near the lesser curvature, and usually nearer the cardiac orifice, and more frequently on the posterior wall. It is more common in women than in men, in the proportion of two to one. It is especially common in old and advanced life, although Dr. Budd once met with it in a child of fourteen. Dr. Brinton records two cases under the name of *perforating ulcer*. It is relatively more frequent amongst the poor than the rich; and is especially found in maid-servants, in whom it has been found in eighteen and twenty-five.

[Virchow believes the round ulcer of the stomach to be the result of some obstruction in the artery supplying the stomach, thereby depriving the alkaline blood from coming in contact with the gastric juices; and that, first, the mucous membrane, and then the coats, are softened and destroyed by the acid at the point of the access of the current of blood. See foot-note,

It tends to prove fatal by—(1.) *Perforation*, (2.) *Hæmorrhage*, (3.) *Exhaustion*.

When the ulcer eats its way through, the aperture gradually narrows, as it reaches the peritoneum, to little more than a point, which corresponds to the centre of the ulcer. The peritoneum inflames at this spot, and sloughs, when perforation is completed by the separation of the slough. An important sanitary effort is here made by the adhesion of the peritoneum surrounding the slough to some opposed surface, such as to the liver, pancreas, colon, or abdominal wall. Fistulous openings may then be established, so that food escapes from the stomach when such passages communicate with the alimentary canal. A most comprehensive record of such cases is published in *The Edinburgh Monthly Journal* for July and August, 1857, by Dr. Charles Murchison, physician to the London Fever Hospital and to the Middlesex. According to Dr. Brinton's observations, about 13.4 per cent. of cases of ulceration terminate by perforation; and he considers that the liability to perforation decreases as life advances; and, as Dr. Crisp first showed, during the ages from fourteen to twenty it is most frequent. The patient may survive the first shock of the accident, ultimately to succumb to the combined effects of peritonitis and gastric exhaustion.

*Hemorrhage*, while it is one of the most frequent and important symptoms of ulcer, is also a mode of fatal termination. It generally occurs soon after a full meal; and about from  $3\frac{1}{2}$  to 5 per cent. of the cases prove fatal in this way.

"This long and exhausting malady," says Dr. Brinton, "predisposes the constitution to a variety of other diseases, and renders unusually fatal many of those attacks of illness which, in the course of years, very few persons altogether escape."

**Symptoms of Gastric Ulcer.**—"The malady is announced by disturbances of gastric digestion: at first by mere uneasiness and pain; then nausea and vomiting, or regurgitation, that expel the food previously taken; or a tasteless or acid watery secretion. At this stage of the disease it is sometimes cut short by the occurrence of perforation, with its sequel of fatal peritonitis. Failing such an accident, the dyspeptic symptoms are next complicated by hemorrhage from the stomach; sometimes a sudden and dangerous gush, oftener a slow and intermittent drain of blood. The anæmia produced by this hemorrhage is generally associated with a cachexia which seems to be essentially independent of it; being chiefly the result of the inanition necessarily implied by frequent vomiting of the food, or by large destruction of the gastric mucous membrane, and consequent impairment of its function. In young females another symptom is often present, in the form of more or less complete amenorrhœa, which may be associated with either of these two states of anæmia or cachexia; in other words, may be connected with ulceration, with hemorrhage, or with both. The gradual acquisition of all these symptoms conducts the disease, in a variable period, to a climax, whence we may next briefly trace it towards its termination. Retaining the liabilities to death by perforation, by hemorrhage, by vomiting, and by exhaustion, which the above organic results of ulceration severally imply, the lesion often ends by one of these modes of dying, or by two or more of them in combination. In other cases a spontaneous subsidence of these symptoms, in something like the inverse order of their occurrence, an-



nounces a recovery ; or a similar amendment is only effected by a careful medical treatment, such as quite entitles us to dignify it by the name of a cure" (*Med-Chir. Review*, p. 159, July, 1856).

The character of the pain is peculiar, at first being little more than a feeling of weight or tightness, of a dull character, and continuous. It gradually becomes intensified into a burning or gnawing sensation, which produces a kind of sickening depression. It generally comes on from two to ten minutes after the ingestion of food, and remains during one or two hours, which correspond to the period of gastric digestion, after which it gradually subsides, or, if vomiting empties the stomach, it also invariably ceases. The pain is generally expressed at the centre of the epigastrium, or at the middle line of the belly, immediately below the extremity of the ensiform cartilage, often confined to a mere spot, and rarely to a space more than two inches in diameter. A dorsal pain, first described by Cruveilhier, is also subsequently established, generally in a few weeks or months after the epigastric pain. It is expressed by a gnawing sensation, interscapular, or from the spine of the eighth or ninth dorsal to that of the first or second lumbar vertebra. Pressure in the epigastric region is sometimes unbearable, and, for obvious reasons, must be applied with the utmost care and delicacy.

*Vomiting* usually occurs when the pain reaches its height ; and, completely emptying the stomach, generally affords relief. For further details of symptoms, the reader is referred to the admirable papers of Dr. Brinton, from whom the statements have been condensed.

**VI. Carcinoma.**—The cachectic state which ulcer of the stomach ultimately induces has caused many cases of simple ulcer of the stomach to be recorded as cancer ; so that it has been a popular belief that cancer of the stomach is a very common disease, till the inquisitive researches of Dr. Brinton have shown that the mortality attributed to gastric cancer is by far too great. Of necropsies performed at four of the great London hospitals, only about 1 per cent. of the total mortality is due to this lesion. The disease is thus far less frequent than ulcer of the stomach. Next to the female *breast*, the *stomach* seems, in this country, to be the part most liable to cancer, and the male is more frequently affected than the female—"a fact," writes Dr. Brinton, "which it is impossible to avoid connecting with the exclusive amenability of the female to the *mammary* and *uterine* localization of cancerous disease."

The orifices of the stomach are the parts which are almost exclusively the seat of cancerous lesions.

**VII. Abnormal Functional States.**—These are due—*First*, to what have been called sympathetic relations with other organs, themselves in a morbid state, and which are now explained by the phenomena of "*reflex action*." Examples of this may be referred to in the *vomiting* which attends irritation of the *lung*, *brain*, *liver*, or *uterus*. *Second*, a scanty secretion of the gastric juice, character-

ized by slowness of digestion and long retention of food by the stomach; prolonged distress after eating, especially of weight and uneasiness at the pit of the stomach; peculiar tendency to decomposition of food in the alimentary canal; the evolution of fetid gases; and the appearance of unaltered ingesta in the stools. Often an inheritance by birth, the conditions which tend to produce this morbid state are, mental over-exertion, prolonged anxiety, especially after meals, gluttony, drunkenness, and sedentary habits, and the consumption of more food than the system requires. The "*causes, symptoms, and treatment of imperfect digestion*," from these points of view, are admirably treated of by Dr. Leared in his little book on this subject.

**Urine in Dyspepsia.**—Alterations in the free acidity are the most important signs. But in some dyspeptic cases, especially those attended with torpid digestion or with acid vomiting, it has seemed to Dr. Parkes that the urine is more alkaline than usual; so that there may be abnormal acidity as well as abnormal alkalinity in cases of dyspepsia. When the urine is more acid than usual after food, it is also often scanty, and deposits urates and oxalates of lime—conditions associated chiefly with cardialgia, nausea, and frontal headaches. Chloride of sodium is small in amount when digestion is imperfect; and the more that is in the urine, the more perfectly has digestion been carried on. Oxalate of lime crystals are common in dyspepsia (PARKES, *l. c.*, p. 333).

**Treatment.**—Congestion, catarrh, ulcer, cancer, and functional states associated with what are called dyspeptic symptoms, or "*imperfect digestion*," are the causes of stomach diseases for which the physician is called most frequently to prescribe.

When there is reason to believe *congestion* exists, a sparing and easily digested diet is to be prescribed, and total abstinence from fermented drinks is imperatively demanded; and in cases where there is reason to believe that catarrhal inflammation prevails, the blandest food must be given in very small quantities. In severe cases leeches are to be applied over the region of the stomach, and the patient may sip iced water, or suck small pieces of ice, to relieve the thirst.

For obvious reasons ulcers are most difficult to heal, and such cases are to be treated by rest to the stomach, and by dietetic rather than by medicinal means. The patient must eat in small quantities, and especially of milk compounded with arrowroot, macaroni, semolina, sago, tapioca, biscuit powder, Indian meal, or oatmeal gruel. Iced milk, combined with one-quarter to one-third of lime-water, is particularly recommended by Dr. Chambers; two or three tablespoonfuls of which are to be taken at short intervals, so that about two quarts of milk may be thus used during the day, rendering regular and more bulky meals unnecessary. The lime-water tends to prevent coagulation of the milk, and the milk thereby more readily passes unaltered into the intestines, to be digested by them rather than by the stomach. Dr. Balthazar N. Foster, of Birmingham, has well laid down the details of managing such cases, in "*A Lecture on the Treatment of Gastric*

Ulcer" (*Brit. Med. Journal*, June 3, 1865). The most complete rest possible ought to be given to the affected viscus, by stopping the supply of all nutriment by the mouth, and supporting the patient for several days by nutritive enemata. Perfect quiet in the recumbent posture must be observed, the lips and tongue being moistened from time to time by a little water. The patient may thus be kept for eight or nine days, or even longer, on nutrient enemata alone, when the pain, the irritability of stomach and of the system will cease. The substances Dr. Foster has found most useful for enemata are milk, strong unsalted beef-tea, raw eggs beaten up in milk, occasionally a little brandy, and generally in two enemata daily ten or twenty minims of tincture of opium. The enemata should be as small as possible, from two to six ounces only at a time. The interval to the stomach thus gained of complete rest is of the greatest importance towards success in the treatment of gastric ulcer, and, combined with the restricted diet described, will usually effect a cure in three weeks.

Next to dieting, bloodletting by two or three leeches applied to the region of the stomach about twice a week affords obvious benefit, the patient often gaining weight during their use. Blistering applied to the spine is also said to relieve the dorsal pain. Small lumps of ice may be swallowed if the stomach is irritable, and astringent remedies, especially the *salts of metals*, such as a combination of *iron* and *alum*, are of great benefit; and, as a change, gallic acid, nitric acid, and bitter barks may be administered. Constipation is to be counteracted by enemata. When hemorrhage occurs, Dr. Budd recommends small bits of ice to be swallowed, that rest be maintained in the horizontal posture, and that astringent medicines be administered, such as *oil of turpentine*, *acetate of lead* and *opium*, *alum* and *tannic acid*. *Oil of turpentine* should be given in doses varying from ten to twenty minims in *cold water*, and repeated more or less frequently according to the urgency of the symptoms. When hemorrhage is copious, a mixture containing ten minims of *diluted sulphuric acid* and five grains of *gallic acid* in water, taken every two or three hours, is the best remedy (Dr. B. FOSTER). Dr. Leared justly puts great stress upon the necessity of strict attention to diet in all cases of impaired digestion. If fulness and uneasy sensations are experienced after dinner, less food should be taken at that meal, and more at breakfast, the principle being to apportion the amount of food necessary to sustain the body more evenly over the waking hours than is commonly done. The great fault in the dietetic system of this country consists in the fact that most people are supported mainly by dinner. This meal is consequently too large. The quantity taken at dinner should therefore be resolutely diminished till breakfast is appreciated (LEARED, *l. c.*, p. 150).

Special symptoms, common to various morbid states, require special modes of treatment. Excess of acid is best neutralized by *lime-water* or *magnesia*, and the gastric fermentation which is apt to be established may be checked by *brandy* and *various aromatic spirits*. *Bicarbonate of potash* and *nitrate of potash*, in the proportion

of *eight* parts of the former to *one* part of the latter, is useful in cases of habitual acidity; and all these antacid remedies should be taken about three or four hours after a meal. Pills containing from a quarter to half a minim of *creasote*, given with each meal, will in general counteract fetid eructations. It checks that fermentation in which acetic and carbonic acids are formed; while *conium* and *belladonna* are the medicines which better than *opium* allay general nervous irritability.

In cases of slow digestion, with deficient secretion of the gastric juice, the rules of treatment are—(1.) To let albuminoid food be as liquid as possible; (2.) To let the day's allowance be taken in small quantities at regular intervals; (3.) That by the administration of alkalies the food may pass to the intestines, and be digested there, rather than by the stomach. This latter mode of treatment by alkalies, recommended by Chambers, is contrary to that recommended by Dr. Budd. Both are consistent with physiological facts, and the course to be followed must be determined by the nature of the case. The amount of food taken is of great importance to be attended to in cases of slow digestion. If a fair amount of exercise be taken, the following dietary, slightly modified from that proposed by Dr. Leared, will be found appropriate in such cases:

## BREAKFAST (8 A.M.).

Bread (stale), . . . 4 oz.	{ Mutton Chop, or other meat (cooked) free from fat and skin, 8 oz }	Tea, or warm milk and water and sugar, or other beverage, . . . . . $\frac{1}{2}$ pint.
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## LUNCHEON (1 P.M.).

Bread (stale), . . . 2 oz.	{ No solids, such as Meat or Cheese. }	Liquid, . . . . . $\frac{1}{2}$ pint.
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## DINNER (5 or 6 P.M.).

Bread (stale), . . . 8 oz.	{ Meat (cooked) free from fat and skin, . . . 4 oz. }	Liquid, not more than $\frac{1}{2}$ pint.
Potatoes and other Vegetables, . . . 4 oz.		

## TEA OR SUPPER (not sooner than three hours after dinner).

Bread (stale), . . . 2 oz.	{ No solids, such as Meat or Cheese. }	Tea, or weak brandy and water, or sherry and water, or toast and water, to the extent of $\frac{1}{2}$ pint.
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For excellent advice on this subject, the reader is referred to Dr. Leared's book, p. 160, *et seq.*; and to Dr. T. K. Chambers's *Lectures chiefly Clinical*.

Forms of indigestion marked by excessive acidity and heartburn may be relieved by bicarbonate of soda, in doses of fifteen grains, combined with a few grains of nitre, and taken two or three times a day. At the same time, free excretions from the liver and bowels must be sustained by occasional small doses of *blue pill* or *podophyllin*, combined with extract of *colocynth* and of *henbane*; while exercise and diet are duly attended to.

Weakened digestion from over-fatigue may be often restored under the use of *carbonate of ammonia*, conjoined with compound

*tincture of gentian*, or with *extract of gentian* in the form of a pill. Extracts of *nux vomica* or *strychnia* are valuable remedies. *Half a grain of extract of nux vomica, half a grain of sulphate of iron, and four grains of compound colocynth pill*, form a combination which, taken early in the morning, generally induces gentle action of the bowels (LEARED). *Compound rhubarb pill* may be substituted in place of the *compound colocynth pill*.

Indigestion from habitual drunkenness is best relieved by bitter infusions, such as *gentian*, *quassia*, and *calumba*, singly, or combined in a mixture, so that a dose may be taken two or three times a day, an hour before each meal. Small doses of *opium* or of *morphia* in an ammonia mixture may also be given at bedtime, so as to secure sleep at night.

In prescribing the mineral acids, the following general rule, stated by Dr. Bence Jones, ought to be kept in mind—namely, that the influence of *sulphuric acid* is astringent, while that of *hydrochloric acid* promotes digestion, and of *nitric acid* secretion.

#### [THE NEUROSES OF THE STOMACH.]

(DR. CLYMER.)

This is a class of affections very common, ill-understood, often badly managed, and attended with much bodily discomfort and mental suffering. They are obscure in their origin, are accompanied by no appreciable structure-changes, and are seemingly caused by disorders of function and over-sensibility of the organ. The symptoms are deranged secretions, perverted appetite, local distress, pain, nausea, and vomiting, and, frequently, impaired digestive power; these phenomena may exist severally or be variously combined. The gastric neuroses may be divided pathogenetically into two varieties: (1.) Centric, where the cause appears to be in the nerves of the stomach, probably the sympathetic ganglia; (2.) Reflex, in which the functional disturbances and morbid sensibility are attributable to irritation in distant organs. The former are invariably associated with systemic asthenia, and lowered and disordered innervation, and the latter frequently so.

1. A nervous person will, after eating an ordinary or over-full meal, or some particular article of food, have suddenly an indescribable feeling at the pit of the stomach, as if he would die (*anxiété épigastrique* of Trousseau)—a sense of sinking—or a fluttering, or a weight, or soreness, or some perversion of healthful sensation, with a host of depressing emotions, as fear, apprehension of future trouble, low spirits. The natural temper may be altered during the seizure, becoming impatient, inconsiderate, or sour; the judgment is uncertain; the will weak; the mind sluggish; and the disposition fitful, suspicious, and cantankerous. Heart-palpitation is not infrequent. Soon after digestion is over, or large quantities of flatus are belched up, all this passes away, and the patient is in his usual way. Such cases are constantly happening amongst hysterical women and hypochondriacs, in the under-fed, and, in an increasing class in this country—the mentally overworked.

The process of digestion should go on without giving rise to any consciousness of its progress; a person in health should never know that he has a stomach. But many patients afflicted with these disorders, are pain-



fully and constantly aware of the presence of a stomach, and of the digestive function; attention is concentrated upon the organ, and it is closely watched; a *visceral sense* is, as it were, created, by what Dr. Carpenter has called this "expectant attention," and every shade of sensation about the epigastrium noted and exaggerated.\* With the mind's eye fixed steadily and microscopically upon one organ, such persons are miserable, dejected, and helpless. Some remarkable instances of perverted sensibility in this region have been told; in one there was a sensation of a bright flash of light, and in others darkness in the same part was complained of.

Again, over-sensibility of the stomach, causing actual and severe pain, generally paroxysmal, and most often coming on at a variable period after eating, though not infrequently when the stomach is empty, is a wearisome and obstinate symptom in these affections, and may be the chief or only morbid manifestation. This form of pain is described under the names of *spasm of the stomach*, *gastrodynia*, *gastralgia*, when unassociated with structural lesion. It varies from a sense of constriction to the severest kind of colic or gripe. It may be continuously, or intermittingly gnawing, or burning, or aching; and extend through to the back between the shoulder blades; it often is eased by deep pressure; flatulence or pyrosis constantly accompanies it, and, in such cases, ceases when the wind or the sour fluid are got rid of.

The principal derangements of secretion are *heartburn* (cardialgia) and *waterbrash* (pyrosis). Though the urgent symptom of the first is a burning, caustic feeling in the stomach, the condition is a secretory change, and the production of an intensely acrid fluid, which is constantly ejected, sometimes along with gas, scalding the fauces and mouth, and causing most disagreeable sensations and soreness about the epigastrium. In *waterbrash*, a large quantity of acid, or alkaline, or muco-alkaline, fluid is secreted, and eructated from the stomach. Another perversion of gastric secretion is the generation of gas by the mucous membrane, though it is generally the result of a weakened state of the digestive powers, and a defective gastric juice, permitting fermentation and putrefaction to go on. At times, when the atony of the muscular fibres is great, the distension of the stomach may be enormous.

The appetite may fall off (anorexia), the sense of hunger being lost, and replaced by a præcordial sinking or faintness. The thought of eating excites loathing and disgust, and food has to be "forced down," and then is apt to be followed by uneasy feelings. Romberg says: "The psychical relations of the sense of hunger are altered; there is no appetite, and taking food affords no enjoyment." Or the appetite may be voracious (bulimia); there is an excessive longing for food, and though eating at all hours of the day and night, it is unsatisfied, and when food is withheld, there is sickness, pain, and faintness. This is often associated with a condition of digestion to be presently noticed. And, finally, there is depravation of appetite (pica), sometimes coexisting with bu-

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\* [The production of actual disease by the habit of centring the attention on internal sensations of a subjective kind, has been commented on by Sir Henry Holland. He says of the hypochondriac: "In fixing his consciousness with morbid intentness on certain organs, he creates not merely disordered sensations, but often also disordered actions in them. There may be . . . flatulence and other distress of the stomach . . . all arising from this morbid direction of attention to the organs in question." And again: "A similar direction of consciousness to the region of the stomach creates in this part a sense of weight, oppression, or other less definite uneasiness; and, when the stomach is full, appears greatly to disturb the due digestion of the food." (*Medical Notes and Reflections*, chap. v.)]

limia, but which is more frequent in these disturbances of innervation. A craving for unnatural articles, as chalk, slate pencils, &c., and fancies for the most incredible and loathsome substances, or a desire for immoderate indulgence in articles of food usually taken in small quantities, is common. This condition is frequent in hysteria, chlorosis, and pregnancy.

Nausea and vomiting occur in hysterical cases, and food is rejected almost as soon as swallowed. It may be constant or remittent, days or weeks elapsing between the attacks, the appetite and digestion being natural during the intervals. Bricquet remarks that often in hysterical women, even when every meal is thrown up almost as soon as it reaches the stomach, the appetite is unimpaired, or even increased. More frequently, however, there is no desire for food, and constant nausea. In many cases of hysterical vomiting there is no body-waste.

The digestive process is generally, as has been seen, more or less weakened in these several neuroses of the stomach; still in some instances, in spite of the local uneasiness, pain, and hypersecretion, the act of digestion goes on, and the patient is fairly nourished. This especially happens in hysteria.

The associated symptoms are very various, and depend on the complaint with which the stomach-disorder is connected. Anæmia, nervous excitability, flutterings about the heart and palpitations, beatings in the abdominal aorta and in the carotid, breathlessness, vertigo, fainting spells, copious discharges of pale urine, constipation, or nervous diarrhoea, may one or all be met with. The tongue is commonly clean, but pale, broad, and flabby.

2. Amongst the instances of disordered gastric innervation produced by causes acting at a distance are, pain and sickness from a blow on the testicle, or from inflammation of that gland, diseases of the ovaries and kidneys, uterine derangements, and the state of pregnancy. A case is reported by Liederer, and cited by Fox, of a young lady, in whom a false tooth fixed to the socket of a diseased one brought on regularly returning attacks of pain and vomiting, which ceased immediately on removal of the pressure from the nerve. Barras mentions two cases in which pain in the stomach was connected with suppression of the menses, and Niemeyer one in which there was pain at each menstrual period, and which could be brought on by the application of leeches to the neck of the womb. In flexions of the womb, nausea and vomiting are not infrequent, particularly at a period when the organ is congested, and one of the most obstinate cases the writer has seen, followed the operation of neck-slitting in the surgical treatment of dysmenorrhœa. In all such cases, general nutrition is faulty, and the subjects emotionally impressionable.

**Pathogeny and Causes.**—These have been for the most part anticipated in the brief just given of the symptoms. The functional derangements of the stomach from disordered innervation, are much more frequent in the female than in the male; Georget says in the proportion of ten to one. They are most often met with between fifteen and forty-five years of age, and in women particularly at the beginning and ending of uterine activity. In men they happen in the ill-nourished, and in those whose brains are exhausted by over-work. Long fasting, venereal excesses, masturbation, are named amongst the exciting causes. In chlorosis, hysteria, and hypochondriasis, they are constant complications. They follow the abuse of tea and coffee, particularly the former, and perhaps of tobacco. The action of the stomach is largely influenced by the emotions, and digestion may be checked, the secretions arrested or increased, and the sense of hunger destroyed, by the influence of terror, anxiety, fright, disgust, &c. The

subjects of lead or malarial toxæmia are liable to temporary nervous gastric disorders. The frequent association of vomiting with diseases of the womb has been already stated; while, with respect to frequency, though not perhaps to severity, leucorrhœa and disordered menstruation have the first rank among this class of etiological conditions. When occurring in pregnancy, it is far more common amongst the weakly and nervous than the healthy (Fox).<sup>\*</sup> It may be stated in general terms, that except under the temporary influence of some known specific exciting cause, as an error of diet, these affections happen almost exclusively in persons of enfeebled vitality, and with consequent nervous erethism.

**Diagnosis.**—Most of the morbid phenomena described as happening in the neuroses, occur also in those affections of the stomach marked by structural changes. The distinction between them and inflammatory conditions of the stomach was pointed out long ago by Whytt, who wrote: “An uncommon delicacy of the nerves of the stomach, which may be either in a great measure natural, or brought on by disease, excessive grief, or other causes, is to be distinguished from that increased sensibility, which is the consequence of an inflammation, or of an aphthous state of those parts” (Works, p. 544). It becomes important to be able to discriminate between the two conditions of the organ. In a great measure an opinion must be founded on the general state of the patient, and the nature of the associated disorders. The difficulty of diagnosis is greatest in the early stages of chronic ulcer and cancer of the stomach. The tongue and the body-temperature do not give much help, for their appearances are variable and uncertain in the latter complaints. The remitting or paroxysmal type of the hyperæsthesia, and its association with other hyperæsthesiæ (ROMBERG), are valuable, though not always reliable, aids. The absence of any cachexy will often direct to a correct diagnosis.

**Prognosis.**—The duration of these complaints is very variable, depending upon the nature of the associated disorder, though generally they are obstinate and enduring, while again they will disappear suddenly, particularly as the patient gets older. Though annoying, they do not compromise life, except so far as they may give rise to organic disease, at present a disputed question.

**Treatment.**—The management of these cases, to be at all successful, should be directed to the improvement of the lowered systemic vitality, on the principles already given. With a healthy state of general nutrition, the stomach-troubles will be lessened, and finally pass away. Local disorders, as uterine affections, giving rise to reflex irritations, must be removed.

Innumerable remedies for relieving the immediate gastric symptoms have been recommended. In gastrodynia, the sulphate of iron in one- or two-grain doses has been praised by Abercrombie, in combination with aloes and compound aromatic powder; and Hensch prescribes it alone,

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<sup>\*</sup> The connection between uterine disorders and functional disturbance of the stomach, associated with defective systemic tone, as to relative frequency of cause and effect, is according to Dr. Wilson Fox's experience as follows:

a. (1) General asthenia without special symptoms, followed by (2) disturbed menstruation or leucorrhœa. (3) Sympathetic disorder of stomach (pain, &c.).

b. (1) Dyspeptic symptoms. (2) General asthenia. (3) Disorder of uterus. (4) Aggravation of stomach symptoms.

c. (1) Disorder of uterus. (2) General asthenia and disorder of stomach, proceeding *pari passu*.

d. In rarer instances—(1) Disorder of uterus. (2) Derangement of stomach, which apparently induces a depressed condition of the general system. (*The Diagnosis and Treatment of the Varieties of Dyspepsia*, London: 1867.)

or with morphia. Iron should not be persisted in one time. The oxide and nitrate of silver have Leared tells of complete and rapid success with (Fowler's solution), especially when the neuralgia. Whytt, Barras, and others insist upon opium; it of hypodermic injections of morphia. Hydro laurel water succeed in milder cases, and Dr. F. when associated with bismuth. The testimony is strong and general, and "it frequently prepares bear ferruginous preparations, which had been (Fox). The writer has combined it with small advantage. The application of a belladonna plaster and after the latter dusting the reddened skin powdered camphor, will frequently give relief. Faradization. Pain from flatulence is best treated of ammonia, sulphuric ether, and tincture of nux.

In the wearying uneasiness at the pit of the stomach (trique), Trousseau speaks highly of valerian and compress, over which a piece of oil silk and a bandage for some hours, will often remove it.

In pyrosis, the carbonate or nitrate of bismuth, magnesia and opium, followed by kino powder. Flatulence may be abated by aromatics and charcoal made from vegetable ivory is the best.

Vomiting is often very difficult to check, particularly caused by uterine trouble or pregnancy. Besides cold effervescing draughts, a few drops of laudanum (and permanently efficacious), the oxalate of cerium and tincture of nux vomica, Faradization, pyrexia given. A small cup of coffee, or some slight nourishment be taken before the patient rises in the morning. there seems to be little doubt of its real efficacy. attempts to discredit it; Dr. Chambers says its enabling the food to be retained, whereby the heart is kept up. Where the bowels are sluggish, or there is accumulation of feces should be avoided.

In all these functional derangements, it may be the palliative drug from time to time.]

## DISEASES OF THE INTESTINES

**Definition.**—*Under this heading, as with stomach, is proposed to notice the more certainly ascertained or the lesser intestinal canal, expressed by function, texture, or both.*

**Pathology.**—The diseases usually named are enteritis, ulceration, perforation, abscess in the stomach, abscess, fistula (fecal), hemorrhage, melana, distension, stricture, intussusception, internal stricture.

Although enteritis is generally described as if it is a rare disease, and seldom affects the intestine in whole extent. The several parts under which it is described are variously influenced by local it

The peculiarities of anatomical organization, such as the various forms of minute glandular parts, determine in some measure the forms by which these organic lesions are expressed. With some modifications, explained by such peculiarities of structure, the *organic* lesions of the lesser intestinal tract are in many respects precisely similar to those described in the stomach; while the functional disorders are indicated by the various forms in which the intestines express irritation or perverted action, such as by *spasms*, *colic*, *flatulence*, *indigestion*, *constipation*, and various forms of *fluxes* or *diarrhœa*.

I. **Softening of Tissue** has been described in the small intestines similar to that affecting the stomach, but it occurs much less frequently, and is not expressed by any recognizable clinical phenomena.

II. **Glandular Lesions and Degenerations** are a much more frequent occurrence, if not a constant one, over limited portions of the intestine. The lesions are peculiar, from the structure of the parts; and the *degenerations*, as they are termed, are analogous to those described by Dr. Jones as occurring in the stomach (see p. 857). As in the stomach, so in the intestines, there is to be observed in some cases, on the one hand, an entire destruction of the gland-tissue, with actual loss of substance, so that when the mucous membrane is delicately dissected from the muscular parts, it may be viewed as a transparent object with a lens; on the other hand, there may be no actual loss of bulk or of substance, while the structure is nevertheless greatly altered—(1.) By alterations of the contents of the mucous tubes, without change of form; (2.) By an interstitial deposit of fibrinous matter encroaching upon and ultimately obliterating the proper glandular tissue. Thus, externally and in bulk, the parts may seem to be unchanged; but they are found to be materially altered in specific weight, and the range of alteration is considerable. Thus, through lesions or degenerations of the gland-tissue, *atrophy* of the mucous membrane of the intestines becomes expressed in two ways (as originally expressed by Dr. Bucknill with reference to the nerve-substance of the brain), namely,—(1.) *Positive atrophy*, in which the tissue of the gut wastes, while the glandular texture is altered; (2.) The tissue of the gut may not have wasted; on the contrary, it may have gained in bulk, but the glandular tissue has been changed, or been altogether replaced; while (3.) The two conditions may be coexistent.

The observations which appear to me to prove these statements are, alterations in the specific gravity of the mucous membrane of the intestine, associated with characteristic morbid appearances, as shown by microscopic examination of sections. These combined modes of examination, not only in mucous membrane, but in all parts in which I have applied the test, have yielded one characteristic result—namely, that atrophic states of prolonged duration, which are generally described as *granular degenerations* of minute tissue, are, for the most part, of comparatively low specific gravity, and with a chemical reaction under the microscope, indicative of the presence of fat; while, on the contrary, the acute inflammatory conditions of tissue (where loss of peculiar minute structures,



Like the glands of the intestines, is due to destruction from exudation and replacement by it; have uniformly high specific gravities. These results are similar to those which Dr. Bucknill has expressed with reference to the brain (*Med. Chir. Review*, January, 1855, p. 212). A specific gravity of 1.032 to 1.33 of the mucous membrane, when it is free from congestion, may be regarded as associated with the healthy state of the gland-tissue. In conditions of *positive congestion* throughout, it descends to 1.030; while in conditions of *relative congestion*, where bulk is unchanged, but where glandular parts are displaced or destroyed by exudative deposits, the range of specific weight of parts examined has been as high as 1.044, and in the large intestines as high as 1.050. The specific gravity of Peyer's patches I have found to vary from 1.032 to 1.044, and even in patches from the same intestine there is often a considerable latitude in the range of the specific weights of the glands. The atrophic states of these glands are indicated—(1.) By a reticulated condition, void of all glandular elements, and which seems to occur naturally with the advance of years beyond forty: or to occur after extensive *infarction* during the progress of typhoid fever, the elimination of the product thus taking place without ulceration. (2.) By the remains of the cicatrices of ulceration in the form of dark granular deposits: or of a thin, clear, skin-like membrane, of a pale color, and with a wrinkled contracted border. These morbid changes are most commonly associated with the prolonged continuance of complex morbid processes, as in constitutional diseases, such as those of anæmia (p. 88, *ante*), or in organic lesion leading to impairment of the constitution generally. They are also associated and expressed more or less locally amongst the peculiarly local glands or accumulations of glands of the gut, such as the solitary lenticular glands and the patches of Peyer, during the progress of some zymotic diseases, such as in cases of *cholera*, *dysentery*, and *typhoid fever*.

The definite morbid conditions in which the glandular apparatus may be found are as follows: (1.) *Intumescence, stuffing, or cramming* of the gland, by different kinds of deposit, by debris of normal elements, or of both, and associated with redness and vascular congestion of the submucous tissue—conditions which correspond to the so-called "*infarction*" of the older pathologists; (2.) *Softening, degeneration, and elimination* of the abnormal constituents in various ways; (3.) *Ulceration*, sometimes leading to sloughing, of whole patches of glands; (4.) *Collapse of solitary glands*, or otherwise cicatrized remains of ulcerations of solitary and aggregate glands; (5.) *Accumulation of melanic matter*, characteristic of pre-existent and long-continued vascular action, with changes in the tissue from hæmatine (*Pigmentary degeneration*; see vol. i, p. 121); (6.) *Lardaceous degeneration* (see vol. i, p. 124).

The cramming of the glands, associated with submucous vascularity, is generally due to a milky-like exudation, with the variously metamorphosed epithelial elements. Such exudation may subsequently undergo various kinds of metamorphoses yet to be determined; but generally it may be stated that *corpuscular* and *fibrinous*

elements coexist in the various forms in which the glands are found intumescent.

There can be no doubt, from the observations of Dr. Handfield Jones, as well as from what is consistent with daily observation, that many of these obvious changes go on independently of any expressions of the phenomena of inflammation as commonly understood. But it is now well known also that the undoubted results of the inflammatory process sometimes occur without the manifestation of symptoms, such as *pleuritic effusions*, and which yield to appropriate remedies. In such cases the inflammation has been considered and described as latent. Does it not therefore appear consistent to associate the phenomena of such latent inflammations with the phenomena described by Goodsir and Redfern as the first stage of lesion in the cartilage-cell; with those of the early degeneration in the epithelium of the uriniferous tubes in Bright's disease; with the degeneration of involuntary muscle described by Quain; with the changes described by Virchow in the cornea, by Bucknill and Skae in the brain; with the early changes in the epithelium of the mucous surface of the bronchi, where they lose their cohesion, separate with abnormal rapidity, and ultimately assume forms and characters not to be distinguished from pus; and, lastly, with the phenomena of alteration and degeneration of the minute contents of these glandular parts now described; and consider such changes as expressions of one and the same complex morbid process, and which Virchow has described by the name of *parenchymatous inflammation*? (See vol. i, p. 107.) The ultimate results and further expression of the inflammatory process varies, according to the texture of the part and other circumstances; but here we have the initiative of the process similar in all tissues, while its ultimate results are various; and while the complex state known as "fever" has found a constant expression in "the elevation of temperature," may we not be entitled to say that "the no less complex process of inflammation" has a no less constant expression in the diminished cohesion of the minute elements of tissue?

There seems to be conclusive evidence to show that much of the intestinal catarrh described by the common name of diarrhoea is associated with an *erythematous* congestion of the mucous surface of the lesser intestine, extending over a considerable extent, and rarely attended by increased arterial vascularity of the submucous tissue. When the symptoms of such congestive states are manifest during the progress of other diseases which terminate fatally, there may frequently be observed, besides the congested state of the mucous membrane, a marked increase of vascularity in other parts, such as the gastro-splenic omentum, mesentery, and glands, or infarction of the gastric glands, associated with congestion of the stomach generally. These phenomena for the most part are associated with a congested state of the hepatic system; and, occurring in a person otherwise in good health, give rise to symptoms which have been considered as a disease, and variously named *enteria*, *enteritis*, *erythemoidea*, *diarrhoea mucosa*, seu *catarrhosa* vel *catarrhale*. During the autumn and winter months in this country it is common to meet

with such cases of disordered bowels in adults; and in children at any season, characterized by frequent fluid alvine discharges, and associated with extensive superficial irritation of the mucous surface. When the irritation predominates towards the upper part of the intestine—in the duodenum, for instance—the symptoms are an inclination to sickness, speedily followed by copious feculent discharges; the surface is easily affected by cold, and the individual may even shiver. There is also thirst, and a feeling of internal heat over the epigastric region. The functions of the liver are manifestly disordered at an early period, as expressed by the dull yellowish color of the conjunctiva, and sallow darkness of the complexion, especially round the eyes. The tongue is generally moist, but viscid, clammy, and furred. The appetite is completely lost in the first instance. The skin is dry, and the palms of the hands and soles of the feet become unpleasantly hot and burning. The bowels generally become distended with flatus; and there is an uncomfortable sensation of distension, incapacity to expel the air, and occasional griping of the bowels, which are constantly producing a rumbling noise. The stools are at first large, feculent, and consistent, but subsequently they become watery, and even mixed with blood; then *tenesmus*, or a tendency to strain at stool, comes on and increases. Undigested articles of food are also passed—the characteristic symptom of *lientery* or *diarrhœa crapulosa* of the older authors. The belly is not painful when pressed, as in peritonitis or acute inflammation of the bowel, but there is often a deep-seated sense of uneasiness. This state soon terminates, in general favorably. It is more especially brought on by exposure to great changes of temperature in humid and moist weather, by wet feet, damp beds or clothing, and improper dieting (after over-feeding, especially in summer weather) at irregular times; certain articles of food, imperfectly fermented malt liquors, acid wines, and sour unripe fruits, drastic purgatives, and various mineral poisons.

A diarrhœa of whitish stools is often extremely persistent, and may reduce the patient to the very verge of death. It may supervene after injuries which may induce cerebral concussion; or it may come on after dysenteries have been cured. There may be no fever, the appetite may be good, and digestion may seem tolerable, but emaciation and weakness become daily more and more marked. At first the motions only increase to two, in place of the usual single daily one. Afterwards the calls to stool increase, so that, during the twenty-four hours, there may be eight or ten in the day. The desire to evacuate the rectum becomes sudden; and the stools are apt to pass involuntarily, preceded by little or no premonitory sensations, and consisting merely of two or three tablespoonfuls of muco-gelatinous matter, resembling thick milk or puriform fluid, or like a jelly. A similar state of mucous membrane lining the small gut gives rise to a white secretion from its surface; so that white milky stools are observed to flow—*Diarrhœa alba* of Hilary—a form of bowel disease which is sometimes epidemic in Barbadoes. No remedy is of so much service in this affection as the *extract of nux vomica*, to the extent of a fourth to a half grain dose;

or *strychnia* to the extent of *one-twelfth* of a grain, in a pill twice or thrice a day, with the *sulphate of iron* and *extract of calumba*. Dr. Maclean gave *tincture of the perntrate of iron*, gr. x every half hour, in this form of diarrhœa, with great benefit; and it is in diarrhœa of this kind that iron is of so much service. My attention has been recently called to a new preparation of iron by Mr. W. A. Moss (dispenser of medicines in the Army Hospital Corps at Dublin), which seems to possess some desirable properties, especially as to solubility and freedom from the inky astringent taste of preparations of iron. Its preparation is given in the note below.\*

Black or dark stools (*melæna*) are not so much due to bile (*atrabilis* of Abernethy) as that such stools, resembling pitch, are principally composed of morbid or impaired secretions from the intestines (HOFFMAN, HOME, GRAVES). In such cases the discharge of the black matter is followed by a feeling of relief to the system generally. In cases of true *melæna*, where the dark color is due to blood, great debility and sometimes fainting may follow the evacuations. Stimulating and tonic remedies, such as turpentine, are of benefit (GRAVES).

**III. Inflammation of the Bowel, or Enteritis.**—The phenomena of inflammation, when they do occur, generally express themselves in the ileum; and *Typhlitis* is understood to mean an inflammation of the cæcum.

Acute diffuse inflammation is marked by redness, thickening, and impaired cohesion. The redness is of a *deep venous red* approaching to blackness, either partial or general in extent, and in dotted, arborescent, or striated patches. It is distinguished from mere passive congestion by the increased arterial vascularity of the submucous tissue. The thickening is generally sensible, and often considerable. The impaired cohesion is not so obvious as in the stomach, but the mucous membrane may be removed much more easily than in health, from its attachment to the parts subjacent. In the chronic forms of diffuse inflammation the color, thickening, and cohesion of the gut are not greatly changed; but in general the thickness is more considerable, the cohesion of parts, instead of being impaired, is often rendered more tenacious, while the dark venous hue, on subsiding, leaves a grayish or slate-colored tint, from a deposit of melanic matter in the substance of the membrane.

Serous inflammation of the mucous membrane of the small intestines may be inferred to exist from the large quantities of serous fluid often discharged by stool during life, at the same time that the abdomen is the seat of pain and tenderness. After death the

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\* *Ferri Ammonio-Phosphas.*—Heat common phosphate of soda to redness. Take of the pyrophosphate of soda so obtained ℥ij. Dissolve in one pint of warm water. Then take of protosulphate of iron ℥iv. Dissolve in twelve ounces of water. Mix the solutions, collect, wash, and dry the precipitate at a gentle heat over a water-bath. Take of this precipitate ℥j, Liq. Ammonia P. L. ℥iss, water q. s. Dilute the Liq. Ammonia with an equal volume of water, and rub up with the phosphate of iron in a mortar until the latter is dissolved. Then dilute to ℥viiij. Filter the solution, and evaporate at a heat not exceeding 120° Fahr., over a water-bath, and proceed as for the other scale preparations of iron. The dose is one fluid drachm.

fact may be proved by the loose diffuent fecal matter often found in the small intestine: at the same time the mucous membrane is partially or generally inflamed.

The effusion of coagulable or fibrinous lymph in the small intestines is an extremely rare occurrence. "I have," says Dr. Baillie, "seen in violent inflammation scattered portions of coagulable lymph thrown out upon the surface of the villous membrane. This, however, is very uncommon" (p. 158). Billard has seen it but twice in the intestines of children. Dr. Handfield Jones also notices that the surface is sometimes the seat of an exudation much resembling that of croup; the attacks recurring several times, each presenting a stage of irritation, which ends in the formation and throwing off of a false membrane. This membrane sometimes forms a layer of some thickness, extending pretty uniformly over the surface, or appearing in the stools as tubular casts of the intestines, and sometimes it is as thin as a wafer, or consists merely of tattered shreds. In one case mentioned by Dr. Copland there were shreds of dysmenorrhœal false membrane discharged from the uterus, but not at the same time (*Pathological Anatomy*, by Jones and Sieveking, p. 526). The diphtheritic exudations described by Rokitansky are of a similar nature. Pseudo-membranous inflammations of the bowels have also been described by Dr. W. Cumming, of Edinburgh, and Dr. Simpson.

IV. **Ulceration** is much more common, and is indeed by no means unfrequent, especially from the action of typhoid and malaria poisons; and this ulceration may take place either at the free or adherent surface of the membrane. When it takes place at the free surface, the ulcer, says Andral, may form in the centre of a point of inflammation, the mucous membrane around being healthy, or it may form in the midst of an extended patch of diffuse inflammation, without the follicular structure appearing to be in any degree affected. Again, the submucous tissue may inflame and become the seat of a number of small abscesses, which may point like so many pustules of small-pox. The apices of these abscesses become thinned and softened, till at length the mucous membrane ruptures, and the pus they contain is poured into the cavity of the intestine. These constitute miliary vesicles, entirely of morbid origin from the first, and are to be distinguished from the infarction and ulceration of solitary or lenticular glands. The form, edge, and base of these ulcers are not unlike those found in the stomach, except that the ulcer with a sharp perpendicular edge, as if made by a punch, is much more rarely seen.

Besides *inflammation* and ulceration of the membrane over its general surface, the solitary glands may be either separately or conjointly with the former the seat of inflammation and of ulceration, subsequent collapse, atrophy, and obliteration.

The glands of the small intestines are thus liable to be enlarged and transparent, looking like a drop of pellucid water from serum, having a small black point in the centre, which is the mouth of the duct leading to the distended follicle. When the patches of Peyer are ulcerated, the ulcers generally take the oval form of the



patch, and these ulcers sometimes burrow so deep as to rupture the intestine. As these forms of lesion, however, principally occur in typhoid fever, dysentery, and cholera, as a result of the morbid process, the reader is referred to those articles.

Inflammation of the intestines, says Dr. Baillie, sometimes, although rarely, advances to mortification. When it does so, the mortified part assumes a dark livid color, loses its tenacity, and is very readily torn.

Ulceration and mortification sometimes lead to the perforation or rupture of the intestine, when, the contents of the bowels escaping into the cavity of the abdomen, the patient dies of peritonitis.

The symptoms of *enteritis* partake more or less of those already stated as belonging to *intestinal catarrh*, combined with pain, aggravated by pressure, as a characteristic. The chief seat of pain is generally about the umbilicus, or the right iliac fossa. The discharges from the bowels relieve for the moment the griping pains. The pulse is excited, and generally full and strong.

The absence of intense pain and tenderness, of vomiting, of constipation, of excessive vomiting and tympanitis, of the small and frequent pulse, all of which are characteristic of peritonitis, is sufficient to distinguish enteritis from that disease.

**V. Waxy or Lardaceous Degeneration of the Intestinal Mucous Membrane.**—Next in frequency to the spleen, liver, and kidney degeneration of the lardaceous kind, is that of the intestines, where this degeneration especially affects the arterial capillaries of the villi, and the surrounding networks of the mucous and submucous tissue. Its progressive involvement of parts is as follows:

It is seen—(1.) In the points of villi; (2.) Involving entire villi; (3.) In the mucous and submucous capillaries of inflamed parts; (4.) As annular infiltration round solitary glands; (5.) As degeneration of the vessels surrounding the sacculi of Peyer's patches. Virchow says he has seen the whole tract of arterial capillaries from the mouth to the anus in a uniform condition of amyloid degeneration. I have repeatedly met with this condition in dead soldiers at the invaliding hospital of the army, now at Netley. Anæmia of the mucous membrane, with a peculiar glistening or shining aspect of its surface, are the most characteristic features. Otherwise, there are no outward signs of the lesion to attract attention. Pallor, anæmia, and atrophy ought to excite suspicion; but the application of the iodine reagent is absolutely necessary. The atrophy has advanced so far that Virchow has known the villi to drop off, and the intestine to be bare of villi where villi are usually present. The walls of the fine arterial twigs of bloodvessels become pellucid, transparent, glistening, rigid, and thick, and a reduction in the size of their calibre is the result. Blood ceases to pass through them, nutrition is impaired, and atrophy results, extending over large tracts of the bowel,—most decided towards the duodenum. The substance of the villi has frequently been found changed into the lardaceous or albuminoid material. Sometimes the mucous membrane is also destroyed, and ulcers are developed which penetrate deeply into the tissue. The capsules of Peyer's patches have

been seen enlarged, as well as the solitary glands (FRERICHS). Lambl has traced the degeneration and destruction of the intestinal epithelium through the substance of the villi, the follicles of Lieberkuhn, and the muscular coat itself.

**Treatment.**—The treatment of enteritis, when not arising from a morbid poison, is by leeches to the abdomen, gentle purgative medicines combined with an opiate, fomentations, and purgative or opiated enemata. After the inflammation has subsided, mild tonics, as the compound tincture of gentian with nitro-muriatic acid, may be substituted, with the prospect of recovering the lost tone of the parts.

When diarrhœa exists, if there is reason to believe that much congestion prevails connected with the hepatic region, a cathartic dose of calomel followed by castor oil may be necessary. If the stools indicate an acid reaction, *magnesia* may be given with advantage. When membranous films or shreds of coagulable lymph are passed, electro-galvanic applications over the abdomen and dorsal spinal region, combined with creasote or tar in the form of a pill, is quoted by Dr. Wood, on the authority of Drs. Cumming and Simpson, of Edinburgh, as worthy of a trial. Next to bloodletting are those remedies which determine towards the skin; and for this purpose combinations of Dover's with James's powder, or a solution of tartar emetic with laudanum, are the most useful medicines; and their beneficial action is very much aided by the use of the warm bath, and a flannel roller applied with firmness round the abdomen (CRAIGIE).

The greatest care ought to be given to the management of the diet. It ought to be strictly antiphlogistic, consisting entirely of slops and light puddings.

Animal food must be entirely withheld, and only permitted occasionally in the form of soup. The safest diets are those of arrowroot, sago, tapioca, gruel of oatmeal, *sowens*,\* barley water, toast water, burnt oatcake water. This last substance, used as a drink, is said to allay irritation and morbid sensibility of the bowels (CRAIGIE). When the abdominal pains and stools subside, and the appetite increases, the diet must be, if possible, still more scrupulously attended to. Oatmeal porridge boiled to the utmost possible degree of pulpiness, and not too consistent, is one of the best of diets, which may be alternated with ground rice, alone or combined with barley flour, all of which may be eaten with diluted milk from the cow, or with milk undiluted of town-fed cows.

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\* "The husk and some adhering starch separated from oats in the manufacture of oatmeal are sold in Scotland 'under the inconsistent name of *seeds*.' These, if infused in hot water, and allowed to become sourish in this state, yield, on expression, a mucilaginous liquid, which, on being sufficiently concentrated, forms a firm jelly known by the name of '*sowens*.' Not less than a quart of the *seeds* are to be rubbed for a considerable time with two quarts of hot water, after which the mixture is to be allowed to rest for several days till it become sour. It is then strained through a hair sieve, and the strained fluid left to rest till a white sediment subsides. The supernatant fluid is to be poured off, and the sediment washed with cold water; after which it may be either boiled with fresh water, stirring the whole time it is boiling, or it may be dried and prepared when convenient in the same manner as arrowroot. It may be eaten with wine, milk, or lemon-juice and sugar" (Pereira *On Food*, p. 326).

VI. *Colic* is a painful affection of the lower portions of the abdomen, caused by a violent contraction of the muscular fibre of some portion of the intestinal canal. The remote causes are indigestion, exposure to cold, or other general cause, the effect of lead poison; and all periods of life, from infancy to old age, are liable to the affection. It also attacks either sex. It is seldom that persons die of colic; but such instances have occurred; and dissection has shown some portion of the intestines intussuscepted—affording a strong presumption that this affection depends on a spasmodic constriction of some part of the intestinal canal. This view of the case is supported by Mr. Blane, who states that in fatal cases of colic in horses, different portions of the alimentary canal are found strongly contracted, and much oftener of the small than of the large intestines, which also sometimes contain gas. The bladder appears to participate in the spasm, the urine being either frequently ejected or suppressed. Colic, therefore, is a spasmodic contraction of the stomach or intestines,—usually sudden in its attack; and the patient consequently, without any previous indisposition, is often unexpectedly seized with a severe fixed pain in some part of the abdomen, but which is relieved on pressure, so that he either sits doubled up, or rolls on the ground, or lies flat on the belly. In other cases, where much air is secreted the bowels are greatly distended, and the pain is compared to a twisting or wringing around the navel, accompanied with soreness. The walls of the abdomen also participate in the internal spasm, so that the navel is often drawn in towards the back, or the heads of the *recti* muscles are exceedingly prominent, resembling so many round balls. The bowels are generally but not always constipated, and the stomach may or may not be irritable. In the latter case it often rejects both food and medicine. The pulse is little altered at the commencement of the attack; but if the paroxysm be prolonged, and the patient exhausted by pain, it may be hurried and frequent. The tongue is generally clean, although sometimes white and coated.

*Gastralgia*, or stomach *colic*, is a severe pain in the stomach, often so completely idiopathic that the slightest cause produces it. One person cannot eat a strawberry, another a gooseberry, another an egg, without being seized with it. In other cases every sort of diet produces it, so that the patient is racked with pain after every meal. Those affected are usually adults; and women are more frequently the subject of it than men.

The attack of *colic* is generally sudden, the patient being unexpectedly seized with a pain, which attains its greatest height on the instant, round or above the umbilicus. This attack is generally accompanied by sickness or vomiting, by great flatulence, and by a confined or sometimes by a purged state of the bowels. It may last from a few minutes to a few hours, and often ceases as soon as the stomach is emptied or the bowels have acted; but when the patient is costive, it very constantly continues till he is relieved by medicine, when it subsides almost as rapidly as it commenced, leaving, however, a soreness behind it. The pulse in this affection

is natural, there is no fever, and the pain is relieved on pressure—circumstances which readily distinguish it from inflammation. The disease may subside after one attack; but genuine gastralgia sometimes lasts for many months.

**Diagnosis.**—This disease is distinguished from inflammation by the pain being relieved on pressure, and by the quiet state of the pulse.

**Prognosis** is in every case favorable.

**Treatment.**—The treatment of colic is by opiates, chlorodyne stimulants, and purgative medicines. When the bowels are constipated, *five grains of calomel, fifteen grains of jalap, and one grain of opium* should be administered immediately, and followed by the following:

R. Mist. Camphoræ c. Magnesiae Sulphat., 3j; Tinct. Hyoscyami, ℥xv to xx; Tinct. Cardamomi, 3j. To be repeated every five or six hours until stools are obtained.

In mild cases a scruple of rhubarb, or half an ounce of castor oil, or other mild purgative, combined with a grain of opium, may be substituted for the opium, calomel, and jalap. Some have doubted the propriety of administering opiates at the onset of the disease; but it is certain that a mild purgative, combined with a mild narcotic, will effect more than a drastic purgative without such combination. Enemata often give immediate relief. Externally, the application of large bags filled with hot *chamomile flowers*, or of *heated sand*, or of the *stomach-warmer filled with hot water*, are useful. The *warm bath fomentations*, or a *large linseed or mustard poultice* over the abdomen, are also highly useful auxiliaries. Some patients are said, when these remedies have failed, to have been benefited by dashing cold water over the lower extremities; but the experiment is hazardous. The diet should, during the attack, be sago and arrowroot, with a little brandy: and for some time after the patient has recovered it should be light, and perhaps limited to fish and puddings.

VII. **Obstruction** of the intestine occurs when some obstacle mechanically impedes the passage of contents through the bowel. A typical case is thus related by the late Dr. Brinton, whose monograph on this subject ought to be read by every student. He did more in his too short life to elucidate the pathology of such cases than any one who has written on this subject:

“A person, perhaps hitherto healthy, experiences a sudden constipation, attended with disproportionate uneasiness, or flatulence, soon merging into pain and distension of the belly, with violent rolling movement of the intestines. The distension increasing, nausea and vomiting supervene; and gradually becoming more frequent, end by rejecting not merely any casual alimentary contents of the stomach, or the greenish, bilious, alkaline fluid commonly thrown up when this organ is unoccupied by food, but a fluid of greater opacity, color, and consistence, with a distinctly fecal odor. A further aggravation of these symptoms now conducts the malady to its termination. This, if fatal, is usually preceded locally by signs of paralysis, inflammation, or even rupture of the distended bowel, and constitutionally, by exhaustion or collapse replacing a febrile reaction. In other cases, the obstacle being removed by nature or

art (if by the former, rarely before life is in extreme danger), the symptoms subside with comparative celerity. The pain, distension, and vomiting cease; the bowels are relieved by copious stools; and the patient (if not placed in further peril by any of those conditions incidental or consecutive to obstruction just hinted at) is rapidly restored to comparative health" (*Intestinal Obstruction*, p. 6).

The most remarkable and most characteristic symptom of intestinal obstruction is the fecal vomiting, explained by a doctrine that remained almost unquestioned since the time of Galen, until the observations and experiments of Dr. Brinton exposed the error, and established the pathology of intestinal obstruction on a rational basis. When, about twenty years ago, he exposed the error of the doctrine, then and even now entertained, it was supposed that fecal vomiting was effected by an *anti*-peristaltic movement of the intestinal canal; that, at a certain stage of obstruction, the natural peristaltic action of the bowel above the occluded point was reversed: so that instead of proceeding towards the anus, as heretofore, it took the contrary direction,—thus impelling the intestinal contents in a similarly retrograde course, so as to return them to the stomach, whence they were vomited.

Dr. Brinton showed by abundant proof, experimental and incidental, that the notion of *anti*-peristalsis was contradicted by direct observation, and a careful study of the phenomena of intestinal obstruction, as witnessed in the human subject, and as artificially produced in experiments on animals, led him to the following theory:

"The movement proper to the healthy intestine is a circular constriction or peristalsis, which, travelling slowly and intermittently down its muscular wall, propels its contents in a direction from the stomach to the anus. And when any part of the intestine has its cavity obliterated by an immovable mechanical obstacle, its contents, propelled by such a peristalsis, are stopped at the obstructed point. Here they gradually accumulate, so as first to fill and then to distend a variable length of the canal with a more or less liquid mass. But a peristalsis engaging the wall of a closed tube filled with liquid, and falling short of obliterating its calibre, sets up two currents in that liquid;—one at the surface or periphery of the tube, having the direction of the peristalsis itself, and one in its

FIG. 66.

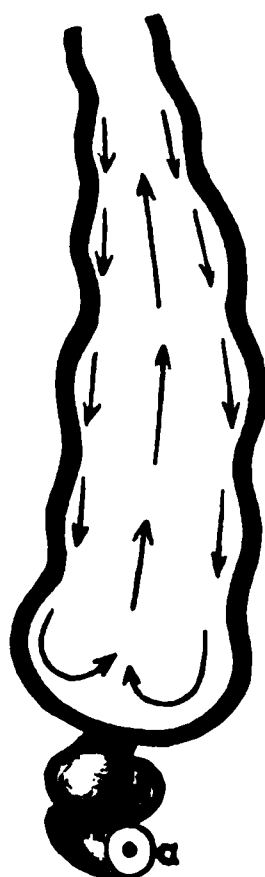
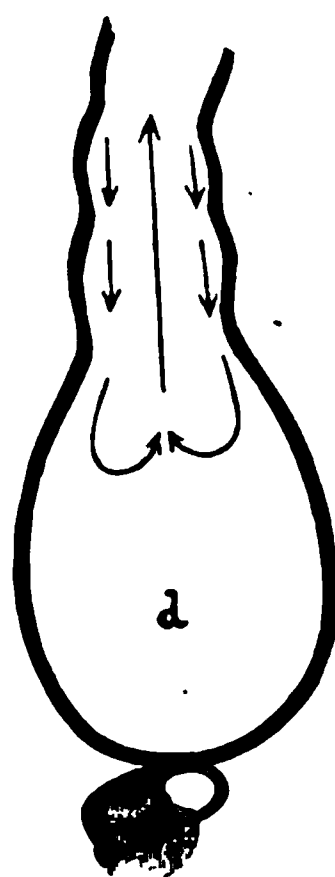


FIG. 67.\*



\* Figs. 66 and 67: Diagrams to illustrate the peristalsis of an obstructed bowel, after DR. BRINTON.—Fig. 66. Stage of moderate distension, with forward and backward currents, as indicated by the arrows traversing the whole tube above the obstacle; (a) Contracted segment of intestine below the obstacle. Fig. 67. Stage of extreme distension, in which (d) the dilated and paralyzed segment above the obstacle is scarcely engaged by either of these currents.



centre or axis, having precisely the reverse course. Those particles of the liquid which are in contact with the inner surface of the tube are propelled onwards by the muscular contraction of its wall. And this propulsion is necessarily accompanied by a backward current in those particles which occupy the axis or centre of the canal."

Fecal vomiting is thus shown to result from the reflux of the intestinal peristalsis—a backward current in the liquids occupying the centre of the tube.

The ordinary course of intestinal obstruction from Dr. Brinton's point of view divides itself into two stages. In the first stage the healthy actions of the bowel are continued; in the second stage they are arrested or utterly and permanently annihilated by paralysis, enteritis, or peritonitis.

In the first stage, abnormal *distension* of the intestine can generally be felt through the yielding wall of the belly—as a condition of fecal vomiting—from the very commencement, and continuing a persistent physical sign through all the stages of the obstruction. Experience led Dr. Brinton to assert "that the accumulation of intestinal contents immediately above the obstructed point may sometimes be detected as a slight fulness to palpation, and a much more definite dulness to percussion, where many of the other indications of obstruction are scarcely perceptible, or even absent." The movements of the obstructed intestine may thus be traced rising visibly against the walls of the belly covering the obstructed tube, "in coils that may be fancifully compared to those of a writhing serpent," until the paralysis and collapse usher in the fatal issue.

The character of the pain in obstruction is variable. It is sometimes sudden and violent, and often rises to great intensity in a very short time. It is distinct from the burning pain of peritonitis. It is usually intense in intussusception, and in the impaction of gall-stones; somewhat less marked in the obstruction produced by twisting of the bowel, or by bands and adhesions; scanty in the obstruction of stricture; and almost absent in the obstruction caused by the impaction of feces in the large intestine (BRINTON).

Exclusive of hernia, Dr. Brinton has estimated, from an analysis of 12,000 necropsies, that obstructions of the intestine cause about 1 in every 280 deaths from all diseases indifferently; and the chief varieties of obstructions have to each other the following proportionate frequency: Intussusceptions or invaginations, 43 per cent.; obstructions by bands, adhesions, diverticuli, or peritoneum, external to the bowel, 31½ per cent.; strictures (including a few tumors) involving the intestinal wall, 17½ per cent.; torsion of the bowel on its axis, 8 per cent.

The forms of intestinal obstruction are mainly as follows:

(a.) Intussusception, of which the varieties are,—*Ileo-cæcal*, 56 per cent.; *Iliac*, 28 per cent.; *Jejunal*, 6 per cent.; *Colic*, 12 per cent.

(b.) Obstructions due to bands, adhesions, diverticula, gall-stones, lesions, such as rupture of *mesentery*, and other peritoneal lesions. The small intestine is the seat of the obstacle in 94.53 cases per cent.

(c.) *Obstruction due to strictures, tumors, or twistings of the bowel and mesentery*, give about 87.36 per cent. of cases involving the large intestine.

*Intussusception* is the accidental insertion or protrusion of an upper into a lower segment of intestine. It occurs more frequently in infancy and childhood than at any other period of life. Of twenty-five cases observed or collected by Rilliet, *seven* occurred in children of six months or under; *six* during the first year of life; *seven* between five and ten years of age; *five* between ten and under fifteen years of age. Dr. Brinton's experience goes to show that half the ileo-cæcal intussusceptions are infants under seven years of age; many but a few months old; and he gives the average ages of the ileo-cæcal, jejunal, and colic respectively as 18.57, 34.6, and 31.4 years.

Invaginations of the small intestines are so frequently found after death, in comparatively young and well-nourished subjects, that it is generally believed they are formed with great facility, and that they often occur during life, giving rise to temporary bowel derangement; but that they also soon become disentangled again by the normal peristaltic movements. Of 300 children examined by Louis at the Salpêtrière Hospital, and who died there, the greater number had two, three, or more *volvuli* without any inflammation of the parts; and there were no circumstances in their history during life which led to the suspicion that these children suffered from intussusception (*Mem. de l'Acad.*, vol. iv). Dr. Baillie, also, in his great work *On Morbid Anatomy*, says, "In opening bodies, particularly of infants, an intussusception is not unfrequently found which had been attended by no mischief; the parts appear perfectly free from inflammation, and they would probably have been easily disentangled from each other by their natural peristaltic motion." Dr. Macintosh states that he scarcely ever opened a child without finding partial invagination of the small intestines (*Practice of Physic*, vol. i, p. 256). Dr. Hodgkin, in his valuable work *On the Morbid Anatomy of the Mucous Membranes*, and Rokitsansky in his *Pathological Anatomy*, each make mention of the frequent occurrence of invaginations in the bodies of adults as well as of children, and they consider them to be produced in the majority of instances during the last moments of life—in the death-struggle, or in the *rigor mortis* of the dead intestine. I have frequently observed such invaginations in post-mortem examinations, not associated with symptoms during life, and easily reduced by traction; but they have been generally in cases where the irritability of the bowel had been greatly increased during life by excessive diarrhœa, with or without ulcerations of the intestines. Such invaginations were invariably in the small intestines.

Cases of intussusception *in the adult* are rare—so rare, indeed, that in the extensive experience of one of the largest civil hospitals in London (Guy's), Dr. Wilks records that "he has never seen but one case of intussusception *in an adult*, and in this case the obstruction was never complete, and death did not occur for some weeks" (*Pathological Anatomy*, p. 292). In the *Transactions of the*

*Pathological Society of London*, extending over the first fifteen years of its existence, there are only seven cases of intussusception in the adult on record—no two of which occurred in the individual experience of any one man. The ages of these seven cases are respectively as follows—namely, eighteen, twenty-five, thirty-two, thirty-four, forty-one, and two at forty-four years of age. In one case the symptoms continued for three months, and at last ended in recovery after the passage of a portion of ileum (containing a polypoid tumor) by the rectum. In another case the symptoms continued during four months, and terminated fatally by exhaustion. In my own experience I have never made a post-mortem examination of a case of intussusception in an adult, nor have I ever seen a case of intussusception in an adult during life. In the Museum of the Army Medical Department at Netley there are preparations showing the lesions and morbid relations of the parts preserved, from at least eight cases occurring in soldiers at ages varying from twenty to forty-two years of age.

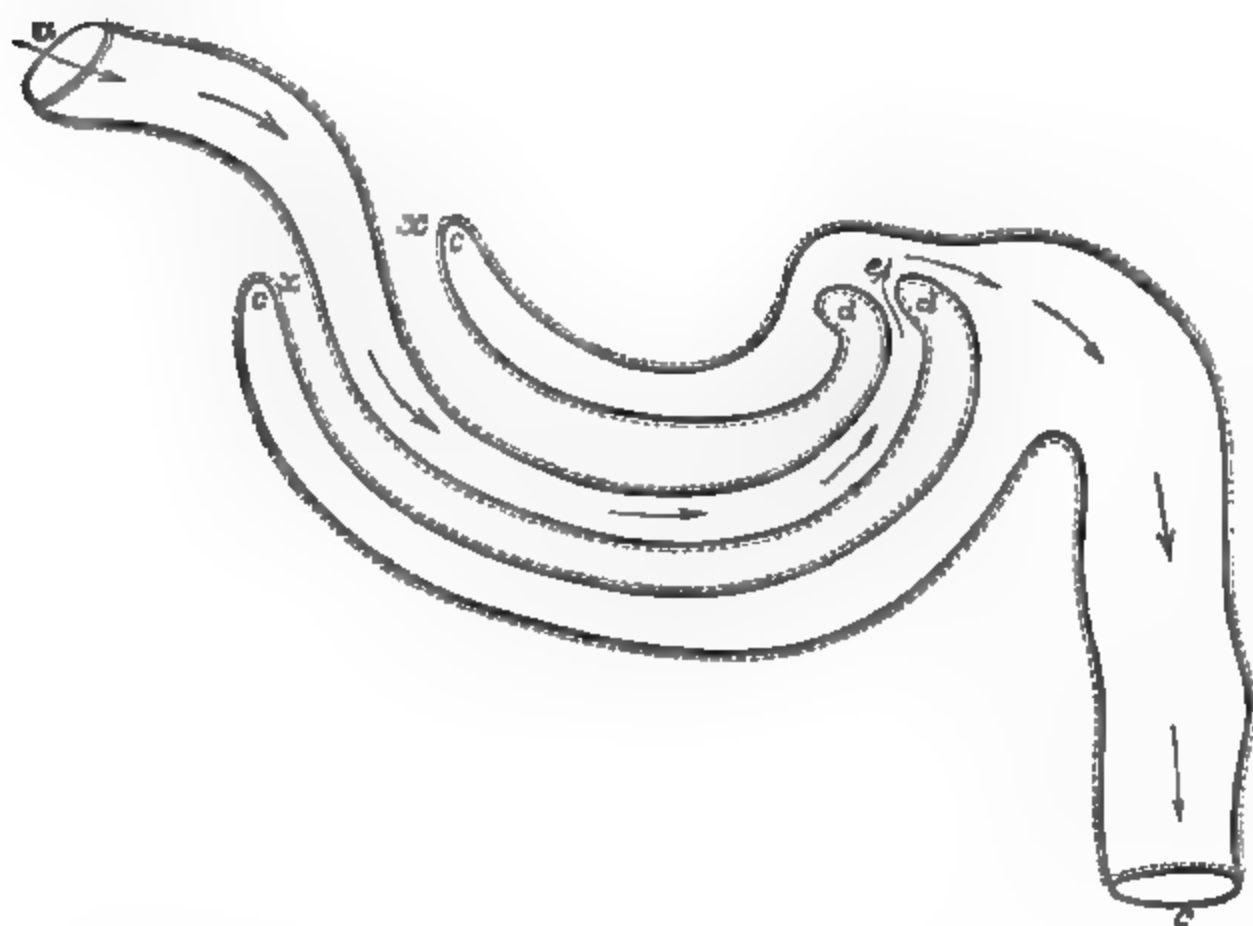
A case recorded by Dr. Todd, in the *Army Reports for 1864*, p. 532, is of great interest, inasmuch as the dissection of the parts, made by me at Netley, shows that the intussusception was associated with a large polypus growing from the mucous surface of the small intestine. The history of the case further shows that it was preceded and accompanied by intense and severe diarrhœa; and in the course of examination of the parts sent to Netley, my coadjutor, Dr. Davidson, discovered that the mucous membrane of the *caput cæcum* was infested by the minute parasite known as the *trichocephalus dispar*. This parasite is a very minute round worm, with its head-end of hair-like fineness, usually firmly fixed to the mucous membrane of the intestines, while the rest of the body is generally coiled upon itself and hidden amongst the mucous secretion of the gut. The natural history of this entozoön shows that it has oftentimes been associated with severe epidemics of diarrhœa. Indeed, its discovery more than 100 years ago (1760–61) was made during the prevalence of a severe epidemic of diarrhœa (*morbus mucosus*) amongst the soldiers of the French army, associated with the presence of this parasite in the *caput cæcum* of those who died of the disease.

Five cases of intussusception are recorded in the *Transactions of the Pathological Society* which are associated with polypoid tumors of the intestine at or near the site of lesion. The latter case referred to in these *Transactions* terminated favorably after the passage of the invaginated portion of gut (containing the polypus), the symptoms having continued for three months.

The *volvulus*, or portion of the gut where the obstruction exists, consists—(1.) Of the external portion formed by that part of the bowel into which the other has slipped; (2.) Of the middle; and (3.) Of the internal part, composed of the reflection of the invaginated portions. As it is not always easy to follow the anatomical relations of the several layers of structures composing an intussusception, it may be of use to give a diagrammatic outline of the relation of the serous and mucous coats of the intestine in such

lesions; because, it is of practical importance to remember that, although the parts are greatly displaced, yet the anatomical relations of the serous and mucous surfaces of the intestine are never altered. Textures of the same anatomical character are always in contact one with another, and the channel of the gut along its mucous surface is always open. That such is the case may be understood by taking the leg of a long stocking from which the toe-end has been cut off, so that the stocking may be converted into a continuous tube open at both ends. If one portion of the stocking be then drawn into the other, a correct imitation of the relation of surfaces in an *intussusception* will be obtained.

FIG. 68.



In the diagram (Fig. 68) the tube, *a b*, may be traced to be continuous, as indicated by the arrows. The dotted line is meant to correspond to the *serous* surface, and the thick dark line to represent the *mucous* surface of an intestine comprehending an *intussusception*. From the outer to the innermost surface at the site of lesion, on cutting through one layer, the *first* of the inclosing gut, a **MUCOUS SURFACE** is reached, which has a *cul de sac* reflection at *c*. Thus, the **MUCOUS** surfaces of the including and the included portions of intestine are in constant apposition, rubbing against each other. On cutting through the *second* layer of the *intussusception*, a **SEROUS SURFACE** is reached which has a *cul de sac* reflection at *d*. Thus, the **SEROUS** surfaces of the including and the included portions of intestine are in constant apposition, rubbing against each other. At *e* the mucous canal is always more or less open in cases of simple *intussusception*; but the orifice is invariably turned or curved to

one side, and may be so firmly applied against the mucous surface of the including intestine (by the dragging of the mesentery which has been included) that the orifice may be closed, like a valve, by simple apposition and compression. In consequence of the lateral attachment of the mesentery to a line along the serous surface of the intestine, a portion of mesentery equivalent to the extent of the serous surfaces in apposition is also dragged into the containing gut, and exercises a most important influence upon the nature of the lesions. In consequence of the one-sided attachment of the mesentery, and the dragging of its parts, the included gut necessarily takes the form of a curve. It thus appears highly corrugated over its mucous surface, dragged to one side and curved upon itself, as it lies exposed on cutting up the outermost layer of intestine. The orifice of the contained or invaginated intestine is thus turned upwards, and is not to be found at what appears to be the lowermost part of the extreme end of the included portion of bowel. This great dragging of the mesentery necessarily also obstructs the mesenteric vessels, and leads to the gradual effusion of blood between its layers. This effusion is seen after death in the form of compact indurated masses of a dark color. Blood is also gradually effused from the mucous surface of the gut, which becomes gradually strangulated; and, combined with other symptoms, this persistent effusion of blood is almost pathognomonic of incarceration of a bowel in the form of intussusception.

In some cases the pressure of the inclosed bowel on the containing gut, and the dragging of the included mesentery, are so intense, that the invaginated portion has actually effected an opening by ulceration through the inclosing bowel, and so projected into the cavity of the peritoneum, before death took place. In such cases the fatal result was generally by peritonitis. Four specimens in the Museum of the Army Medical Department illustrate this fact in the pathology of intussusception. One has no history; another is quoted as an example of the bad effects of purgation; the third is from a woman aged twenty-four, who had been ill ten days, when an extensive opening occurred through the inclosing bowel, and peritonitis of a severe form speedily proved fatal. A fourth occurred in a soldier aged twenty-two. In this specimen a very large mass of gut is involved, and it is very significant of the injurious influence of purgation in such cases. Although this soldier is recorded to have had persistent diarrhoea, flatulence, bloody stools, and other symptoms of intussusception, he was nevertheless alleged to be a malingerer. He was treated with purgatives, and lived long enough for the end of the included gut to wear a hole, by pressure and rubbing, through the substance of the containing bowel. No one can doubt but that such a lesion would be greatly aggravated by purgative remedies.

In all cases of intussusception, whether occurring in children or in adults, the administration of purgative medicines tends to aggravate the lesion and the symptoms. Accordingly the rule of practice is absolute—namely, “to withhold all purgative medicines from the commencement in cases of intussusception.” The bowel



being incarcerated, the stimulus of purgation proceeding from above downwards is quite unable to undo the incarceration of an intussusception. A purgative, therefore, acts injuriously as a stimulus which cannot be obeyed; and the obvious tendency of a purgative is to increase the peristaltic action of the bowels, and therefore to increase still more the invagination. That such is the case will be readily understood by a physiological consideration of the phenomena of intussusception; for although it is not always easy to account for the first beginning of an invagination, yet physiology enables us to understand how, an invagination once begun, the lesion tends to increase—(1.) From the peristaltic action of the bowel, greatly stimulated and increased by irritation of every kind, so long as tonic irritability continues; (2.) From the spasmodic action of the part of the gut *above* the invagination preventing spontaneous return; (3.) From the invagination being thus completed, it continues permanent, tenesmus occurs, and thus the violent and repeated contractions of the abdominal muscles tend still more to maintain and increase the lesion. The constant motion and pressure of parts one upon another in some cases is so great, that the end of the invagination has been known to penetrate through the walls of the inclosing bowel, so as to appear in the cavity of the peritoneum.

The injurious tendency of purgative medicines will be also still more apparent if a lesson is taken from the teachings of morbid anatomy. Post-mortem examination, combined with a study of the phenomena of intussusception during life, shows that the increase of the lesion takes place mainly at the expense of the *external containing* portion of the bowel; and therefore, also, it can readily be understood how some fixed point in the bowel is the first starting-point of an invagination. Most frequently (56 per cent., BRINTON) it is the ileum and cæcum which pass into the colon, then the colon passes into itself, so that the *appendix vermiformis cæci* becomes included. Two orifices then exist at the extreme end of the invagination: one is that of the lesser bowel, the ileo-cæcal valve, the other is the entrance into the *appendix cæci*. Two preparations in the Museum of the Army Medical Department show this arrangement of the parts composing the lesion in cases of invagination. In 32 per cent. (BRINTON) the small intestine forms all the layers. In 12 per cent. (BRINTON) the colon is exclusively involved; and the rectum scarcely ever forms more than the outer layer.

**Causes.**—In all the dissections of invagination whose history I have examined, they have either been associated with the diarrhoea of irritation (as from worms, undigested masses of food); or with cerebral lesions (as in the cases of children in whom invaginations are very common); or with ulcers of the intestines, or polypoid growths.

In giving a summary of cases, Dr. Peacock finds that, while in some cases no cause could be assigned for the affection, in others the disease appeared to have been excited by accidents, taking injudiciously large meals or improper food, by the irritation of drastic

purgatives, or the presence of worms. Of the cases which he analyzed, "in one instance the disease followed a kick, in another the carrying of a heavy weight, and in a third the taking of a large meal; in one an active purgative had been taken a few days previously, and in one there were worms in the bowels." In the case he particularly records to the Society, "the predisposing cause might possibly be the small polypus which was found attached to the mucous membrane, near what appeared to be the upper end of the invaginated portion" (*Trans. Path. Soc.*, vol. xv, p. 117).

As to how the lesion first commences some notion may be obtained, and the physiology of the process may, to some extent, be comprehended by experiments on the intestines of animals while under chloroform, or just after having been killed. If a portion of the small intestine be pinched with a pair of forceps, active circular contraction and constriction of the gut immediately commences at the site of irritation. This constriction continues for some time, and is transmitted, or advances onwards, under the influence of the usual peristaltic action of the intestines. Wave upon wave of constrictions may be made in this way to follow each other in succession, so long as the vital irritability of the intestine continues. If the advance of the constriction onwards is impeded by any cause, such for instance as an undigested mass of food, a scybalous portion of fæces, a foreign body, or a polypoid growth, or even another constriction, and if the onward motion of the bowel fails to dislodge the obstruction, a partial invagination very readily occurs; but where the obstruction is necessarily localized (as from ulcers, polypoid growths, or fixed parasites) permanent invagination commencing in the vicinity of such local lesions is more readily induced. The mere weight of a polypoid growth would necessarily favor the occurrence of invagination by dragging down the bowel to which it is attached, and so inverting its coats. In 5 per cent. (BRINTON) the intussusception is so caused; and often situated above the ileo-colic valve, the polypus having made its way through it, the spasmodic contraction of the valve would effectually prevent the spontaneous return of the invagination.

Another efficient and increasing cause of obstruction exists in the included mesentery. By its inclusion it causes such a drag upon the included portion, that the mucous surface of the bowel is not only greatly corrugated, but by apposition against the containing bowel the narrow canal through the invagination becomes completely closed. The anatomy of the parts at once shows how this is effected. The mesentery being attached only to one side of the gut, it drags the intestine to that side; and the greater the amount of bowel invaginated, the greater will be the curvature of the included portion upon the mesentery as an axis; and the greater and earlier will be the complete obstruction.

Thus, when a case of intussusception is examined after death, the invaginated portion of bowel is always seen in the form of a curve lying within the including intestine. The extreme end of the invaginated portion thus comes to be so turned upon itself that the canal by apposition is completely shut up. The bloodvessels

of the impacted mesentery also undergo great and increasing congestion; and as strangulation and obstruction become complete, indurated masses of blood may be found effused within the folds of the mesentery. As strangulation increases, blood exudes from the mucous surface of the intestine, so that small flocculi of blood, as well as fluid blood mixed with mucus and free from fecal matter, continue to pass *per rectum* as long as the canal remains open, and such symptoms (hemorrhoids excluded) are justly regarded as pathognomonic of incarceration of the bowel from a simple intussusception not yet completely closed.

**Symptoms of Intussusception.**—The symptoms indicate obstruction and inflammation. *In the child* they mainly consist of restlessness, sudden fits of crying, and straining as if at stool; a discharge of mucus, more or less mixed with blood and free from fecal matter, sickness, and anxiety of countenance. These phenomena are generally, but not invariably, preceded by a sudden and violent action of the bowels. A physical examination of the belly may disclose a tumor or swelling of the intestine; and sometimes, as in a case described by my friend and neighbor, Dr. Orsborne, of Bitterne, to the Medical Society of Southampton, the invaginated part could be reached with the finger introduced into the rectum.

*In the adult* the symptoms and phenomena in cases of complete strangulation of the gut by strictures, like internal strangulation from bands of lymph, or twisting of the gut round such constrictions, are sudden; and if the stricture be not relieved, the case proves fatal about the fifth or sixth day. On the other hand, in cases of intussusception the symptoms of the incarceration are by no means sudden nor rapid in their progress; or, rather, they are comparatively slower in their development and progress to a fatal issue than cases of complete obstruction by strangulation are.

In cases of intussusception the impediment from the first is partial, and in some cases the obstruction is never complete, although the symptoms may extend over many days, or weeks, or even months. One case is on record in the *Transactions of the Pathological Society* in which the symptoms of incarceration were present during four months, and although adhesions had formed between the serous coats of the invagination, yet obstruction of the intestinal canal was never complete.

A summary of the prominent phenomena of intussusception may be stated as follows, showing the comparative slowness and incompleteness of the obstruction, as well as bringing forward those phenomena which (when weighed with other symptoms) may be regarded as pathognomonic of intussusception. Diarrhœa early is generally the first indication of illness. In connection with this diarrhœa and its prevalence, the existence of parasites must not be overlooked, inasmuch as their existence in one patient renders it probable that they may exist also in others, as a cause of the prevalence of diarrhœa in an epidemic form. Collapse, nausea, and diarrhœa, are the next symptoms to supervene, followed by tenesmus, or a feeling of fulness in the rectum; and after going two or three times to stool, *blood only* is observed to pass, and similar discharges

continue. An enormous discharge of *fæces* may sometimes take place after an enema, but generally accompanied by a *large flow of blood*. So long as the patient lives *he continues to pass blood at stool*, and on occasions *scybala*. Small portions of hardened *fæces* may continue to be got rid of, but every effort is attended by *the usual flow of blood*. Purgative remedies aggravate the symptoms. Death is comparatively slow; and hence the extensive coagula which are found in the cavities of the heart.

**Diagnosis** is therefore mainly differential between this and other forms of *alvine obstruction*. The *colic* is extremely severe, and the marked remissions of pain are followed by exacerbations, which increase in violence with each repetition. Physical diagnosis ought to be had recourse to in every case. From *hernia*, cases of *intussusception* are to be distinguished by the absence of the *hernial tumor* at the respective abdominal apertures where *hernia* is usual; but a tumor within the abdomen is a physical sign of the greatest value; and is not often absent, though easily overlooked, as it is often of small size (BRINTON). The condition of the rectum ought always to be ascertained by a digital examination. The vomiting is less urgent in proportion as the obstruction is lower down in the bowel; if the duodenum is involved, vomiting is almost incessant. Dr. Orsborne, of Bitterne, lately brought me the stomach and intestines of a child that died of this affection; and the relations of the parts involved were peculiar in this respect, that in the reflection of the colon, which contained the *volvulus*, the mesentery of the duodenum was dragged in, and along with it the adjacent wall of that intestine was also taken in. The parts are preserved in the museum at Netley.

It is necessary to distinguish between *ileo-cæcal*, or the *colic invagination*, and that of the small intestine. The former is distinguished from the latter by:

“(1.) The prominence of *tenesmus*, which is rarely present in any marked degree, where the small intestine only is implicated; (2.) The greater size and fixation, as well as the different site of the tumor, which, if large, generally proceeds towards the left side of the *hypo-gastric* or left *iliac* region; (3.) The subordinate share taken by *hemorrhage*, which, instead of copious bleeding by stool and vomit, is often little more than a scanty admixture, scarcely sufficient to tinge the mucus passed from the bowels, with violent and frequent straining by the patient; (4.) The still more subordinate share taken by obstruction, which not only seems to be often anticipated by death, as regards any complete symptoms of its presence, but to be really absent, owing to the *patulous* state of the axis of the invagination; (5.) The presence of the end of the invagination in the rectum” (BRINTON).

Obstruction of the small intestine is chiefly characterized by (1.) The umbilical seat of the pain, which is also more early and severe; (2.) Vomiting is more early, severe, and frequent; obstruction is much more rapid, constant, and complete in the small than in the large intestine.

**Prognosis** is not always hopeless, but is nevertheless most grave.

The cases which recover are almost invariably chronic or protracted ones. In the fifteenth volume of the *Transactions of the Pathological Society of London*, Dr. Peacock gives an account of the passage of a large piece of bowel by the rectum in a case of invagination, followed by recovery; and in that paper he refers to eighty-eight cases of intussusception in which portions of bowel are reported to have passed by the rectum. Those which end by expulsion of the *volvulus* have a duration of from twice to thrice as long as that of the fatal cases; and it is not till the second, third, or even sixth week that the remission of symptoms occurs which announces the relief of the obstruction, and which often precedes by a day or two the first healthy alvine evacuations.

It is to be remembered that distension of the involved bowel measures not only the danger, but the rapidity of the fatal issue.

The cases of most serious import are those which, commencing in the small intestine, involve the ileo-colic valve; and the majority of fatal cases are those in which the cæcum and ascending colon have swallowed up, as it were, a large portion of the small intestine. The danger may not be at first appreciated, because the obstruction is never complete in the first instance, as we know from the nature of the lesion as well as from the accurate history of cases. The bowel, at first, is merely incarcerated by the invagination, and it is not until the middle and internal portions, with their contained mesentery, become compressed, constricted, and ultimately strangulated, that complete obstruction ensues. Till this occurs, the continuous expulsion of bloody mucus from the central tube of the inversion goes on. Inflammation and sloughing thus commence as the parts become subject to more and more increasing pressure.

Two forms of inflammation prevail—namely, one, serous, between the opposed peritoneal surfaces, and commencing at the angle of reflection of the middle on the external layer (*x, x, x*, in the diagram, p. 863), at the part where the one portion slips into the other. It is here that the peritoneal surfaces of the invagination commence to adhere; and up to the period of adhesion the ancient remedy inculcated by Hippocrates, of injecting air into the great gut by a long tube, introduced *per rectum*, has effected the greatest number of cures—forcing, by gentle, persistent, and equal pressure, the invagination backwards, and so causing it to be undone. The other form of inflammation takes place between the two opposed mucous surfaces (*c, c*, in the diagram). At this angle of reflection an abundant white leucorrhœal-like secretion (corpuscular elements of inflammation with mucin) commences very early to be discharged by the rubbing of the opposed surfaces, and eventually the inflammation (mucous and serous) may be so destructive that ulceration and sloughing of the whole invagination may be the consequence. In this way continuity of the canal may be restored—the invagination having passed, *per rectum*, as a slough. Dr. Brinton believes this favorable termination occurs in not less than one in every two or three cases of intussusception. On an average, separation of the slough is not complete before the eighth day, and the liberated bowel is rarely expelled *per anum* before the tenth or twelfth day,



and is sometimes only prevented by the death of the patient on the fifth or sixth day. Of such cases eighty-eight are now on record, an analysis of which, by Dr. Peacock, has been already referred to (*Path. Soc. Trans.*, vol. xv, p. 114).

**Treatment.**—The following are the details of treatment for cases of intussusception, advocated by the late Dr. Brinton. The chief indications and treatment are—(1.) *To prevent distension*, by reducing in every possible way, the quantity of food and drink, restricting the latter to small but frequent sips (preferably through a long straw or tube) of cool, iced liquids. Food is to be given, as strong beef-tea, soup, or milk, with equal frequency and caution. Small doses of alcohol (as brandy with water, or soda-water) is to be alternated. But if any repugnance exist to food or drink, or if vomiting is excited by these articles, the amount given must be reduced. Water, milk, and gruel are to be given freely in often-repeated enemata.

(2.) *To assuage pain and to mitigate excessive peristalsis* suggest the same kind of remedy—namely, *opium*, which is to be given continuously and alone, in the solid form—preferably in the *extract*. The practical limit of the dose is indicated by the comparative arrest of pain, the approach of narcotism, and decided contraction of the pupil.

*Belladonna* is only of use as a remedy to diminish the straining peristalsis. It may be given *combined with opium*, as two parts of *extract of opium* to one, two-thirds, or even one-half part of *extract of belladonna* in a pill.

*Enemata* are useful as a mechanical aid to removing obstruction. They may gradually distend the bowel at the site of obstruction, so as to effect such a change in its position and arrangement as may release the impacted portion. The administration of enemata is only safe and efficient if undertaken by a person of competent skill. The quantity of fluid must be injected little by little, and must be retained as long as possible; and the patient must resolve to tolerate some pain in reaching that climax of distension at which only enemata are calculated to relieve obstruction.

In cases where the intussusception is in the large intestine, inflation of the bowel with air, as originally suggested by Hippocrates two thousand years ago, in his third book (*Περί Πληθύνων*), has of late years been revived and adopted, first in America, and subsequently in this country (Gorham, in *Guy's Hosp. Reports*, and *Med.-Chir. Trans.*, vol. ix). This Hippocratic remedy has undoubtedly been more successful than any other. Of twenty-eight cases, the details of which were collected by Dr. Orsborne, there were only seven recoveries; and three of these were effected by inflation of the colon with air. Dr. Murphy and Mr. Erichsen have borne strong testimony to its value in discussions on the subject; and the former had recourse to the operation on one of his own children with success (MS. notes of Dr. Orsborne). For its success, Dr. Orsborne is of opinion that the remedy should be employed at an early period, before there has been time for adhesion between the contiguous surface of the volvulus. My friend Dr. David Greig, of Dundee, has

had recourse to this method of treatment in numerous instances with perfect success, and has published an account of his experience in the *Edin. Monthly Med. Journal* for October, 1864. By means of the ordinary elastic enema tube, fitted to the pipe of a small pair of bellows, he was able to pass a considerable quantity of air into the rectum, continuing the process till the belly shows signs of considerable distension, and even till uneasiness prevails. At the same time warm fomentations are to be applied to the belly. Its beneficial action is indicated by the relief of the urgent symptoms, such as straining and vomiting; and gradually a fecal evacuation is obtained from the bowels. The use of large enemata, with manipulation, has also been recommended. A long stomach-tube is to be passed as high up the colon as it will go, and the anus being firmly compressed round it, warm water is to be slowly injected, so as to distend the bowel as much as possible. When the fluid is allowed to come away, the abdomen should be pressed upon with the hands, so as to move about the coils of intestine (TANNER).

The earlier any of these remedies are had recourse to, the greater will be the chance of recovery; and the patient may be put under the influence of chloroform to facilitate the manipulations. When all remedies fail, gastrotomy may be thought of, and its chances of success considered. The operation is advocated by Benjamin Phillips, and in some cases it may be justifiable (see his paper in *Med.-Chir. Trans.*, vol. xxxi). The only cases for which it seems suitable are in obstructions from bands, diverticula, and the like lesions affecting the small intestines. The object of the operation is to divide the cord-like cause of strangulation (BRINTON).

### SECTION III.—RELATIVE WEIGHT OF THE SOLID VISCERA OF THE ABDOMEN.

The liver, the kidneys, the spleen, and the pancreas are the organs which require special notice here; and in order to obtain positive data for the solution of many questions regarding their pathology, it is necessary to know the relative weight of these viscera.

According to the experience of Frerichs, the relative weight of the liver in healthy individuals may vary from  $\frac{1}{17}$ th to  $\frac{1}{8}$ th of the body; and in adults it fluctuates between  $\frac{1}{24}$ th and  $\frac{1}{10}$ th. During the period of greatest growth of the body, the liver does not become enlarged in a manner proportionate to the increase of the entire body; and its diminution in old age is for the most part in advance of that of the body. Its substance in this respect, therefore, presents a marked contrast to the muscular tissue of the heart; for whilst the heart increases progressively up to an advanced age (Bizot, Boyd), the mass of the liver diminishes (FRERICHS, BOYD); and in old age, as a general rule, there is senile atrophy of the organ.

TABLE SHOWING THE RELATIVE AVERAGES OF BODY-WEIGHT, AND THE WEIGHT OF THE SOLID VISCERA OF THE ABDOMEN, AS TO AGE AND HEIGHT (Boyd)

Age and Sex.		Body-weight.	Body-height.	Weight of Liver.	Weight of Spleen.	Weight of Pancreas.	Weight of Kidneys.
Years.		Lbs. Oz.	Inches.	Ounces.	Ounces.	Ounces.	Ounces.
1 to 2	Male, . .	14 6	28.5	11.7	1.34	.44	2.65
	Female, . .	13 2	27.7	11.17	1.04	.49	2.4
2 to 4	Male, . .	20 0	31.6	16.85	1.58	.76	3.33
	Female, . .	18 7.5	31.6	13.49	1.28	.68	3.14
4 to 7	Male, . .	25 8	37.5	19.13	1.85	.8	4.05
	Female, . .	24 9	37.0	19 56	1.65	.8	4 26
7 to 14	Male, . .	42 6	47.0	34.71	3.03	1.68	6.58
	Female, . .	38 6	45.0	25.86	2.54	1.34	5.75
14 to 20	Male, . .	68 0	60.5	57.76	5.19	2.19	9 34
	Female, . .	63 14	57.7	54.33	4.68	2.64	9.69
20 to 30	Male, . .	92 14.5	66.7	60.29	7.19	3.54	11.57
	Female, . .	86 13	62.0	52.74	6.53	2.95	10.17
30 to 40	Male, . .	98 3.5	66.5	58.11	7.12	3.47	11.35
	Female, . .	87 0	62.0	53.61	6.13	3.05	10.34
40 to 50	Male, . .	102 0	66.8	58.06	6.19	3.48	10.89
	Female, . .	84 9.5	62.0	49.03	5.04	2.73	8.8
50 to 60	Male, . .	102 0.5	66.0	55.37	6.23	3.46	9.1
	Female, . .	86 0	62.0	44.0	4.67	2.83	8.55
60 to 70	Male, . .	103 13	65.7	48.24	4.82	3.1	8 83
	Female, . .	86 14	61.5	42.98	4.07	2.8	8.28
70 to 80	Male, . .	106 13	65.7	46.33	4.93	3.22	10.68
	Female, . .	80 4	61.0	38.37	3.57	2.62	7.63
80 to 90	Male, . .	99 0	66.7	41.01	4.27	2.83	8.25
	Female, . .	79 0	60.0	34.64	3.46	2.37	6.86

SECTION IV.—DISEASES OF THE SOLID VISCERA OF THE ABDOMEN.

DISEASES CONNECTED WITH THE LIVER AND ITS FUNCTIONS.

**Definition.**—Under this heading (as with the previously considered diseases of the abdominal viscera connected with the process of digestion) it is proposed to consider some of the more definite morbid states expressed by functional disorders, lesions of texture, or of both.

**Pathology and Morbid Anatomy.**—The morbid states associated with hepatic disorder may be referred—(1.) To congestion of the liver in various forms; (2.) To *hepatitis*, or inflammation of its substance, and the formation of (3.) *Hepatic abscess* (*pyæmic*, *dysenteric*, or the effect of injury); (4.) *Chronic enlargement and induration*;

(5.) To *increased secretion and elimination of bile*; (6.) *Jaundice*; (7.) *Acute atrophy*; (8.) *Thickening of the capsule*; (9.) *Cirrhosis*; (10.) *Fatty liver*; (11.) *Amyloid disease of the liver, waxy or lardaceous liver*; (12) *Fibroid deposit*; (13.) *Syphilitic deposit*; (14.) *Cancer*; (15.) *Colloid*; (16) *Non-malignant tumor*; (17.) *Cysts*; (18.) *Tubercle*; (19.) *Hydatid*; (20.) *Obstruction of vena porta*.

**I. Congestion of the Liver.**—This morbid state is expressed in various forms: (1.) There may be *congestion from increased secretion of bile*, and the bile-ducts especially are then gorged with bile; or there may be (2.) *Passive congestion* of the hepatic veins, or of the portal veins; and (3.) *Active congestion*, chiefly involving the arterial capillaries.

Morbid anatomy assigns to each of these forms of congestion peculiar and characteristic appearances.

The liver, after death, is found enlarged principally upwards, so as to encroach upon the capacity of the right side of the chest. The ducts are generally distended with bile, and this may arise from various causes, such as compression of the ducts from over-distended veins, frequently brought about by intermittent and remittent fevers. It is also a condition associated with the early stages of *cirrhosis*, when the appearance of the liver is that known by the name of *nutmeg liver*. The distension of the veins seems to be of a passive kind, and to depend on recession of blood from the surface of the body. An ultimate result of hepatic biliary congestion seems to lead to the filling of the hepatic cells with dark yellow matter, and when forms of passive vascular congestion are long associated together, ultimate atrophy and degeneration of the hepatic cell ensue.

The central part of an hepatic lobule is, in health, distinguished by a red spot, centrally situated, and measuring about half the diameter of the lobule. When this dimension increases it is characteristic of *intralobular congestion*—a morbid state in which the hepatic veins are especially engaged.

In *intralobular congestion*, where the *portal* veins are specially congested, the centres of the lobules are comparatively pale, while the lobules are surrounded by red vessels.

The condition known as the *nutmeg liver* is explained by the various forms and results of congestion, as well as by other morbid changes of texture. It is so named from the resemblance, in combination and arrangement of colors, in a section of the liver, to that seen in the section of a *nutmeg*. In its most marked forms a deep red congestion forms patches and streaks, which occupy the central parts of the lobules, and are *partially* surrounded by patches of a grayish or dirty white color, and which are devoid of blood. The light-colored parts may be caused either by fatty degeneration of the cells, which, enlarging, compress the capillaries and expel or prevent their being filled with blood; or it may be due to an increase from deposit and condensation of the Glissonian capsule in the early stage of that morbid condition known as *cirrhosis*. It also is a morbid state in which the capillaries are emptied by com-

pression and contraction of tissue, or in which they have undergone atrophy.

By far the most frequent cause of hepatic congestion is valvular disease of the heart; and especially obstruction to the circulation through the right side, such as results from the causes of *apnœa*. These congestions, so far as the liver is concerned, are of a passive kind; and their most marked and constant effect is to produce *ascites*, with or without *anasarca*. If such congestions continue long, they lead to bilious contamination of the blood.

*Congestions of the liver* are chiefly brought about by sudden chills, the cold stages of fevers, over-abundant feeding, intemperance in alcoholic or malted liquors: products of faulty digestion are at once carried to the liver, and influence its circulation, and so also does excessive bodily exercise in the heat of the sun. The liver bulges, and may be accompanied by uneasiness on exploration, or of weight on getting into the erect posture. The countenance and complexion may be pale, sallow, or dusky livid; and the tongue will be found coated, the bowels constipated, the appetite defective, and there may be nausea, vomiting, and headache. The pulse is slow, compressed, and irregular, or it may be quick and feeble; and generally speaking, the symptoms are obscure. Dr. Budd, in his exhaustive work *On Diseases of the Liver*, thus notices how congestion is so common,—“Amid the continual excesses at table of persons in the middle and upper classes of society, an immense variety of noxious matters find their way into the portal blood that should never be present in it, and the mischief which this is calculated to produce is enhanced by indolent or sedentary habits. The consequence often is, that the liver becomes habitually gorged. The same, or even worse effects, result in the lower classes of our larger towns, from their inordinate consumption of gin and porter” (*op. cit.*, p. 66).

**Treatment.**—Congestions of the liver are mainly relieved by a restricted and judicious diet—abstinence from all rich dishes and fermented liquors. Active exercise in the open air must be taken till fatigue is produced sufficient to induce a desire to rest. Horse exercise is especially beneficial.

With regard to medicine, Plummer's pill at bedtime, followed by a Scidlitz powder in the morning, or by the usual black draught, or by such saline purgatives as *sulphate of magnesia* and *bitartrate of potash*, which cause a drain from the portal system of veins. An electuary composed of equal parts of *sublimed sulphur* and the powdered gum resin of *guaiacum*, will be found a most useful remedy in cases where the congestion of the liver is associated with hemorrhoids, which is extremely common. When dropsy prevails, doses of the diuretic pill, of *hydrargyrum*, *squills*, and *digitalis* (a grain and a half of each ingredient), taken night and morning, are attended with benefit.

**II. Inflammation of the Liver, or Hepatitis**, occurs in various forms, and results in several consequences. In all countries in the temperate zone, at least two forms of inflammation of the liver occur. One of these terminates in simple or granular induration, and has a clinical history of its own; the other terminates in softening and



acute atrophy of the gland, associated with jaundice and febrile phenomena of a malignant typhoid type (FRERICHS). In tropical climates a third form of inflammation of the liver has a tendency to result in suppuration; and it is described under the name of *suppurative hepatitis* (ANNESLEY, CAMBAY, HASPEL, BUDD, MOREHEAD, MACLEAN).

The inflammation may have its seat in the fibrous envelope of the gland, or in the sheath of the vessels in Glisson's capsule; or it may attack the glandular parenchyma, the portal or hepatic veins, or the bile-ducts.

Inflammation of the glandular or hepatic parenchyma is either circumscribed, leading to abscess or to granular contraction, or it is diffusely extended over the entire organ; and then, according as the process involves all the anatomical elements of the gland, or is limited to the areolar matrix between the lobules, it gives rise sometimes to softening and acute atrophy, or at other times to induration or cirrhotic degeneration.

III. **Inflammation of the Capsules of the Liver** (*Perihepatitis*) and of Glisson's capsule is rarely accompanied by serious derangements, unless the inflammation extends to the portal or hepatic veins, or causes obstruction of the larger bile-ducts—events of rare occurrence (FRERICHS). Peritonitis, disease of the liver itself, or inflammation of neighboring structures, such as pleurisy, are the usual causes of *perihepatitis*. The chief symptoms are, tenderness of the hepatic region on pressure, motion, or deep inspiration, without any change in the volume or situation of the organ. Jaundice, as a rule, is absent; so also are febrile phenomena.

IV. **Inflammation of the Granular or Hepatic Parenchyma** occurs either as a circumscribed process limited to isolated patches, or it is diffuse, extending over the entire organ in a more or less uniform manner. It is the former variety which leads, in most cases, to suppuration and the formation of abscesses. The diffuse form, on the other hand, at one time induces rapid destruction of the glandular elements, with softening and atrophy of the organ, and at another time it induces induration and cirrhotic degeneration.

In the primary stage of the *acute form of diffuse inflammation of the liver* there occur patches of hyperæmia, while the peripheral portions of the lobules are infiltrated with gray matter, and a juice or fluid serum, rich in albumen, flows from the cut surface of the gland, and the capsule is rough and opaque. Destruction of the glandular cell results in all the cases; but whether atrophy of the gland occurs or not depends upon the stage at which the disease becomes fatal, and partly upon the condition of the liver at the commencement of the process of infiltration. The disease is usually accompanied by a similar condition of the kidneys and spleen, when large quantities of albumen pass off in the urine, and the spleen exists in a state of acute tumefaction.

Violent mental emotions, the condition of pregnancy, typhus, pyæmia, scarlet fever, tropical malarious fevers, are the circumstances under which this form of inflammation is most apt to occur; and the destruction of the liver cell-tissue which ensues is indi-

cated during life by the symptoms of atrophy, and by those peculiar changes in the urine which, at the height of the disease, are almost pathognomonic. In the cases described by Frerichs the urine is described of a brown color, and smelling of sulphuretted hydrogen, having a feebly acid reaction, and containing a small quantity of bile-pigment, but no albumen.

The most important post-mortem changes to be seen in livers are, the occurrence of softened portions, having an irregular form, of a pale yellow or reddish-brown color. All traces of the outline of the lobules are obliterated, while they are distinct in the firmer portions. The secreting cells of these parts are everywhere destroyed, and their place supplied by numerous oil-globules, granules, and particles of coloring matter. Sometimes the softening follows the ramifications of the portal vein, and the hepatic cells in the softened parts are destroyed, being converted into a granular debris, oil-globules, and pigment-molecules. In the former portions the entire cells are loaded with fine granules. The gall-bladder, when treated with chloroform, fails to yield pigment. Tyrosine crystals are formed on the cut surface of such livers after some hours' exposure to dry air; and chemical analysis of the parenchyma yields large quantities of leucine and tyrosine. The symptoms of this form of inflammation are included in the account of the disease now known as *acute or yellow atrophy of the liver*, which will be given subsequently.

V. **The Chronic Form of Diffuse Inflammation** expresses itself by simple or granular induration of the substance of the liver, sometimes called *cirrhosis*, *interstitial hepatitis*, *hob-nailed* or *gin-drinker's liver*. An accurate knowledge of the nature of this induration was first obtained through the investigations of Kiernan, Hallmann, and Carswell. They showed that an increase of the interlobular connecting tissue of the hepatic parenchyma was the essential feature of cirrhotic degeneration. Rokitansky distinguishes two different modes of origin of the granular induration—the one proceeding from a morbid development of the capillary bloodvessels, owing to an excessive secretion of bile; the other due to a chronic inflammation of the hepatic parenchyma, tending to impermeability or obliteration of the finest ramifications of the portal vein. As a rule, the disease only comes under observation when it is more or less completely developed, and when consecutive disorders always associated with it, draw attention to the state of the liver.

The commencement of cirrhotic degeneration is marked by increased consistence of the liver. The increased consistence is due to increased and increasing hypertrophy of the areolar framework. The nature of the cell-contents also affects the consistence of the organ. The gland is softer when the cells are loaded with fat, and firmer when they are transformed into *amyloid* (*albuminoid*) material. The amount of blood and of serous infiltration affects the consistence; but the induration of cirrhosis and tenacity of the liver are mainly due to the increased development of the areolar framework.

In the slighter forms the liver may be enlarged or of normal

size, rather smaller than natural; its surface covered by an opaque and thickened capsule, and exhibiting flattened projections, varying in size from a pin's head to a pea. Similar nodules are observed in section, separated by narrow strips of gray areolar tissue more or less vascular. Sometimes the color is dark from bile-pigment; sometimes pale from fat.

In the advanced form the liver is reduced in size, particularly the left lobe, which not unfrequently is shrivelled up into a small membranous-like appendage, with a soft flabby rim of connective-tissue at the margins of the organ. Semi-globular knobs, more or less prominent, sometimes of a uniform, at other times of an unequal size and form, are thickly scattered over the surface.

In the lardaceous or amyloid infiltration combined with cirrhosis the liver exceeds its normal size.

The serous capsule is always thickened, and of a grayish-white color, especially in the depressions between the granulations. Numerous bands of connective-tissue pass from it to neighboring organs, such as the diaphragm, colon, stomach. On section, the organ has a cartilaginous hardness and coriaceous tenacity, showing at one place narrow, at another broad lines of connective-tissue surrounding the granulations, and sending streak-like processes into their interior. The granulations are generally dark or pale yellow. The elements of tissue are changed as follows:

(a.) *Condition of the Hepatic Cells.*—In such livers a large portion of the hepatic cells are destroyed, and their remains are found in the form of small masses of brownish pigment scattered through the filaments of the newly formed connective-tissue. Another portion of the hepatic cells constitutes the substance of the granulations, and may remain for a long period intact; but ultimately they become filled with fat and various sorts of pigment. In nearly one-half of the cases fatty degenerations occur. The connective-tissue, compressing the commencements of the bile-ducts, causes a retention of the secretion and a jaundiced condition of the liver; hence the deposit of pigment, which accumulates in the form of fine orange or sulphur-yellow granules, more rarely diffused through the cavity of the cells. Another tint may occur from decomposition of the red matter of the blood, especially where branches of the hepatic veins are obliterated.

(b.) *The Connective-Tissue.*—Its increased amount presents numerous varieties as regards its mode of distribution; and to it are due the differences in the size of the granulations.

The earliest increase is usually upon the finer subdivisions of the vessels in the interior of the liver, gradually involving the lobules. The bands of tissue sometimes surround single acini; at another time, three, five, or even seven *acini* may be inclosed. These bands increase at the expense of the *acini*, so that a little mass of brown pigment may be all that remains of the *acini*; and in this way large masses of connective-tissue take the place of gland-substance. The new connective-tissue varies as to elementary characters: at the circumference of the lobules it is fibrillated; in the substance of the lobules it is disposed to be amorphous; and in the thickened capsule it may be fibro-cartilaginous.

(c.) *The Vascular System* of the liver in cirrhosis undergoes great and important changes. The smaller divisions of the portal vein are in most cases narrowed by the shrivelling of the connective-tissue, or by partial impermeability of the finest ramifications of the portal vein from inflammation, obliteration, or compression. The vessels lose their round form, and become angular and bulging. Thrombi ought to be looked for in the branches when destruction of capillaries is obvious; and such destruction of the capillaries of the portal vein is in proportion to the disappearance of the glandular substance of the liver. So long as the hepatic cells exist, the peculiar mesh-like capillary network also exists; but where the cells disappear, their place is supplied by a connective-tissue, and entirely new capillary channels make their appearance, forming elongated meshes, communicating as well with the veins as with the hepatic artery (FRERICHS).

The trunk of the hepatic artery also becomes enlarged, and its capillary ramifications more extensive than in the healthy state, black pigment accumulating in its branches. A branched and tortuous network of vessels, of comparatively large calibre, appears in the connective-tissue, their peculiar and tortuous mode of distribution demonstrating their new formation.

The hepatic veins, as a rule, are unchanged; but their obliteration may take place by inflammation, from the capsule of the liver, being propagated to the walls of the vessels, when the capillaries of the hepatic vein are gradually destroyed, and their communication with the portal capillaries is interrupted.

(d.) *The Bile-Ducts*.—Their origin at the periphery of the lobule is destroyed by the pressure of the new connective-tissue; and there is apt to be catarrhal tumefaction of the mucous membrane of the larger branches.

These various changes may give rise to a long series of functional derangements, which in practice constitute the *symptoms of cirrhosis*. These are mainly—(1.) Derangement of the chylopoietic organs, from impediment to the passage of blood through the portal vein into the hepatic veins; and its stagnation in the radicles of the portal vein; (2.) Impairment, passing to complete suspension, of the functions of the liver.

**Causes.**—The chief cause of cirrhotic induration is the abuse of spirituous liquors. In other words, it is due to the specific action of alcohol as an irritant or stimulant poison. Other causes are syphilis and malarious fevers, especially frequent attacks of intermittent fever. The cirrhosis from syphilis is generally associated with the amyloid degeneration about to be described.

**Symptoms.**—Derangements of the stomach, a loaded tongue, nausea, and occasionally vomiting and faint jaundice, are the earliest symptoms in the clinical history of this disease, which usually comes on very insidiously; and although they may abate, the degenerative process in the liver advances, and gradually undermines the constitution. Digestion continues feeble, and distension and tenderness of the epigastrium, along with heartburn, flatulence, and constipation, are developed. The patients lose flesh and strength, and their

color becomes pale, or dirty yellow, while the skin is dry and rough. The abdomen becomes distended and fluctuates, but the liver is found reduced, and the spleen increased in size; and increasing ascites or tympanitis induces more or less dyspnœa. Hemorrhages from the stomach or intestines are apt to occur as the disease advances. Enlargement of the superficial veins of the belly may supervene, is characteristic of cirrhosis, and demonstrates that the current of blood in the portal vein is greatly impeded. Slight febrile excitement only manifests itself towards the close, and in most cases diarrhœa terminates life by exhaustion. In other cases the fatal end is by pneumonia, acute pulmonary œdema, or peritonitis. Occasionally death occurs under symptoms of acholia; the patients become jaundiced, purpura spots or ecchymoses are scattered over the skin; while delirium, convulsions, and deep coma close the scene.

**Diagnosis.**—"Slight sallowness of complexion," writes Dr. Budd, "a dull pain, or some degree of tenderness in the right hypochondrium, with occasional feverishness, in a person above the age of thirty, who has been long in the habit of drinking spirits to excess, are almost conclusive evidence of the existence of cirrhosis, even before there is any direct proof that the circulation through the liver is impeded" (*op. cit.*, p. 158).

**Prognosis** is always unfavorable; and the main question regarding

**Treatment** is the possibility of relieving the disorders of function which mainly threaten life, and so to delay the fatal termination. Complete abstinence from spirits is indispensable, and the diet should consist of mild, simple articles of nourishment, especially easily digested animal food. Coffee, spices, and articles irritant to the liver must be avoided. Swelling and tenderness indicate leeches and fomentations. Mild saline laxatives may be given; and when the tenderness ceases, the bowels must be kept open by *rhubarb* and *salines*. When nausea or vomiting occurs, *hydrocyanic acid*, *bella-donna*, *morphia*, or *extract of nux vomica*, are particularly suitable. When pain prevails, cupping or leeches are indicated over the liver. Saline purgatives, such as *sulphate of magnesia* or *bitartrate of potash*, should also be administered, while iced drinks and low diet must be the rule of life. After ascites is established, bleeding is apt to do much harm; mild diuretics are then indicated, and they are especially beneficial after the operation of "tapping."

**VI. Circumscribed Inflammation and Abscess of the Liver**, or, as it is sometimes called, *suppurative inflammation of the liver*, is always limited to one or to several isolated patches; and, with the exception of congestive turgidness of the contiguous texture, the remaining portions of gland-tissue are rarely implicated. The inflamed patches are usually found in a state of suppuration, which makes its appearance at an early period, gradually destroying the hepatic cells. Sometimes the abscess-cavity is inclosed by a cyst; at other times there is no defined boundary; but the inflammatory process extends till perforation occurs and the pus finds an outlet. The pus is rarely passed into the abdominal cavity; for adhesive inflammation of the capsule covering the abscess almost invariably occurs, so



that attachments form to the abdominal walls and adjoining organs. If the abscess bursts into the abdominal cavity, the result is fatal peritonitis. Frequently the abscess perforates the thoracic or abdominal wall superjacent to the liver, and opens directly outwards; or the pus may discharge itself into the pelvic, inguinal, or sacral regions, close to the spine. Sometimes the abscess tends in an upward direction, to penetrate the diaphragm, when it generally empties itself into the right pleural cavity, but more often forces its way into the substance of an adherent right lung, by a distinctive suppurative process, and in favorable cases passes by a free opening into a bronchus, whence it is discharged. The stomach, the duodenum, and the colon are the principal abdominal organs into which abscesses of the liver discharge themselves.

The hepatic abscesses are sometimes superficial, but more frequently deep-seated, and may be developed in any part of the gland; although they are most frequently found in the posterior portion of the right lobe. In size and in number they vary greatly, although they are rarely more than three, except in cases of pyæmia.

**Causes.**—Abscess of the liver is rare in temperate climates. It has been caused by—(1.) Contusions; (2.) *Metastatic* or *pyæmic* inflammation, as inflammation of the portal vein, the irritation of veins from disease about the rectum—*e. g.*, fistula in ano, operations for hemorrhoids; (3.) Inflammation and ulcerative processes in the gastro-intestinal canal, as in dysentery (see vol. i, p. 558); (4.) Inflammation and ulceration of the stomach, gall-bladder, or gall-ducts.

**Symptoms.**—Fever, rigors, and severe headache and delirium are not uncommon as early phenomena of suppurative inflammation of the liver; but sometimes there are no symptoms pointing to disease of the liver; and the difficulties which embarrass the diagnosis of suppurative hepatitis cannot be overrated. In 13 per cent. the disease runs a perfectly latent course, and in only 8 per cent. are symptoms at all well marked (Louis). In most cases a correct diagnosis will only be arrived at by not relying upon individual symptoms: but by taking a general view of the mode of origin and entire clinical history of the case, and after excluding, by comparison, the diseases of the liver and the neighboring parts which may give rise to symptoms similar to those of hepatitis.

Practically the expression of symptoms denotes either—(1.) A superficial and adhesive inflammation of the organ; or (2.) A deep-seated suppurative inflammation of the substance of the liver.

The most prominent symptoms of hepatitis are, however, some tumefaction, pain, or uneasiness of the liver, or of the adjoining parts, as the thorax, abdomen, or right shoulder; an affection of the bowels, as diarrhœa or dysentery; and, lastly, pyrexia in a continued, remittent, or intermittent form.

When pain is present, it is found to be in most instances aggravated by lying on the right side, apparently from the greater weight pressing on the liver; while, in a smaller number of instances, the pain is felt most acutely on turning on the left side, probably from adhesions having formed to the ribs. In general, however, the easiest position is on the back, or a little over to the left side; and

towards the termination of the disease the patient is sometimes observed lying in a position which he had previously declared himself unable to assume. As the disease advances, the pulse becomes frequent and hard, the skin hot, dry, and constricted, while pain, cough, and dyspnoea increase, and indicate advancing disease (MARTIN). Such a train of symptoms indicates superficial inflammation.

In a few instances acute hepatitis exists without any pyrexia. Some fever, however, is commonly present, and in general it often commences with shivering, vomiting, and purging—symptoms which gradually diminish in a day or two, leaving the patient comparatively free from fever, and the pulse nearly natural. These paroxysms, however, occur at intervals of various durations, sometimes returning as regularly as those of intermittent or of remittent fever; while, in other cases, the periods are less marked, the chief symptoms being rigors occurring at irregular intervals, frequent pulse, and sweats, the latter chiefly occurring in the night, and so copious as in some instances to pour off the body of the patient.

The state of the tongue is usually furred and loaded; but in the course of a long disease it becomes clean, or is only slightly foul. In some few instances, however, it becomes brown and dry.

These general symptoms of suppurative inflammation are insidious in approach, and the destruction of the substance of the liver proceeds silently and rapidly. The existence of the disease is often not known till severe structural changes in the organ manifest themselves, and then they run a rapid and often fatal course. This is especially the case when the inflammation is induced by the combined effects of heat and malaria. The miasmatic affection of the blood, from deranged mucous surfaces, and from contaminated matters, brought by the splenic vein, contributes to hyperæmia of the liver, which is often unnoticed till extensive inflammation develops itself. A burning sensation, with a mottled appearance of the skin of the hands and feet; an irritable temper; a capricious appetite; languor and persistent feverishness; frequent, settled, and increasing pains in the shoulder and back, are all pathognomonic signs of suppurative inflammation going on in the liver. The fever may be from time to time of a continued or intermittent type, and *the patient emaciates slowly*, becoming sad and desponding. The face becomes pale, cachectic, and has a patched or blotchy appearance. Sometimes there is jaundice, followed by anasarca and ascites, intestinal catarrh, bilious diarrhoea and dysentery. When such signs are observed, it is the duty of a physician to recommend a change of climate (LOWE).

The animal functions, as in phthisis, are often marked by the “cheerful hope” which illumines every hour the patient has to live; but in others the depression amounts to despondency, with restlessness and want of sleep. At last, however, delirium obliterates the past, and throws a veil over the future, till the patient dies.

In the midst of the symptoms that have been mentioned, perhaps an abscess points; and the pus may have formed in the absence of rigors usually indicative of its formation. The patient

then becomes hectic, his pulse rapid, and he is covered with a copious and clammy sweat. His life now in a great measure depends on the part where the abscess points; and when it tends to open spontaneously through the thoracic or abdominal wall, the locality is usually the space below the ensiform cartilage of the sternum.

**Treatment.**—The great experiments which have hitherto been made in the treatment of hepatitis are by bleeding, the use of mercury, and active purgatives; and it may be affirmed as a general result that bloodletting will not cut short the morbid process in acute hepatitis; and Frerichs is of opinion that it should only be ventured on in cases of traumatic hepatitis, and in robust plethoric persons, where there is great tenderness, with enlargement of the liver, and urgent dyspnoea. In the young and sthenic European in the East Indies, it is in general necessary to take fifteen to twenty ounces of blood, or till the skin becomes soft and relaxed, or the pain abates. Much benefit may be derived from the local application of leeches and large blisters.

One practical rule seems to be established with respect to the use of mercury in the treatment of hepatitis, which is, that it is not only inefficient, but injurious, and should not be given except as an occasional purgative. When congestion is known to exist, leeches may be applied with benefit, combined with purgative extracts and antimonials. The local abstraction of blood is also efficiently accomplished by leeches round the anus. They act more directly on the portal circulation than over the hepatic region. Ice applied to the region of the liver is also of great service, and the diet should be as limited as is consistent simply with the maintenance of life.

Purgatives are of use when the intestinal functions are sluggish.

In considering the treatment of abscess, and especially the points bearing on surgical interference, it should be remembered that hepatic abscesses tend to point or empty themselves in the following directions. These are stated in the order of their relative frequency: (1.) Through the lungs (from 10 to 30 per cent.); (2.) Through into the peritoneum, the stomach, or some part of the intestinal tract (nearly the same proportion); (3.) Through the external integuments (in the minority of cases).

With regard to the treatment of abscess when it tends to make its way outwardly, it is still a question whether or not an artificial opening ought to be made, the prominences of the false ribs and obliteration of the intercostal spaces being considered sufficient, in the absence of fluctuation, to justify the operation (FRERICHS). In its performance care must be taken to prevent the entrance of pus into the abdominal cavity. With this object in view, the following method has been recommended by Bégin and Recamier: (1.) Being decided as to the limits of the abscess, the patient is (2.) laid on his back, with the upper part of the body bent forward and the thighs flexed upon the abdomen; (3.) An incision from two to three inches long is to be made over the abscess, dividing the skin, the subcutaneous adipose tissue, the muscles, and the aponeurosis; (4.) The peritoneum is then laid open, as in operating for hernia,

by slitting it up on the grooved director to the same extent as the primary incision ; (5.) The wound is then to be dressed with charpie, and to remain untouched for three days ; (6.) At the end of three days the dressing is to be removed, when the capsule of the liver will be found to have contracted adhesions to the margins of the wound so firm that the abscess may be opened without the danger of pus passing into the abdominal cavity.

Dr. Budd mentions the following expedient as a method of indicating whether the liver adheres to the abdominal parietes or not. When the liver is large, and the abdominal parietes are thin, it may be made out "by feeling the edge of the liver, or some prominent part of its surface, and marking the place of this with a pen on the surface of the belly. If the liver be adherent to the abdominal parietes, the line or spot so marked will correspond to the edge or prominence of the liver in all positions of the body. If it be not adherent, the liver will slide along the wall of the belly when the patient draws a deep breath, or changes his posture—the liver will fall, for example, towards the left side when he turns from his back over to that side, and the line or spot will no longer correspond to the edge or prominence in question" (*op. cit.*, p. 122).

A simple puncture is only warrantable when the pus has already penetrated through the superficial layer of the abdominal aponeurosis, or the intercostal muscles. In all other cases it ought to be avoided as dangerous. Indeed, Mr. Lowe (whose valuable records of cases of hepatic abscess in the *Madras Quarterly Journal*, for April, 1863, merit the most careful study) thinks it better to allow an abscess of the liver pointing through the abdominal wall to open of itself, for the following reasons: (1.) Because of the inelastic structure of the lobular substance of the liver not permitting the cavity to contract when a free opening has evacuated the pus ; (2.) Because air invariably enters when an artificial opening is made, and rapid decomposition of the pus takes place, and renewed inflammation of the walls of the sac sets in ; (3.) This renewal of inflammation and fever may end in gangrene, and may thus rapidly prove fatal ; (4.) When the operation is entirely left to nature, *small worm-eaten-like openings serve to discharge the pus*, so that it has slow but constant escape. As these apertures never close up, and as the matter is always oozing out, air cannot enter, no decomposition takes place (no septicæmia occurs), and no secondary fever sets in. The patient feels no shock from the loss of the matter, which escapes so gradually, and as it escapes, so Nature closes up the walls of the sac. Such is also the opinion and advice of Dr. Budd.

But others consider that the abscess ought to be evacuated under certain conditions. For example, a most interesting case of hepatic abscess is related by Sir Henry Cooper, of Hull, in the *British Medical Journal* for May 23, 1863. Sir Henry Cooper justly takes exception to the method of Bégin and Recamier, because it may provoke the very danger it is desirable to avoid ; and he believes that in all cases in which the suppuration has extended so near the surface of the liver as to give the sense of fluctuation, irritation

and adhesion of contiguous surfaces have taken place. The process also interposes a serious delay at a critical period. Therefore he advocates a direct opening into the abscess, *when there is reasonable ground for believing that an external outlet for the matter is the direction taken by the abscess*. Peritoneal connections are sure then to have taken place. Tenderness of the tumor he regards as the most satisfactory indication for the operation, and a hardened base of effused lymph a certain confirmation. Delay exposes the patient to the risk of rupture of the walls of the abscess by coughing, sneezing, or the like, and to the laceration of any adhesions which may have formed; or it allows him to perish unrelieved from the effects of constitutional irritation and consequent exhaustion.

A middle course was devised and carried out by Dr. Graves, in the Meath Hospital, in the case of a robust man. External elevation, hardness, and pain, eventually confined to one spot in the right hypochondrium, after acute inflammation of the liver, left no doubt of the formation of an abscess. The hardness was followed by deep-seated softness, yet no tendency was shown by the abscess to point outwards. The swelling remained stationary, and the integument of natural color; but the general strength began to fail, and it became an important question whether the abscess should be opened by operation. It was objected—(1.) That the external tumor was diffuse; (2.) That the exact site of the abscess was uncertain; (3.) That failure to evacuate its contents might prove detrimental. A decision was therefore given *against* operation, by the surgeons of the Meath Hospital. Under these circumstances, Dr. Graves remembered that he had seen several cases where an incision made over a deep-seated abscess had failed to give vent to the matter in the first instance; yet in the course of a few days the pus found its way to the incision, and burst through it. He therefore proposed that an incision about four inches long should be made, exactly over the centre of the tumor—that it should be carried through a considerable depth of muscle, and if possible, be continued *to within about one or two lines of the peritoneum*. This incision was then to be plugged up from the bottom of the wound with lint, and thus kept open, in the hope that the pus might tend towards the incision, and finally burst through it. Such an operation was performed. The abdominal muscles were found of considerable thickness, and quite healthy; and although the incision was very deep, yet the situation of the hepatic abscess was not felt more distinctly, so that it now became quite evident that no prudent surgeon would have persevered in an attempt to open directly into it. A result was therefore waited for. In two days afterwards the patient sneezed, when purulent matter in very large quantity burst forth through the wound. On closer examination it appeared that the incision had not been made exactly over the abscess, but rather to one side of it; for the matter did not come from the bottom, but from the side of the wound, and pressure on the liver on that side caused matter to flow out in abundance through the wound, which communicated laterally with the abscess.

This case teaches that if the attempt had been made to open the



abscess directly, by continuing the incision, it would have failed, and the opening into the peritoneum and liver perhaps proved fatal (*Dublin Hosp. Reports*, vol. iv, p. 40).

After the abscess has opened, strict rest must be enjoined ; and sometimes the cavity is very long in closing up. Convalescence is always tedious ; and sometimes the cicatrization of the abscess is imperfect, continuing to discharge pus at intervals for years (FRERICHS).

Because the liver has been repeatedly punctured (it is said, deeply) with an ordinary trocar without any evil consequences, beyond slight local irritation, it has even been argued that our efforts should be directed to detecting by acupuncture the seat of an abscess, and evacuating it as soon as possible ; and that this method of exploration or "prospecting the liver" should be commenced as soon as symptoms present themselves indicating abscess of that organ ! It has been argued that because some cases have recovered after such haphazard methods of finding and evacuating an abscess, it is a practice which should be made the rule, as already stated, in place of waiting for some indications that pus has even formed in the liver (MURRAY, CAMERON). Statistical data regarding the actual value, or even the safety, of this method of thrusting trocars into the liver in search of abscesses have not been published ; therefore the recommendations to the plan rest upon the opinions or impressions of two or three individual men, the records of whose practice are unknown. Certain it is that the plan recommended is opposed to the well-established principles upon which surgical interference rests, and is opposed to all that is known regarding the pathology of hepatic abscess, as set forth in the carefully recorded facts of Waring, Martin, Morehead, Frerichs, Budd, Maclean, and Lowe.

It is also stated by Dr. Cameron (the latest exponent of this method of puncture), that in cases of hepatic enlargement, where the trocar has been used more than once in an unsuccessful search for an abscess, the operation was followed by the speedy absorption of an enlargement of the liver, which had resisted all the routine methods of bringing about its dispersion—and that this is a native mode of dispersing hepatic and even splenic enlargements in India (*Lancet*, August 8, 1863, p. 169).

Puncturing parts, or acupuncture with a very fine needle, is a surgical operation said to be much in use amongst the native Chinese and Japanese hakims ; but the statement requires confirmation, and accurate information regarding the results of such operations are not on record. It is obvious, therefore, that such modes of dealing with large livers, or *probable* hepatic abscesses, cannot be recommended here ; and it should be remembered that an hepatic abscess always begins from multiple foci (ROKITANSKY). But we must still look to physicians of large Indian experience for information on this important subject.

The time which elapses after opening an abscess till the patient's recovery is generally from one to two months. When convalescence is established, the functions of the liver remain torpid and its sub-

stance often indurated; and subsequently, also, the spleen may be enlarged. In such a state Sir Ranald Martin recommends the use of *nitro-muriatic acid* baths. They promote the depurative functions of the liver, kidneys, bowels, and skin.

“The form and manner of preparing and using the acid bath are as follows: Take of hydrochloric acid three parts; nitric acid, two parts; mix the two acids carefully and slowly, so as to avoid evolutions of heat; and having waited for twenty minutes, add of distilled water five parts, and mix the whole carefully.

“*For a General Bath in which to Immerse the whole Body.*—(1.) Pour into the bath about five pailfuls of cold water; add two quart bottles, containing sixty-four fluid ounces of the dilute nitro-muriatic acid, prepared as above, and then sufficient boiling water to raise the temperature to 96° or 98°. The body is to be quickly and thoroughly dried with warm towels; and afterwards the patient must retire to a well-aired and warm bed. The use of the bath is only to be discontinued when tenderness of the gums or general *malaise* occur; and cuticular irritation from the acid is to be avoided by diminishing its strength. Iron and other forms of tonic remedies may also be administered at the same time, as well as opiates” (Martin *On Climate*, p. 564, *et seq.*)

**VII. Increased Secretion and Elimination of Bile**—This condition is brought about by some increased excitement of the liver, as by certain kinds of food or drink; but more especially, as is now well known, it is brought about in Europeans by exposure to unusually high ranges of temperature. In summer and autumn it is a morbid state not uncommon in our own country; but to the European on first landing in India it most commonly occurs, and in either case it occasions what is termed “bilious diarrhœa.” Great increase of temperature, combined also, perhaps, with change of climate and an unsuitable mode of life, is now acknowledged to have a direct influence on the functions of the liver, expressed, in the first instance, by an unmistakable increase in its secretion. As a complication of other diseases, hepatic disorders are of most frequent occurrence, and for various reasons no true estimate has yet been made relative to the frequency of hepatic diseases either in this or other countries. Like most other exciting agents, however, the prolonged exposure to the influence of increased temperature, under conditions such as are experienced by the European in India, ceases to have a stimulant effect, and a depression in the powers of the organ results, corresponding to the previous excitement. In India the duration of the exaltation of hepatic function is not found to be of long duration, but is confined to the earlier years of residence, declining from that time. This increase of secretion and its elimination, so long as it lasts, is justly regarded as a salutary effect which nature provides to maintain the health. It is the decline of the powers of secretion, the arrest of the function, and the more or less sudden suppression of the secretion, which is attended with danger. It always precedes the expression of the inflammatory tendency, and is associated with congestions of an active kind, in which the arterial system becomes prominently excited. This is consistent with what

occurs in every other secreting organ. Previous to the expression of the inflammatory process becoming developed in a gland, the secretion at first flows freely under excitement (not necessarily morbid); but so soon as the inflammatory tendency becomes expressed, the secretion is at the same time dried up. This is well seen in the phenomena of a common cold, when the exciting cause at first merely stimulates the nasal passages, as marked, for instance, by an increased flow of mucus from the nose; but so soon as the inflammatory state becomes fully expressed, the secretion is dried up, and when re-established, is unmistakably altered in its properties. So it is with the liver when the increased flow of bile is suspended, from exposure to cold or some other exciting cause, a warning is given that the inflammatory tendency is about to be expressed by congestive and exudative processes in the texture of the organ.

**Symptoms.**—With excessive secretion of bile, the patient has purging of bilious stools, causing scalding of the anus. There is slight sickness just before the bowels act. A bitter taste is felt in the mouth, and the tongue is foul.

Copious draughts of hot water act as an emetic—dilute the bile; and if some saline purgative be added, the congestion of the liver will be relieved. If pain prevails over the liver, leeches and fomentations are indicated, to be followed by Plummer's pill at night and a black draught in the morning. The diet must be extremely limited and strictly regulated, as already noticed under congestions of the liver.

In persons who die soon after arriving in India, Mr. Twining remarks that the gall-bladder is commonly distended with bile.

**VIII.—The Degenerations of the Substance of the Liver** consist of—(1.) *The lardaceous*; (2.) *The fatty*; and (3.) *The pigmentary*; the general pathology of which have been already fully considered in the first volume of this text-book, p. 124.

1. *The Lardaceous Degeneration*, though frequently associated with fatty degeneration, has no necessary connection with it, but is an immediate alteration or degeneration of the glandular hepatic cells (BENNETT, FRERICHS).

The lardaceous liver is usually pale or fawn-colored—sometimes congested, so that it has a distinctly mottled appearance. The limits of the acini are unusually well defined, and the section or cut surface appears smooth, dry, and firm. The tissue is extremely slow to decompose; its specific gravity is notably increased, in some instances above 1080; but it may be reduced by fatty degeneration coexisting. Microscopically the entire structure seems changed, but it does not appear in which tissue the change begins. Meckel says it begins probably in the cells, and Virchow has traced its origin very clearly here. The peculiar structural formation of the liver has enabled the advance of the degeneration to be observed. His observations show that,—First, the branches of the hepatic artery (intralobular vessels) are affected; and when the degeneration has become established in an acinus, three zones, varying in color and appearance, are observable,—(1.) The outermost is composed chiefly of portal vessels, and is generally the seat of more or

less fatty degeneration; (2.) The innermost zone is made up of the intralobular vessels (of which the vein is prominent in the centre when divided in sections); these are surrounded by pigmentary deposit normal to the liver; (3.) The intermediate zone is the site of the amyloid material, translucent and firm. After the vessels have degenerated, the secreting cells about them become involved. They acquire a homogeneous hyaline appearance, like particles of rough ice. They lose their nuclei. A cell-wall cannot be seen; but they appear as an ill-defined, pellucid, glistening mass.

When the degeneration exists in an extreme degree, the greater is the translucency of the tissue, and the pellucid appearance gradually extends over the entire area of a lobule, and the few blood-vessels it seems to possess emit a little watery blood only. Rarely it occurs in isolated patches; generally it is distributed throughout, but more marked in some places. Towards the circumference of the liver, and shining through the clear peritoneal covering, the lobules or acini may be seen mapped out in a remarkably defined manner; in fact, in no disease of the liver is the appearance of lobules (which as a rule are not marked) so distinct as in this particular morbid condition (Wilkes's *Guy's Hospital Report*, p. 123, 1856). This is due to the way in which the change begins and progresses. The fatty degeneration at the margins makes the acini still more clearly obvious; and the approximation of the two degenerations (fatty and amyloid) gives an opaque white appearance, which encircles the acini and maps them out in this perfectly defined manner. A section well washed will also show the dead-white opaque substance running in the course of the portal vessels, surrounding the more transparent substance of the acini, in the centre of which the hepatic vein is apparent.

By these characters the lesion must be distinguished from the simple fatty degeneration of the liver; for it is now certain that up till the year 1854 these two forms of degeneration have often been confounded. But in the appearances shown by the livers affected by these several degenerations the differences are well marked. Louis evidently confounds the waxy and fatty liver in phthisis under the latter description; and the confusion has been transmitted from this great authority.

The large and hypertrophic livers, in their extreme degree, are for the most part due to the waxy degeneration, either simple or combined with the fatty degeneration; whereas the fatty degeneration is often present in the liver to an extreme degree without much or any hypertrophy. In the purely fatty liver, also, the specific gravity is not raised, but reduced. It is recorded as low as 1005. When the fatty coexists with the waxy degeneration, the specific gravity of the organ may be normal. Fatty liver consists of precisely the same elements as the normal liver in a similar structural arrangement, but an essential element is morbidly increased in its cells. The aggregate of solids and water may be slightly increased or diminished, but the percentage on the whole is reduced. Oil may be deposited to an enormous extent in the gland-cells; and these growing or swelling out, the volume of the

organ, as well as its weight, may be increased to a very great extent.

A form of cirrhosis is due to the waxy liver, as well as to the fatty. In the former it assumes at once the *atrophic* form; and all the essential elements simultaneously disappear. The atrophy commences in the glandular epithelium or hepatic cells, and thus gives rise to an apparently relative hypertrophy of the fibrous tissue.

On the other hand, cirrhosis is a complex process, a lesion due to actual growth, hypertrophy, and contraction of connective-tissue.

*Characters of the Minute Tissue-Elements in Lardaceous Liver—*

(1.) *Gland-Cells*.—The finely granular contents gradually disappear, giving place to a homogeneous, clear substance, which ultimately replaces the cell. In a few hepatic cells the mark or trace of the nucleus may still be seen of a shining lustre; and, when completely altered, the cell resembles a brilliant pellucid homogeneous scale. The cells, so degenerate, are excessively coherent; not easy to separate; and, indeed, so changed that a large solid aggregate mass is alone recognizable, in which neither cell, nor areolar matrix, nor bloodvessel is to be distinguished.

(2.) *Bloodvessels*.—The walls of the more delicate vessels become thickened, rigid, homogeneous, and lustrous. Their channel is narrowed, and ultimately entirely obliterated; while the still patent portions remain patulous on section. They appear as pellucid, transparently colorless cylinders, in which no trace of the delicate structure of bloodvessels can be detected.

It is difficult to determine to which system the vessels so affected belong; but, so far, it is an ascertained fact that the minute ramifications of the hepatic artery are chiefly implicated in the first instance. Frerichs has "repeatedly observed diseased capillaries, the locality of which appeared to correspond to the situation of the portal and hepatic veins. Solution of iodine gives a deep red color. The careful and slow addition of sulphuric acid changes the deep red to a dirty violet, and rarely to a blue tint. The tissue which remains exempt from the degeneration is distinguished by the greater amount of blood, and by greater softness and moisture." The chemical character of the lardaceous liver has been already considered in the first volume; and it may simply be noticed here that Frerichs has especially examined into any relations which may subsist between the *glycogenic material* normally elaborated by the liver, and the *amyloid substance* which is the result of the morbid process now under consideration. Both substances are colored by iodine; but a chemical reaction takes place in the case of the *amyloid substance*, and both are regarded as carbohydrates. The results are mainly negative—sugar being furnished by the *glycogenic material*, when digested with saliva, or when brought in contact with albumen, but not by the *amyloid substance*.

This form of disease has long been known as a liver affection; and Rokitansky first gave a clear account of its essential characters, and recognized the relation of the lesion to certain *cachexia*.



Bund describes the affection as a "scrofulous enlargement," and Oppolzer as the "colloid liver."

**Diagnosis.**—Frerichs relates a case of waxy liver in a girl aged nine years, in whom ascites was present; and the fluid drawn off contained a large quantity of sugar. With the exception of cases of diabetes, sugar has never yet been found in ascitic fluid; and therefore its presence, it is suggested, may be diagnostic of waxy liver. Dr. Wilks found a trace of sugar in the fluid from the peritoneum of a similar case. There are also general and local grounds for suspecting the existence of amyloid disease. The several symptoms may be stated as follow:

1. When there is general ill-health, expressed by *marasmus*, *anæmia*, or *dropsy*, which constitute the primary symptoms in cases otherwise ambiguous, and which in some cases are associated with diarrhoea, vomiting, and cardiac systolic murmur.

2. In cases where (after examining the blood) such symptoms as are mentioned cannot be traced to lesions of such organs as we have hitherto been accustomed to refer these phenomena.

3. In cases where the constitution is enfeebled, and health is impaired by *ulcerations of bones*, *syphilis*, *intermittent fever*, *tuberculosis*, *malaria*.

The local indications of lardaceous liver may be thus given from Frerichs's cases:

(1.) Uniform enlargement of the organ; (2.) Increased consistence, indicated by firmness; (3.) Association of these characters with tumid spleen and albuminous urine; (4.) Association of these characters with any of the conditions enumerated in the general symptoms under (3.)

The prominent general symptoms of this fatal degeneration being "anæmia, prostration, exhaustion," the condition of the liver, spleen, and kidneys should be investigated in all such cases, and their condition recorded in all cases of *syphilis*, *caries*, *necrosis*, and *intermittent fever*; and it is desirable to limit the suppuration of bones as much as possible by early surgical interference. Death usually occurs by exhaustion, unless a rapid end is brought about by *purulent peritonitis*, *dysentery*, *pneumonia*, *gangrene*, or *œdema of the lungs*.

2. *Fatty Degeneration of the Liver* is due to an abnormal increase of the fat naturally contained in the hepatic cells, so that they become engorged with oil. The nuclei are thus obliterated or obscured. The morbid condition is generally associated with free living, the influence of *alcohol*, of *syphilis*, the deprivation of exercise, and exposure to a heated air, with a large supply of food.

The Symptoms are chiefly gastric catarrh, indigestion, diarrhoea, anæmia.

3. *Pigmentary Degeneration of the Liver* is only to be recognized at a post-mortem examination. It is seen in cases of severe remittent, intermittent, or malarious fevers. It is due to the accumulation of pigment-matter in the vascular apparatus of the gland, especially in the capillary network of the portal and hepatic veins; and the minute branches of the hepatic artery also contain quantities of

black coloring matter. In cases where the liver is so affected, similar melanic matter is generally found in the spleen, kidneys, and brain; while the blood itself may be seen to contain dark granular masses, or nucleated pigment-cells, with black granules in their interior. The spleen seems to be the seat of formation of the melanotic matter (FRERICHS), whence it passes into the portal vein.

The effects upon the system of this degeneration are mainly due to the destruction of blood-corpuscles with which it is associated, and tending to a condition like that of *chlorosis*. The bile is usually secreted in large quantity. There is extensive capillary stagnation, which gives rise to obstruction of the circulation of the blood in the roots of the portal veins—and exhausting hemorrhages of an intermittent kind are apt to occur from the gastro-intestinal mucous membrane. Profuse diarrhœa, vomiting, and serous effusions, are also common occurrences. In India it tends to *suppurative hepatitis*.

**Treatment of Degenerations.**—The main indications consist in the removal of the causes, or the cure of the disease which has induced the degeneration. Easily digested and nutritious food must be given, and especially where chronic congestions prevail, a strict diet plays a powerful part in effecting a cure. Alcoholic drinks, sugar, and fat, are to be avoided, as well as amylaceous substances. *Aloes*, *rhubarb*, or *sulphate of soda*, are useful to remove the torpidity of the bowels. *Muriate of ammonia* is recommended by Dr. Budd, in doses of from *five* to *ten* grains three times a day. He believes it probably relieves the liver, and does good by promoting the action of the skin and the kidneys.

**IX. Jaundice, Icterus, or the Yellows**, are terms which comprehend a group of diseases, many of the different tissues and fluids of the body being dyed yellow, but more especially the conjunctiva and the connective tissue.

**Pathology.**—Jaundice, though often a result of every organic disease of the liver or duodenum, yet often occurs when those organs are perfectly healthy or simply congested. On posthumous examination, besides the yellowness of the cutis, the serum of the blood is generally found loaded with bile, and perfectly yellow. If the disease has continued for some time, the fat is also yellow, as well as the bones and cartilages, the serous fluids, and even the milk expressed from the breast of the female.

The theories that have been advanced to account for jaundice are, that the bile exists formed in the blood, and is merely removed by the liver, and that consequently jaundice is a consequence of the non-separation of the bile. A more common opinion is, that bile is a secretion, and not a mere separation, and consequently that in jaundice the bile is first secreted and then absorbed both by the veins and lymphatics; while Portal has proved that it may be absorbed also by the lacteals. The theory of jaundice especially advocated and expounded by Dr. Budd is, that the disease may arise in two ways,—(1.) By mechanical obstruction to the passage of the bile into the intestines, and the consequent reabsorption of the detained fluid into the blood; (2.) The suppression of the biliary secretion arising from some morbid condition of the liver itself, whereby

biliary ingredients accumulate in the circulation. Some of these ingredients or constituents of bile are generated in the liver itself (*e. g.*, the bile acids); others exist preformed in the blood (*e. g.*, the green bile-pigment, or *biliverdine*, and the *cholesterine*).

The mechanism of jaundice has therefore been regarded from two points of view, namely,—(1.) Jaundice from suppression, retention, or non-elimination (BUDD, HARLEY); (2.) Jaundice from reabsorption of bile (FRERICHS).

The former of these forms of jaundice is characterized by the rapid accumulation of green bile-pigment in the blood, until the serum, and the tissues, and the urine are saturated with the pigment. Over this secretion the mental state seems to exercise a very remarkable influence—so much so that mental emotion, favoring congestion of the organ, favors the stoppage of the secretion. Thus jaundice by suppression or non-elimination arises from (1.) Innervation (HARLEY). Active and passive congestion similarly induces jaundice; hence jaundice by suppression or non-elimination arises also from (2.) Disordered hepatic circulation (HARLEY). (3.) Another form of jaundice from suppression or non-elimination arises from loss or destruction of the secreting cells of the liver, as in acute and chronic atrophy, cancer, tubercle, fatty and amyloid degeneration. Hence jaundice by suppression or non-elimination arises also from the loss or absence of the glandular hepatic substance (HARLEY).

The second class of cases of jaundice arises from the reabsorption of the secreted but retained bile. They are also characterized by the accumulation of pigment in the blood; whence it stains the tissues, the urine, and the serum. The bile in these cases is absorbed from the distended ducts and gall-bladder; and the biliary products manufactured in the liver, equally with those formed in the blood, find their way back into the circulation, to be eliminated by the excretions. Hence the bile-acids (absent in the former class of cases of jaundice) are present in cases of jaundice from reabsorption, as well as the bile-pigment. Obstruction may be due to several causes, but chiefly to the two following, namely: (1.) Congenital deficiency (very rare); (2.) Obstruction by disease, generally of parts in the vicinity of the head, of the pancreas, or of the *ductus communis chole-dochus*.

**Symptoms.**—Jaundice, from the different intensities of the color of the skin, has been divided into the *yellow*, the *green*, and the *black* jaundice, arising from functional disease. Jaundice may be sudden in its attack, or it may be preceded for a few days by great depression of spirits, lassitude, and somnolence. It may also be preceded or accompanied by some slight pain in the region of the liver, but more commonly pain is not present.

The first symptom of jaundice is a yellowness of the white of the eyes, then of the roots of the nails. The yellowness next appears over the face and neck, and ultimately over the trunk and upper and lower extremities. As soon as the eyes are affected, the urine becomes of a deep red color, and stains linen yellow, and if nitric acid be added, it is changed to a deep green. The bile, however, is not always in the same state of combination in the urine, nor of

the same quality; for in some instances, where the color of the patient is most marked, and the urine of its deepest hue, the addition of nitric acid effects no change. At the same time that the urine is thus discolored, the stools, often abundant in quantity, are copious and white. The pulse is slow, and the patient complains of a bitter taste in the mouth, has much thirst, an absolute inaptitude for all exertion, and suffers from a lowness of spirits amounting to hypochondriasis. In general the bowels are irritable and easily acted upon; but in a few cases they are constipated. If the patient recovers, the first symptom of recovery is the appearance of bile in the stools, after which the yellowness fades away in the inverse order of the attack.

The duration of this affection is such that in some cases it terminates in about ten days, but more generally it lasts from three to six weeks, and, if badly treated, oftentimes as many months.

**Diagnosis.**—This disease is to be distinguished from *chlorosis* and that sallow state which results from profuse uterine hemorrhage. In these complaints the white of the eye is clear, the urine limpid, and the stools healthy, so that the great characteristics of jaundice are wanting.

The difficulty is to determine how far the jaundice is due either to obstruction or to non-elimination (suppressed secretion); and to Dr. Harley we are mainly indebted for methods of clinically distinguishing these two classes of cases by chemical examination of the excretions. His principle of diagnosis from this point of view assumes that in jaundice from suppression of bile, only those biliary products which exist preformed in the blood accumulate in the circulation; but that in jaundice from obstruction, the biliary products which are manufactured in the liver, equally with those preformed in the blood, find their way back to the blood, to be eliminated by the excretions.

The absence of bile from the stools is indicated by their pipe-clay color, offensive odor, and presence of fat.

The urine of jaundice ranges from a saffron-yellow to a dark olive-green, or even black color; and its amount of pigment is best judged of by the intensity of the color of the uric acid crystals. But it is the presence of *biliary acids* in the urine which Dr. Harley considers characteristic of jaundice from reabsorption, as distinguished from jaundice arising from non-elimination or suppression.

The readiest mode by which the biliary acids may be detected is the following:

“To a couple of drachms of the suspected urine add a small fragment of loaf-sugar, and afterwards pour slowly into the test-tube about a drachm of strong sulphuric acid. This should be done so as not to mix the two liquids. If biliary acids be present, there will be observed at the line of contact of the acid and urine—after standing for a few minutes—a deep purple hue. This result may be taken as a sure indication that the jaundice is due to obstructed bile-ducts. On the other hand, the absence of this phenomenon, and the occurrence of merely a *brown* instead of a *purple* tint, although in the earlier stages of jaundice equally indicative of

suppression, is no indication of the cause of the suppression, which must be gleaned from other circumstances" (Harley *On Jaundice*, p. 61).

Dr. Felix Hoppe's method is as follows:

"(1.) Decompose the icteric urine to be examined with an excess of milk of lime; (2.) Boil for about half an hour; (3.) Filter; (4.) Evaporate the filtered fluid nearly to dryness; (5.) Decompose with a great excess of concentrated hydrochloric acid, and then keep the whole (before being again filtered) at the boiling-point for half an hour; (6.) To avoid spitting of the fluid, it is necessary to renew the volatilized hydrochloric acid from time to time; (7.) Leave the liquid to get completely cold, and then add six to eight times its volume of water; (8.) Filter the dark brown turbid solution thus obtained, wash out with water the residue on the filter, until the same runs through quite colorless; (9.) Dissolve the brown resinous mass on the filter in 90 per cent. alcohol; (10.) Decolorize by boiling with animal charcoal, filter, and evaporate to dryness in the water-bath; the residue is a yellow, resinous mass, which, if bile-acids be present, must consist, for the most part, of pure *cholidic acid*. In such a case it melts by warming, and emits the peculiar musk or soap odor. (11.) Lastly, dissolve in a very little caustic soda and some drops of warm water, add a very small piece of sugar, and allow three drops of concentrated  $\text{SO}_2$  slowly to fall into it. At first the fluid becomes milky and troubled, and resinous flakes separate, which stick pertinaciously to the glass, but afterwards, by the addition of more  $\text{SO}_2$ , these again dissolve, and produce a beautiful purple-red or dark-violet fluid" ("Abstract of Kühne's Paper on the Pathology of *Icterus*," by Dr. George Scott of Southampton, in Beale's *Archives*, vol. i, p. 343).

But these acids after a time gradually diminish and disappear from the urine in cases of obstruction and reabsorption—namely, when the secreting powers of the liver become impaired, when the hepatic gland-cells waste away; and then the abnormal products *leucine* and *tyrosine* appear in the urine. They are readily detected by the slow evaporation of an ounce of urine to the consistence of syrup, which is to be put aside to crystallize. *Tyrosine* is recognized by the fine stellate groups of needle-like crystals or spiculated cells, which appear like a rolled-up hedgehog with its bristles sticking out in all directions (HARLEY). It may be obtained in its pure state by (1.) Adding a solution of *acetate of lead* to four ounces of urine till a precipitate ceases to form; (2.) Filtering; (3.) Freeing the liquid from an excess of lead by a current of sulphuretted hydrogen; (4.) Again filtering; and (5.) Evaporating the clear solution. The *tyrosine* is now colorless, and crystallizes with the microscopic characters better marked. *Leucine* is known by its flat, circular, oily-like discs, without crystalline structure. It is soluble in water and boiling alcohol, but insoluble in ether.

If sugar appears in the urine in cases of jaundice, with a diminution of bile-acids hitherto in quantity, it is generally the forerunner of a fatal termination (HARLEY).

**Treatment.**—As a general principle, the larger number of cases of jaundice from functional disorder (perhaps four out of five) will get well in time spontaneously, but may be aided by remedies judiciously



selected according to the diagnosis already indicated. The first indication is to aim at removing the exciting cause ; and in jaundice due to congestion of the liver, purgatives seem to act beneficially in the form of *blue pill* or *Plummer's pill*, with *aloes*, and *nux vomica* with *rhubarb pill mass.* Acids and alkalies are alike counter-indicated in cases of jaundice resulting from active congestion of the liver. *Benzoin acid* has been recommended ; and Dr. Harley has found it useful in cases of suppression of bile (by innervation, for example) ; but in cases of jaundice from obstruction it is injurious. *Podophyllin* is also of use in jaundice from suppressed secretion of bile. It ought to be combined with *hyoscyamus*. It is especially useful in cases of feeble liver, combined with vegetable tonics, such as *gentian* and *quinine* ; but it ought not to be given in cases of jaundice from obstruction. The medicine of all others which has seemed most generally useful in Dr. Budd's experience is *sulphate of magnesia*, in half drachm to drachm doses, combined with fifteen grains of *carbonate of magnesia*, and half a drachm of *aromatic spirits of ammonia*, given three times a day an hour before food. The *sulphate of magnesia* maintains a free action of the bowels ; and the *carbonate of magnesia* neutralizes any excess of acid in the stomach or bowels (*op. cit.*, p. 289).

#### ACUTE OR YELLOW ATROPHY OF THE LIVER.

LATIN Eq., *Atrophia acuta* ; FRENCH Eq., *Atrophie aiguë* ; GERMAN Eq., *Acute Atrophie* ; ITALIAN Eq., *Atrofia acuta*.

**Definition.**—*Simple jaundice followed by violent constitutional disturbance, expressed by pyrexia, delirium, hemorrhages from various parts, and finally coma. The liver shrinks to one-half or one-third its normal size. A peculiar chemical decomposition takes place in the liver, whereby abnormal proximate principles are formed, which, being carried into the blood, may be discovered in various organs of the body, or, passing out by the kidneys, may be found in the urine.*

**Pathology and Symptoms.**—The disease was first described in this country by Bright and Graves, and more recently and minutely by Frerichs and Wilks. The liver undergoes a most remarkable atrophy, and its section presents a mottled red and yellow appearance. The microscope shows that complete destruction of its secreting cells occurs, so that in the most severe cases not a single cell has been left entire ; dark masses of biliary pigment, hæmatine, and fat alone remain ; and the kidneys are found gorged with new products. The redder portions of the liver are more destroyed than the yellow, which show remains of cells containing bile. The symptoms set in, like a bilious attack, with feebleness, rapidly followed by jaundice, febrile symptoms, and vomiting, delirium supervening by the third or fourth day. Convulsive fits soon pass into unconsciousness, associated with stertorous breathing, foaming at the mouth, clenched teeth, closed eyes, with pupils normal and susceptible. The pulse is at first slow, but at the outbreak of the nervous

symptoms it gradually rises to 110 to 120, and presents remarkable variations as regards frequency and volume. Towards the close of the disease it increases in frequency, and becomes smaller and smaller till it can no longer be felt. The symptoms generally resemble those of uræmic intoxication. The extent of hepatic dulness diminishes more and more as the disease advances, and not unfrequently the dull space disappears completely, without any tympanitic distension of the bowels to cause the dull sound over the liver to be obscured. The spleen, at the same time, is increased in volume. The bowels are almost always confined; and the stools are firm, dry, clay-like, deficient in bile, and at a later period not unfrequently dark-colored and tarry, from the presence of blood. The urine is more or less saturated with a brown coloring matter, presents the reaction of bile-pigment, and deposits a light or greenish-yellow precipitate, in which the microscope detects the epithelium of the passages and of the kidney colored yellow, and *needle-shaped crystals (tyrosine)* covered with coloring matter, either isolated or adhering in crystalline masses (FRERICHS). Sometimes the urine ceases to pass, and may be retained in the bladder as a clear bilious fluid, having a specific gravity of 1030 (WILKS); or ranging from 1012 to 1024, always of acid reaction, and occasionally slightly albuminous for short periods (FRERICHS). The evaporation of the urine gives crystals of *leucine*, which are in round masses, and of *tyrosine*, which are needle-shaped. Sometimes pure tyrosine falls as a sediment. These crystals are also found in the substance of the liver, but only in the hepatic vein, not in the portal vein nor hepatic artery (FRERICHS). The question as to retention of urea, or its lessening as due to a want of complete metamorphosis of albuminous products, is not yet determined (PARKES, *l. c.*, p. 286).

The disease runs a more or less violent and rapid course. In severe cases it has terminated at the end of twelve or twenty-four hours (FRERICHS). In other cases in from two to eight days, urgent symptoms ending the disease by extreme prostration or excessive vomiting (WILKS). It is scarcely ever prolonged to a week, except when it occurs after jaundice, when the whole duration of the disease may extend over four to five weeks. After the commencement of the characteristic symptoms, the disease almost invariably terminates in five days; and as a rule the termination has been fatal to life. The prognosis is, therefore, in the highest degree unfavorable. Hemorrhages are apt to occur (in one-half the cases) simultaneously from various parts of the body, usually from the stomach, bowels, or uterus. Post-mortem examination shows also extravasation upon mucous and serous membranes.

Pressure over the region of the liver gives rise to marked indications of pain, which are expressed even during coma.

**Morbid Anatomy.**—The size of the organ is found after death to be diminished in all directions, but especially in its thickness. The gland is flattened out. The parenchyma is flabby and shrivelled. Where the disease has advanced the farthest, the cut surface has an ochre-yellow or rhubarb-like color; and the bloodves-

sels are empty, and the traces of the outlines of lobules are obliterated.

At places where the process is less advanced extravasations exist, or their remains, in the forms of crystals of hæmatoidine. The lobules are encircled by congested vessels; and a dirty yellow substance separates them from each other. Afterwards the capillary congestion recedes, the size of the lobules diminishes, their color becomes yellower, whilst the intervening gray substance gradually increases.

Bright has recorded the weight of such livers as reduced to *two pounds*, or to *twenty-three ounces*, and even to *nineteen ounces*. Frerichs recorded the weight of one as low as *one pound thirteen ounces*; its relative weight to the entire body being as 1 to 68 or 54—i. e., a reduction of more than one-half.

**Causes.**—The mode of origin of acute atrophy of the liver is still unknown; but the circumstances under which the affection has occurred may be stated as follow: Mental distress, dissolute habits, venereal excesses, syphilis, drunkenness, nervous depression, pregnancy, and the influence of miasmata. Looking to the occurrence of albumen in the urine of pregnant women, it may be that atrophy of the liver and the *nephritis* of pregnancy are both due to some condition of blood which in certain states of the constitution may be induced by pregnancy (WILKS). Atrophy of the liver has been most frequently observed in females; and more than half were attacked during pregnancy; and the period of life between twenty to thirty years of age seems most predisposed to the disease.

Acute atrophy of the liver is apt to be mistaken for typhus fever, complicated with jaundice or with pyæmia. The range of temperature may help to indicate the disease, inasmuch as the temperature is said not to be increased in cases of acute yellow atrophy (see FRERICHS, vol. i, p. 217).

**Treatment.**—English physicians recommend emetics and purgatives (CORRIGAN, GRIFFIN, HANLON). Frerichs recommends that the stronger purgatives should be used simply to remove congestion, such as *senna*, *aloes*, *colocynth*; and the doses should be such as to secure profuse evacuations. When atrophy has distinctly commenced, no benefit results from medicinal treatment. Severe pains indicate the use of leeches, cupping, and cold cloths, or fomentations, as recommended under typhoid fever in vol. i. The occurrence of hemorrhage indicates the use of mineral acids.

[See an excellent article on "Acute Atrophy of the Liver, illustrated by cases, with some remarks upon the Similarity between this disease and the effects of Poisoning by Phosphorus," by Dr. John Homans, of Boston, in the *American Journal of the Medical Sciences*, July, 1868. Twenty cases are analyzed, of which five are American, including Dr. Homans's own case, with the post-mortem appearances minutely described; and three are published for the first time. With the exception of one case they are all more recent than the date of Frerich's work.—EDITOR.]

## [SYPHILOMA OF THE LIVER.

(DR. CLYMER.)

Of all the internal organs the liver most frequently suffers from the effects of constitutional syphilis. Professor Dittrich, of Prague, was the first to call attention to the characters and pathogeny of the syphilitic affections of the liver (*Prager Vierteljahrschrift*, 1850); they have since been studied by Frerichs, Gubler, Wilks, Diday, Von Bärensprung, Lacereaux, Quételet, Virchow, Leudet, and others. Syphilitic disease of the liver may result from hereditary or acquired syphilis. Met with in the foetus in utero, or at term, and in infants and young children, or later in life, where there is no physical or commemorative evidence of primary disorder, it is a common sequence of the acquired disorder at the so-called tertiary period, and may appear during the so-called secondary stage (DITTRICH, DIDAY, VIRCHOW, GUBLER, BLACHEZ). Generally from three to nine years elapse from the time of infection to the development of the hepatic troubles.

The evolution of hepatic syphiloma may be very rapid in the infant, all the several forms being found in the same subject (F. WEBER, VON BAERENSPRUNG, BLACHE, H. ROGER). In the adult its course is variable, though generally it is very slow.

**Anatomical Characters.**—Hepatic syphiloma may be ranged in groups: (1.) Capsular or peri-hepatitis. (2.) Interstitial hepatitis. (3.) Amyloid and other degenerations.

(1.) There is little doubt that clinically peri-hepatitis, partial or general, may exist alone, but on examination after death it is generally associated with (2.) Interstitial hepatitis,\* which may be (a) diffuse, or (b) limited. (a) The first is characterized by islets of connective tissue between the hepatic lobules; cicatricial depressions, more or less deep, and puckering, are seen also on the surface, from which fibrous bands dip down into the liver-tissue, which is atrophied or destroyed. (b) This form is most common; there are large, tough, flint-colored patches, or irregular nodules of fibroid tissue, on the surface, or in the substance of the organ. In some instances these knots have undergone further change, and have yellowish, cheesy, or even calcareous centres. On section, a nodule is found to be composed of resistant yellowish tissue, furrowed with numerous vascular arborizations. Its outline is not well defined, and it appears to be insensibly incorporated with the hepatic parenchyma, being united by bands of embryonic connective tissue. The cut surface is dry, even after scraping with the scalpel. When broken up by needles, large and small round nuclei, and round, ovoid, or fusiform bodies, in various quantities, are found. These nuclei and cells are firmly connected by a strong amorphous or fibrillar tissue. When examined under a magnifying power of 75 diameters, and chromic acid was added, Ranvier† found that each nodule contained a certain number of small nodules, touching each other at the circumference, but differing in structure externally and internally. At the centre are accumulated the nuclei and smaller cells, and a

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\* [In using the terms peri-hepatitis and interstitial hepatitis, it is not intended to convey the notion of an inflammatory pathogeny with respect to syphilitic affections of the liver. It is probable that both diffuse and nodular hepatic syphilomæ are similar to the deposits met with in other organs in constitutional syphilis, and that they cause by their presence subsequent nutritional changes.]

† [*Comptes Rendus de la Société de Biologie*, 1865.]

large proportion of the amorphous matter, in small irregular masses; towards the periphery the structure becomes more fibrillar, so that on the outside the lobules are composed of tissue resembling in all respects connective tissue. The bands passing between the nodules and the lobules of the liver are traversed by the terminal ramifications of the hepatic artery and duct, and of the portal vein. The cellular elements of the new growth are insinuated among the hepatic cells; at the boundaries of the nodule, there are atrophied globular hepatic cells, filled with yellowish granules interspersed in the new tissue. When there is total cell-atrophy, globular and stellate crystals of stearic acid, the remains of the former fatty matter, have been found. (3.) The waxy or amyloid degeneration appears in small scattered masses, pale in color, dense in structure, and closely resembling beeswax, with streaks of fibrous tissue throughout their substance and around their margins. Sometimes there is a diffuse fatty degeneration met with, which is most likely connected with changes in the fibroid and amyloid degenerations. The whole volume of the liver is generally increased—the *compensating hypertrophy* of Virchow—when there has been interstitial hyperplasia, or capsular hepatitis, or gummatous tumors. Even when cirrhotic, or amyloid, degeneration is present in several districts, the size of the organ may be natural, or nearly so; or there may be actual increase, from the supplementary development of the acini and cells in the healthy portions.

**Diagnosis.**—A previous history of syphilis, and the existence of constitutional infection, can only lead to a correct conclusion regarding the nature of the hepatic disorder, for syphilitic disease of the liver has no special symptoms, and indeed offers no single symptom that is even tolerably decisive (OPPOLZER). The pain, heaviness, and dragging, on exercise or exertion, complained of in the right side, the transitory jaundice, ascites, local œdema or anasarca, and enlargement of the organ, are more or less present in other hepatic disorders, and are not characteristic of hepatic syphiloma. The increase in the size of the liver is usually very slow, though cases are recorded where it was very rapid, as that of Pihant-Dufeillay (*Bul. de la Société Anatomique*, 2e série, t. vi, 1861), in which after two months the liver extended one inch and a quarter beyond its natural limits; and another of Axenfeld, which was still more remarkable in progress and bulk (*Bul. de la Soc. Anat.*, 2e ser., vol. viii, 1863). Of the liability of relapse there is unfortunately no doubt, but Frerichs has shown that it is not always fatal.

**Prognosis.**—The prognosis is by no means unfavorable. In the so-called capsular or peri-hepatitis a spontaneous cure is not infrequent. Evidences of previous hepatic syphiloma are constantly met with in persons who have died from other disorders, and have had no recent liver troubles. Leudet found syphilitic changes in the livers of nine patients, who had had constitutional syphilis, but who died,—2 of brain disease, 2 of lung disease, 2 of intestinal disease, and 3 of kidney disease.\* Virchow, in his late work *On Tumors*, says: "It is not unlikely that absorption may happen in syphilitic liver. The rapidity of cure of similar cases allows us to believe that, when the disease is not of long standing, the lesion may disappear" (p. 428). Even when the enlargement of the liver and spleen is considerable, and of some years' duration, and accompanied with ascites and albuminuria, all the symptoms and signs may disappear, and the liver remain in a state apparently not incompatible with good health (GRAVES, HANDFIELD JONES, FRERICHS, D'HERARD, PI-

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\* [*Arch. Gén.*, t. i, 1866, p. 151.]



HANT-DUFEILLAY, BUDD, LEUDET, &c.). By some the diminution in the size of the organ has been attributed to atrophy and tissue degeneration, and the cure regarded as delusive. But if the atrophy is partial, it does not follow that the function of the organ should be materially impaired; for, as Virchow, Frerichs, and others have shown, there is, under these circumstances, compensatory hypertrophy of the hepatic cells. Nor is there any reason to believe that atrophy necessarily or invariably follows diffuse enlargement; and it is not analogically improbable, as Virchow suggests, that, in some instances at least, absorption of the exudation may take place in limited hepatic syphiloma. As a rule, the prognosis is more favorable where there is simple hypertrophy, and the surface is free from nodules, and atrophy has not begun. When degeneration—fibroid or amyloid—has taken place, the prognosis should be guarded; but, from the interesting case of Dr. Grainger Stewart (*Brit. and For. Med.-Chir. Rev.*, Oct., 1864), there is reason to believe that, even where waxy masses exist, they may be measurably removed by subsequent changes and disintegration, leaving only deep fibrous cicatricial depressions. Coincident disease of other organs, and well-marked cachexy, either the result of the parent disorder, or indicative of serious consecutive tissue-degenerations, are adverse elements in the prognosis.

**Treatment.**—The mercurial vapor bath, iodide of potassium, muriate of ammonia, alkaline baths and waters, iron, and good diet, are the means to be relied on to arrest, if not to cure, the disease. Frerichs appears to use only iodide of iron, and alkaline baths and waters. Dr. Budd, of London, strongly recommends a long course of nitric acid, of which, in these cases, there is, he thinks, unusual tolerance, so that it may be taken continuously for many months, without inducing excessive acidity of the urine, or any inconvenience attributable to undue acidity of the stomach.]

#### SECTION V.—DISEASES IN WHICH LESIONS TEND TO BE LOCALIZED IN THE KIDNEYS.

A careful study of the structure and functions of the kidney is of the greatest importance to the student of medicine. The determination of the quality and quantity of the *excreta* eliminated in the form of urine is indeed one of the best methods for enabling him to determine not only morbid conditions of the kidney, but to appreciate many changes which go on in the body during disease—changes which have frequently been referred to throughout these volumes. In every case of disease (in wasting, febrile, and constitutional diseases especially) much may be learned regarding their natural history by a careful examination of the urinary excretions, and of the microscopic characters of the sediment, especially when studied in connection with the ranges of temperature of the body, the number of respirations, and the state of the pulse, during daily periods of twenty-four hours. A formula has been already suggested at page 128 as a method to be followed in daily records; and it is now clearly understood that the *absolute amount excreted in a given time, and referring that amount to body-weight*, are the only conditions which will yield anything approaching to accurate scientific results (ROUTH, PARKES). “In a medical point of view it is a mere waste of time to estimate the constituents in a certain quantity of urine,

passed at any particular hour of the day or night, without ascertaining the relation which that quantity, with its constituents, bears to the whole quantity passed during the *twenty-four* hours; and this is the more necessary as the amount of fluid secreted varies considerably in healthy persons" (SUTTON). The volumetric methods of determining the urinary constituents have greatly facilitated such investigations; and these methods are clearly expounded in the works of Hoppe, Neubaur, and Vogel, Thudichum, Beale, Hassall, and Sutton; and, above all, the profession is indebted to Dr. Parkes for his philosophical exposition of the value of our present knowledge regarding the state of the urine in health and disease, as set forth in his admirable work on that subject, already frequently referred to. From these works the following sections are compiled.

#### SECTION VI.—ON DETERMINING THE COMPOSITION OF THE URINE IN DISEASE.

It is examined for two purposes,—(1.) To discover the condition of the urinary organs; (2.) To determine the course of the abnormal metamorphoses of tissue in the body which lead to alterations in the composition of the several excreta.

The normal urinary constituents have probably a fixed physiological range concurrent with age and weight of the body, so that a person ought not to continue passing regularly an amount of any constituent very greatly above or below the limits of the range proper to him, otherwise some morbid condition must be sought for to explain the nature of the occurrence. The averages of the excreta for at least ten days should be taken in health, in order to arrive at an accurate standard. But as this is not possible in cases of disease, certain empirical formula are laid down by Dr. Parkes for calculating the urinary excretion in a sick person whose normal excretion is unknown. The following are the details of the facts to be determined and recorded:

I. Record the age and weight of the patient in pounds avoirdupois.

II. Collect all the urine passed in twenty-four hours; measure it in cubic centimetres, and record its absolute amount.

III. Observe and record as to the following general properties, viz.: (1.) Specific gravity; (2.) Uræmatine, as determined by Vogel's color table;\* (3.) Clearness or turbidity on emission or after rest; (4.) Deter-

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\* Vogel's color table is made by taking certain colors as starting-points, and representing the following groups for comparison:

•• I. *First group; yellowish urines*, represented by gamboge mixed with a greater or less amount of water, giving three grades, the first of which is almost colorless, as follow: (1.) *Pale yellow* (gamboge with much water); (2.) *Light yellow* (gamboge with less water); (3.) *Yellow* (gamboge with very little water).

II. *Second group; reddish urines*, represented by the above yellow grades mixed with a greater or less amount of red—i. e., gamboge with crimson lake in three grades, as follow: (4.) *Reddish-yellow* (gamboge with a little crimson lake); (5.) *Yellowish-red* (gamboge with more crimson lake); (6.) *Red* (crimson lake with little gamboge).

III. *Third group; brown or dark urines*, represented by gamboge, crimson lake, and more or less Prussian blue, in three grades, as follow: (7.) *Brownish-red* (red

mine the absolute weight, by multiplying the qu in c. c., by the figures expressing the specific g weight in grammes.

IV. Set aside the following quantities for th tion of—

1. Urea, . . . . .
2. Uric Acid, . . . . .
3. Phosphoric Acid, . . . . .
4. Chloride of Sodium, . . . . .
5. Sulphuric Acid, . . . . .
6. Degree of Free Acidity—
7. Sugar, . . . . .
8. Albumen—
- Solids, . . . . .

V. Collect and examine the sediment.

VI. Determine the amount of excretion nor the following empirical formula (PARKES): Mult by the weight of the person in pounds avoid excretion in grains, in twenty-four hours, of th the urine:

	In Men between 20 and 40.	In Women between 20 and 40.	In C 3 and
Urea, . . . . .	1.66	2.96	5.6
Chlorine, . . . . .	0.875	0.817	1.4
Sulphuric Acid, . . .	0.214	0.25	0.4
Phosphoric Acid, . . .	0.386	0.336	0.6

The following corrections are required: (1.) I forty and fifty, calculate according to columns 1 per cent.; for ages between fifty and sixty dedu between sixty and seventy deduct 30 per cent seventy deduct 50 per cent. (2.) If the person b or more days (as in some fevers), deduct one-th made according to the table; if the diet be meas one-sixth; if pretty plentiful, yet still below th tenth. (4.) If there be total inactivity, deduc merely quietude, deduct one-twentieth.

with an admixture of a little brown); (8.) *Reddish-brown* in the last); (9.) *Brownish-black* (almost black, with a (see Thudichum *On the Pathology of the Urine*, p. 184).

\* The precipitate from the urea estimation is sufficien

SECTION VII.—VOLUMETRIC ESTIMATION OF THE MORE IMPORTANT CONSTITUENTS OF THE URINE; AND THEIR PATHOLOGICAL RELATIONS.

*The Volumetric Method of Analysis* is one which enables ordinarily skilful operators, or medical men who cannot devote much time to practical chemistry, to determine with sufficient accuracy the amount of the most important constituents of the urine, such as *urea*, *uric acid*, *chloride of sodium*, *phosphates*, *sulphates*, and the *free acidity* of this excretion. Certain conditions are necessary for success, namely,—(1.) Solutions of the reagents or tests, the composition and strength, or chemical power of which are accurately known; (2.) *Burettes*, graduated tubes or vessels from which portions of the test-solutions may be accurately delivered; (3.) The power of determining by the eye when the decomposition produced by the test-solution with the urine has ceased, so that the quantity of test-solution used (*i. e.*, with which certain components of the urine have combined) may be accurately determined.\*

It is necessary to exercise the greatest care in the graduation of the measuring instruments, and in the strength and purity of the standard solutions; because a very slight error in the process is greatly magnified—in proportion, in fact, as it is multiplied to represent the amount in large quantities when the actual observation has been made upon a small amount.

1. **Estimation of Chlorides.**—They are calculated as *chloride of sodium*; the test-solutions required being—(1.) The *standard solution of the nitrate of mercury*; and (2.) The “*baryta-solution*,” as it is commonly called. The method was devised by Liebig, and its principle is as follows: “If the solution of nitrate of mercury, free from any excess of acid, is added to a solution of urea, a *white gelatinous precipitate* is produced, containing urea and oxide of mercury in the proportion of 1 eq. of the former to 4 eqs. of the latter. But when *chloride of sodium* is present in the solution, the *precipitate does not occur until all the chloride of sodium is converted into chloride of mercury (sublimate) and nitrate of soda*, the solution remaining clear. If the exact point be overstepped, the excess of mercury immediately produces the precipitate above described, so that the *urea* present acts as an indicator of the end of the process. It is therefore easy to ascertain the proportion of chlorides in any given sample of urine by this method, if the strength of the mercurial solution is known; since 1 eq. of oxide of mercury converts 1 eq. of chloride of sodium into 1 eq. each of corrosive sublimate and nitrate of soda” (SUTTON).

The steps of the process are as follow :

(1.) Take 40 c. c. of urine; (2.) Mix with 20 c. c. of the baryta-solution; (3.) Pour the thick mixture upon a small dry filter; and when

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\* The apparatus and the test-solutions required, graduated to the proper strengths, are supplied by Mr. Griffin, 119 Bunhill Row; and by Messrs. Bullock and Reynolds, Hanover Street, London; also Harper and Sutton, Operative Chemists, Norwich.

sufficient clear liquid has passed through, (4.) Take 15 c. c. of it (= 10 c. c. of urine), and just neutralize it, or render it acid by a drop or two of nitric acid; (5.) Bring this urine fluid under the burette which contains the test-solution of the nitrate of mercury, which is to be allowed to drop gradually, drop by drop, into the beaker containing the urine, which is to be constantly stirred with a glass rod; (6.) As soon as the precipitate does not disappear by stirring, the operation is finished, but a permanent precipitate is produced (= urea and oxide of mercury); (7.) The volume of the test-solution used is to be read off the burette, and the amount of *chloride of sodium* calculated therefrom; (8.) The chlorine may be estimated by the following formula: As 58.8 eqs. of chloride of sodium contain 35.5 eqs. of chlorine, the chlorine in the urine is obtained by the equation—

$$58.8 : 35.5 :: \text{amount of chloride of sodium in the urine} : x \text{ (the chlorine it contains).}$$

The average amount of chlorine excreted in twenty-four hours is about 8.21 grammes, or 126.76 grains; 13.6 grammes, or 210 grains, of chloride of sodium, were the chloride always united with that substance—an amount which Dr. Parkes thinks is too great. Vogel and Parkes consider the mean to be 7 grammes, or 108 grains, = 11.5 grammes, or 177 grains, of chloride of sodium in twenty-four hours; the range above and below the mean being from 30 to 60 per cent.

In relation to body-weight, the averages of chlorine are as represented in the second line of the table at p. 924, paragraph VI.

**Pathological Relations.**—The chlorine contained in the urine is wholly derived from the food; and a part, chiefly united with *sodium*, passes out of the system without having entered into the composition of the tissue; another portion, uniting with the tissues, is only set free on the disintegration of these tissues: and therefore, whatever exceeds the amount taken in by the food is derived from and represents change of tissue. In *ague* its elimination is increased during the cold and hot stages. In *acute pneumonia* their elimination is greatly lessened, often entirely absent, but reappearing in the urine eight or ten days after resolution has set in and excess of urea has passed away. It evidently accumulates in the inflamed portion of the lungs. Their amount is also reduced in *typhus and typhoid fever, febricula, scarlatina, erysipelas, puerperal fever, pleurisy, acute capillary bronchitis, acute pulmonary phthisis, acute rheumatism, cholera, acute and chronic Bright's disease*.

**Treatment.**—Elimination is to be effected by the skin, bowels, or urine, mainly by the action of *liquor potassæ*.

2. **The Estimation of Urea** in urine is based on the combination which forms between urea and oxide of mercury in neutral or alkaline solutions. The method was devised by Liebig. The precipitate which is formed is insoluble in water, or in weak alkaline solutions. The standard test-solutions are the same as in the preceding estimate; and the indicator which shows when all the urea has entered into combination with the mercury, and when the latter predominates, is a solution of carbonate of soda.



(1.) Take one volume of the baryta-solution (20 c. c.), and mix with two volumes of urine (40 c. c.);\* (2.) After filtration take 15 c. c. of the fluid (= 10 c. c. of urine) for each analysis, in small beakers; (3.) Bring the beaker under the burette containing the mercurial solution, which is to be added in small quantities so long as a distinct precipitate is seen to form, the mixture being stirred constantly; (4.) A plate of glass or porcelain is now to be sprinkled with a few drops of solution of carbonate of soda, and a drop of the mixture brought from time to time in contact with the drops of soda-solution by means of a glass rod. So long as the mixture of the two drops thus brought in contact remains white, free urea is still present in the mixture, and more of the test-solution must be added to the urine, till the contact of the drops with the soda-solution produces a yellow color, which is distinctly apparent; (5.) Record the quantity of mercurial test-solution used, and so calculate for the amount of urea contained in the 10 c. c. of urine, and hence in the total discharge for twenty-four hours; (6.) Repeat the analysis at least twice.

Another method has been recommended by Dr. Edmund W. Davy (*Phil. Mag.*, vol. vii, Fourth Series, p. 385). The process is based on the fact that *urea* is readily decomposed by *hypochlorite of soda*, when the *nitrogen* being evolved as a gas, the amount of urea is estimated from the amount of nitrogen gas produced by the decomposition. A strong glass tube is required, about fourteen inches long, closed at one end, and its open extremity ground smooth. The bore of the tube must not be larger than the thumb can conveniently cover—i. e., half an inch in diameter. It ought to be graduated into cubic inches, commencing from the closed end, and each cubic inch again subdivided into 10ths and 100ths.

The following are the details of the process:

(1.) Fill the tube more than one-third full of fluid mercury; (2.) Pour in carefully half a fluid drachm to one drachm of urine; (3.) Holding the tube in one hand near its open extremity, and having the thumb in readiness to cover the aperture, quickly fill it completely full, with a solution of hypochlorite of soda (taking care not to overflow the tube), and then instantly cover the opening tight with the thumb; (4.) Rapidly invert the tube once or twice to mix its contents; and (5.) Finally open the tube under mercury contained in a strong cup or small mortar; (6.) The tube is left in the upright position till the evolution of gas ceases, which it generally does in from three to four hours; (7.) Record the amount of gas found, and estimate the urea by the following data: 1.549 cubic inch of gas represents one grain of urea.

The hypochlorite of soda used should always be five or six times the volume of the urine operated upon; and the *liquor sodæ chlorinatæ* should be perfectly pure, prepared according to the process of the Dublin Pharmacopœia, as the *liquor* of commerce always gives erroneous and exaggerated results.

A third method for determining the urea consists in the use of the following table, which is founded on many observations of urine, both of health and disease, of specific gravities from 1003 to

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\* The precipitate may be reserved for determining the phosphoric acid.

		SPECIFIC GRAVITY.																										
Fluid Ounces.		1003	1004	1005	1006	1007	1008	1009	1010	1011	1012	1013	1014	1015	1016	1017	1018	1019	1020	1021	1022	1023	1024	1025	1026	1027	1028	
20	35	43	57	71	85	100	103	106	119	130	136	142	149	156	161	166	168	193	283	241	249	267	265	274	276	278	279	280
21	37	45	59	74	89	105	108	111	124	136	142	149	156	163	168	173	176	205	245	258	261	269	278	288	290	292	294	
22	38	47	62	77	95	110	113	116	130	143	149	156	163	170	175	180	184	216	257	265	274	282	292	301	303	305	306	
23	40	41	65	81	97	115	118	121	136	149	156	163	170	177	181	184	188	225	268	277	286	295	305	315	317	319	320	
24	42	43	68	85	101	120	123	127	142	156	163	170	177	184	188	192	196	235	280	289	299	308	319	329	331	333	334	
25	43	45	71	88	106	125	129	132	147	162	170	177	184	191	196	200	204	245	291	301	311	321	332	342	345	347	348	
26	45	47	73	92	110	130	134	137	153	169	176	184	191	198	203	208	213	254	303	313	324	334	346	356	359	360	362	
27	47	49	76	95	114	135	139	143	159	175	183	191	198	205	210	216	221	264	314	326	336	347	359	369	372	374	376	
28	48	50	79	99	118	140	144	148	165	182	190	198	205	212	218	224	229	274	326	337	349	360	372	383	386	388	390	
29	50	52	82	103	122	145	149	153	171	188	197	205	212	220	226	232	237	284	337	349	361	373	386	397	400	402	404	
30	52	54	85	106	127	150	155	159	177	195	204	212	219	227	233	240	246	294	349	361	374	386	399	411	414	416	418	
31	54	56	87	109	131	155	160	164	182	200	210	217	224	232	239	246	253	303	361	373	386	398	412	426	428	429	432	
32	55	57	89	111	135	160	165	169	188	208	217	224	232	240	247	254	261	313	373	385	398	411	426	438	442	443	446	
33	57	59	93	116	140	165	170	175	194	214	224	232	240	248	255	262	269	323	384	397	411	424	438	452	455	457	460	
34	58	61	96	120	144	170	175	180	200	221	231	241	249	256	263	270	277	333	396	409	423	437	451	468	469	471	474	
35	60	63	99	124	148	175	180	185	205	227	238	248	256	264	271	279	286	343	407	421	436	450	464	479	483	485	488	
36	61	64	102	127	153	180	185	191	212	234	244	255	263	271	278	286	294	352	419	433	448	462	477	493	497	499	502	
37	63	66	105	130	157	185	190	196	218	240	251	262	270	278	285	293	301	362	430	445	461	476	490	507	510	513	516	
38	65	68	108	134	161	190	195	201	224	247	258	269	277	285	293	301	309	372	442	457	473	488	503	520	524	527	530	
39	67	70	111	138	166	195	200	206	230	253	265	276	284	292	300	308	316	382	453	469	485	501	516	534	538	541	544	
40	69	72	114	142	170	200	206	212	235	260	272	284	291	300	308	316	324	392	465	482	498	514	530	548	552	555	558	
41	71	73	116	145	174	205	211	217	241	265	278	291	298	307	315	323	331	401	477	494	510	527	543	562	566	568	571	
42	74	76	119	148	178	210	216	222	247	271	285	298	305	314	322	330	338	411	489	506	523	540	557	575	580	582	585	
43	75	77	122	152	182	215	221	228	253	277	292	305	312	321	329	337	345	421	501	518	535	553	570	589	593	596	600	
44	76	79	125	156	186	220	226	233	259	283	299	312	319	328	336	344	352	431	512	530	548	566	584	603	607	610	613	
45	78	81	128	160	191	225	231	238	266	291	306	319	326	335	343	351	359	441	523	542	561	579	597	616	621	624	627	
46	80	82	130	163	195	230	236	243	271	296	312	326	333	342	350	358	366	450	536	554	573	592	611	630	635	638	641	
47	82	84	133	166	199	235	241	249	277	302	318	332	339	348	356	364	372	460	546	565	585	605	624	644	648	652	655	
48	84	86	136	170	203	240	246	254	283	308	324	340	347	356	364	372	380	470	556	575	595	615	637	657	662	665	668	
49	86	88	139	174	207	245	251	259	289	314	330	346	353	362	370	378	386	480	568	587	607	627	649	671	676	679	683	

60	87	90	106	142	176	212	250	267	266	296	326	340	355	377	400	490	581	602	623	644	656	685	690	694	697	700
61	88	92	108	144	181	216	256	262	270	301	331	346	362	386	406	499	593	614	635	656	678	699	704	708	710	714
62	90	94	110	147	183	220	260	267	276	307	338	353	369	393	416	509	605	626	648	669	692	712	718	721	724	728
63	92	96	112	150	188	225	266	272	281	313	344	360	376	400	424	519	616	638	660	682	705	726	731	735	738	742
64	94	98	114	153	192	229	270	277	286	319	351	367	383	408	432	529	628	650	673	695	718	740	745	749	752	756
65	95	99	117	156	195	233	275	283	292	325	358	374	390	415	440	539	639	662	685	708	732	753	759	763	768	770
66	96	100	119	160	199	238	280	288	297	331	364	380	397	423	448	548	651	674	698	720	745	767	772	776	780	784
67	98	102	121	162	202	242	285	293	303	337	371	387	404	430	456	558	662	686	710	733	758	781	786	790	794	798
68	100	104	123	165	206	246	290	298	308	343	377	394	411	438	464	568	674	698	723	746	772	794	800	804	808	812
69	102	106	125	168	209	251	295	303	314	349	384	401	418	445	472	578	685	710	735	759	785	808	814	818	822	826
70	104	108	128	171	213	255	300	309	319	355	391	408	426	453	480	588	697	722	748	772	798	822	828	832	836	840
71	106	109	130	173	216	259	305	314	324	360	397	414	433	460	488	597	708	734	760	784	811	836	842	846	850	854
72	108	110	132	176	220	263	310	319	329	366	404	421	440	468	496	607	719	746	772	797	824	849	856	859	864	868
73	109	112	134	179	223	267	315	324	335	372	410	428	447	475	504	617	730	758	785	810	838	863	869	873	878	882
74	110	114	136	182	227	271	320	329	340	378	417	435	454	483	512	627	742	770	797	823	851	877	883	887	892	896
75	112	116	138	185	230	276	325	335	345	384	423	442	461	490	520	637	754	782	810	836	864	890	897	901	905	910
76	114	118	140	187	234	280	330	340	351	390	431	448	468	498	528	646	766	794	822	849	877	904	911	915	920	924
77	115	120	142	190	237	284	335	345	356	396	437	455	475	505	535	656	778	806	835	862	891	918	925	929	934	938
78	116	122	144	193	240	288	340	350	361	402	443	462	482	513	544	666	790	818	847	875	904	931	939	943	948	952
79	118	124	146	196	244	292	345	355	367	408	449	469	489	520	552	676	802	830	860	888	917	945	953	957	962	966
80	120	126	149	199	248	297	350	361	372	414	456	476	497	528	560	686	814	843	872	901	930	959	966	971	976	980
81	121	127	151	201	251	301	355	365	377	419	462	482	504	536	568	695	828	856	884	913	943	973	980	984	990	994
82	122	128	153	204	255	305	360	371	382	425	469	489	511	544	576	705	838	867	896	926	956	986	994	998	1004	1008
83	124	130	155	207	268	310	365	375	388	431	475	495	518	551	584	716	849	879	909	939	969	1000	1007	1012	1018	1022
84	126	132	157	210	262	314	370	381	393	437	482	503	525	558	592	726	861	891	921	951	982	1014	1021	1026	1032	1036
85	128	134	159	213	266	318	375	386	398	443	488	510	532	565	600	736	872	903	934	964	995	1027	1035	1040	1046	1050
86	130	136	161	216	269	323	380	391	404	449	495	516	539	573	608	745	884	915	946	977	1008	1041	1049	1054	1060	1064
87	132	138	163	219	273	327	385	396	409	455	501	523	546	581	616	755	895	927	959	989	1021	1055	1062	1068	1074	1078
88	134	140	165	222	276	331	390	401	414	461	508	530	553	588	624	765	907	939	971	1002	1034	1068	1076	1082	1088	1092
89	136	142	167	225	280	336	396	406	420	467	514	537	560	596	632	775	918	941	984	1015	1047	1082	1090	1096	1102	1106
90	139	144	170	228	284	340	400	412	426	473	521	544	568	604	640	785	930	964	996	1028	1060	1096	1104	1110	1116	1120
	1008	1004	1005	1006	1007	1006	1009	1010	1011	1012	1013	1014	1015	1016	1017	1018	1019	1020	1021	1022	1023	1024	1025	1026	1027	1028



There are some cases, which stand by themselves, in which the excretion and elimination of urea is augmented, which were originally noticed by Prout, and which Willis subsequently described as cases of "*azoturia*." They have recently been noticed in an excellent paper by Dr. Sieveking in the pages of the *British Medical Journal* for May, 1865. They are characterized by a great excess of urea in the urine, without increase in the quantity of the urine, and apparently without febrile reaction, but with languor, weakness, and nervousness, associated sometimes with dyspepsia, intemperance, mental anxiety, or sexual excess. They are apt to pass into *diabetes mellitus* (PROUT).

3. **Estimation of Sulphuric Acid** requires the *chloride of barium* test-solution and Beale's filter. The details of the process are as follow :

(1.) Measure off 100 c. c. of urine into a Florence flask; (2.) Add a little hydrochloric acid (twenty or thirty drops); (3.) Apply heat through a sand-bath till the acidulated urine boils; (4.) Allow the chloride of barium test to flow very gradually into the urine; (5.) Remove the heat, and allow the precipitate to subside after each addition; (6.) Continue adding the test till the precipitation is complete; (7.) Use Beale's filter for ascertaining the end of the precipitation. When no precipitate is formed either by chloride of barium, or by sulphate of potassa or soda, the analysis is complete.

The mean of sulphuric acid excreted in twenty-four hours is 2.012 grammes, = 31.11 grains, with a range of 45 per cent. above or below. In relation to body-weight, the average excretion of sulphuric acid is as stated in the third line of the table at page 924, paragraph VI.

*Pathological Relations.*—Sulphuric acid originates in the urine from various articles of food and drink, in which it exists ready formed: from the sulphur of the food, by oxidation in its passage through the system; from oxidation of the sulphur of the tissues or from the oxidation of sulphur contained in substances such as *taurine* and *cystine*. Its elimination is increased in *febricular*, *typhoid fever*, *typhus*, *variola*, *pyæmia*, *milk fever*, *delirium tremens*, *acute pneumonia*, *rheumatic fever*, *chorea*.

4. **Estimation of Phosphoric Acid** is based on the fact, that when nitrate or acetate of uranium is added to a solution of tribasic phosphoric acid, containing acetate of ammonia and free acetic acid, the whole of the phosphoric acid is thrown down as double phosphate of uranium and ammonia, having a light-brown color and a composition represented by the formula  $2(\text{Ur}_2\text{O}_5), \text{NH}_4\text{O}, \text{PO}_5 + \text{Aq}$ . This volumetric method for the estimation of phosphoric acid was devised by Mr. Francis Sutton, of Norwich, independently of Neubauer and Pincus, who, independently of each other, also arrived at the same process; but Mr. Sutton states that Neubauer was the earliest discoverer of the method. The standard test-solution required is the nitrate of uranium, of which 1 c. c. represents 0.1 grain of phosphoric acid. A solution of ferrocyanide of potassium (yellow prussiate of potash), in the proportion of 1 to



20, is to be used as an indicator to stop the process, so soon as the reaction is complete.

The details of the process are as follow :

(1.) Take the precipitate produced by the baryta-solution in 40 c. c. of urine, as mentioned at page 924, paragraph VI, having been set aside after the fluid is filtered from it. (2.) Wash the precipitate once with cold water. (3.) Treat it, while still on the filter, with warm acetic acid, to dissolve all the phosphate of baryta, which passes through the filter, leaving the sulphate behind. (4.) Wash the filter with a small quantity of boiling water, so as to remove the last traces of phosphate. (5.) Add sufficient ammonia to the solution to neutralize the acetic acid, unless the quantity of the latter be large, when somewhat less than enough to neutralize may be added. Under any circumstances the liquid must be fully acidified with acetic acid before being tested as to its strength (*titrated*), and must contain a tolerable quantity of acetate of ammonia. (6.) Take a measured quantity of the urinary phosphate-solution (say 20 c. c.) in a beaker, and gently warm it, and bring it under the burette containing the uranium-solution. (7.) Portions of the uranium-solution are to be delivered in and constantly stirred, until, when a drop is taken out with a thin glass rod, and placed in the middle of a large drop of the solution of yellow prussiate of potash on a white plate, a faint but distinct chocolate-brown color is produced at the point of contact. A slight excess of uranium-solution is required to produce the brown color, which indicates that all the phosphoric acid contained in the amount of urine in the beaker has been thrown down. (8.) Record the amount of uranium-solution used, and estimate accordingly—1 c. c. of the solution precipitating 0.1 grain of phosphoric acid.\*

The mean amount of phosphoric acid excreted in twenty-four hours is 3.164 grammes, = 48.80 grains, with a range in the same person of from 35 to 50 per cent. The amount in relation to body-weight is given in the fourth line of the table at page 924, paragraph VI.

*Pathological Relations.*—Its elimination in excess represents tissue-change or waste, beyond what the food or drink accounts for. In rickets and softening of the bones the phosphates are increased. In other diseases our information respecting phosphoric acid is very uncertain. In alkaline urine the *earthy* phosphates are deposited as a white sediment. Mineral acids, anodynes, alkaline bicarbonates, opium, and preparations of iron, zinc, and strychnia, are often of service, variously combined, according to the nature of the case (see HASSALL, p. 237).

5. **The Estimation of Uric Acid.**—An improved method of estimating the uric acid by iodine has been recently devised by my friend Dr. De Chaumont, and published by him in the *Medical Report of the Army Medical Department* for 1862 (published in 1864). The determination of uric acid by iodine is founded on the fact that solution of iodine is decolorized by uric acid, in a definite proportion—which

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\* Collect all the precipitates in a large bottle, and when sufficient has been obtained, recover the uranium by igniting the dry precipitate in a porcelain crucible, with the carbonaceous residue produced by burning tartrate of soda and potash in a covered crucible. The uranium is thus reduced to protoxide, while the phosphoric acid unites with potash and soda, and can be entirely extracted with boiling water. The protoxide of uranium left may be dissolved in nitric acid, and evaporated to dryness in a water-bath (Sutton, *Volumetric Analysis*, p. 208).

appears to be four equivalents to one of uric acid. Dr. De Chaumont has kindly given me the following short notice of the method:

*The Solutions* required are as follow:

(1.) A standard solution of iodine. The alcoholic tincture being liable to change, it is best to dissolve the iodine in water with the aid of iodide of potassium; thus, 6.35 grammes ( $= \frac{1}{20}$ th of an equivalent) of iodine, with 12 grammes of iodide of potassium, are dissolved in a litre of distilled water. Of this solution, 1 c. c. contains .00635 of iodine, and will decompose .0021 of uric acid. If the materials be accurately weighed and measured, it will be unnecessary to graduate the solution; but, should this be desired, then the following solutions must be prepared: (2.) Solution of uric acid, .168 grammes are to be dissolved in a litre of water. This is best made by dissolving the uric acid in a small quantity of *liquor potassæ*, and neutralizing the excess with dilute acetic acid. Of this solution, 12.5 c. c. will completely decolorize 1 c. c. of the solution of iodine. (3.) A solution of starch, carefully filtered and free from suspended grains.

*The Process.*—(1.) Filter the urine from mucus. (2.) Acidify the urine, should it be alkaline, with acetic acid. (3.) Put 10 c. c. into a beaker-glass, and dilute it to 50 c. c. with distilled water. (4.) Add 5–10 c. c. of starch liquor. (5.) Drop in the solution of iodine from a burette graduated to  $\frac{1}{10}$ th c. c. (6.) Stir each time, and wait till blue color disappears. (7.) Do not add more than  $\frac{1}{10}$ th c. c. at a time. (8.) When the blue color has remained permanent for *an hour*, read off the number of c. c. of iodine used, and multiply by 21, which will give the quantity per litre.

*Example.*—10 c. c. of urine took 3.5 c. c. of iodine,  
 $3.5 \times .21 = .735$  grammes per litre.

For a more accurate analysis, it is necessary to wait for twenty-four hours until all the uric acid is decomposed, taking care to add fresh portions of starch from time to time as it becomes converted into dextrine. A correction is then applied as follows: (1.) Precipitate the salts with Liebig's baryta-solution (see process for urea). (2.) Filter and acidify with acetic acid. (3.) Test with iodine as before. (4.) Deduct the number of c. c. used after the baryta from the gross amount first used, and from the remainder calculate the uric acid as before.

*Example.*—10 c. c. of urine took 3.5 c. c. of iodine.

10 c. c. so precipitated, filtered, and re-acidified, took .45 c. c. of iodine.

Then  $3.5 - .45 = 3.05$  c. c. of iodine.

$3.05 \times .21 = .6405$  grammes in a litre—the net result.

The mean amount of uric acid excreted in twenty-four hours appears to be .555 grammes,  $= 8.569$  grains. The observations to determine the average in any individual must extend over at least *five days*. The range between the maximum and minimum amount is as great as from 20 to 30 per cent. (PARKES).

*Pathological Relations.*—Its elimination indicates metamorphoses of tissue. It increases after full, heavy, indigestible meals; and

exists not alone in the spleen in health, but in still larger quantity in most of the affections, including *ague*, in which the spleen is more especially implicated. It is increased in most of the active febrile and inflammatory affections. It is greatly increased during the paroxysm of *intermittent* fevers, also in *typhoid* and *typhus* fevers. In *small-pox* and *scarlatina* it is increased; and in *pneumonia* the increase is very large. The sediments of the *urates* and of *uric acid* are most abundant from the seventh to the thirteenth day. It is increased in most cardiac affections, in hepatic cirrhosis and leucocythæmia; and is considerably increased in rheumatic fever. During attacks of gout it is lessened or absent in the urine, and present in the blood; but as soon as fever and paroxysms abate, precipitates of *urates* and of *uric acid* become abundant. Its free elimination is a favorable symptom.

In the majority of chronic affections the uric acid is lessened in elimination. When the excess in the urine is due to indulgence in animal food, its mal-assimilation and defective cutaneous excretion, the amount of animal food must be reduced, the diet regulated carefully, and the functions of the digestive organs improved and strengthened. When *lactic*, *acetic*, or *butyric* acids occur as products of faulty digestion, all food known to produce such a result must be avoided. The vegetable bitters—e. g., *cinchona*, *gentian*, *calumba*, *serpentaria*, with or without *rhubarb* and *soda*, and followed by such remedies as keep the colon free, such as *compound galbanum pill*, with *extract of colocynth* and *croton oil*, will be found of great service. The action of the skin must be promoted by warm clothing. Exercise in the open air must be indulged in as much as possible; while cold baths in the morning, and friction with horse-hair gloves aid the action of the skin. The best solvent is obtained by drinking freely of soft or distilled water; but alkaline remedies like the carbonates and bicarbonates, often give relief, as well as the salts of the vegetable acids, the *acetates*, *citrates*, and *bitartrates*, given in sufficient doses. The occurrence of *uric acid* in excess in the diseases mentioned must be managed according to the nature of the treatment of each disease.

**6. Estimation of Diabetic Sugar.**—The principle of the process is thus explained by Mr. Sutton. It is based on the fact that although a mixture of pure sulphate of copper, tartrate of potash, and caustic soda, mixed in proper proportions, may be boiled without undergoing change; yet, if only a trace of sugar be added, a very slight warming is enough to precipitate a portion of the copper as a protoxide ( $\text{Cu}_2\text{O}$ ). It is found that one atom of pure sugar, = 180, is capable of reducing exactly 10 atoms, = 307, of oxide of copper ( $\text{Cu O}$ ) to the state of protoxide. Therefore, if the quantity of copper reduced by a given solution of sugar is known, it is easy to find the quantity of sugar present (*Volumetric Analysis*, p. 210).

A standard solution of pure sulphate of copper with tartrate of potash and caustic soda is required. It is to be prepared as follows:

(1.) 34.64 grammes, = 346.4 grains, of pure sulphate of copper, previously powdered and pressed between blotting-paper, are weighed and dissolved in 200 c. c. of distilled water; (2.) In another vessel, 173

grammes, = 1730 grains, of pure crystallized tartrate of soda and potash (Rochelle salt) are dissolved in 480 c. c. of solution of pure caustic soda (specific gravity, 1.14); (3.) The two solutions are then to be mixed, and the deep clear blue solution diluted with distilled water till the whole measures 1000 c. c. One c. c. of the solution so prepared represents 0.05 grains of grape or diabetic sugar. It must be preserved for use in a dark place, and in well-stoppered bottles kept full. It should bear heating when diluted with about four or five times its quantity of distilled water, without any precipitate taking place; and should always be submitted to this test before being used. If any precipitate does occur, it probably arises from the alkali having absorbed carbonic acid; and in this case the addition of a little fresh caustic soda-solution remedies the evil (SUTTON, *l. c.*, pp. 211, 233).

[This method is best known as Fehling's, and depends in principle on the fact that there is a fixed relation between the amount of a copper salt, reduced to a state of suboxide, and the sugar present. He found that one equivalent of grape sugar, or 180 parts, decomposed exactly ten equivalents, or 1246.8 parts, of sulphate of copper. The following is a correct and an intelligible formula for the preparation of Fehling's standard solution:

Sulphate of copper, 90½ grains; neutral tartrate of potash, 364 grains; solution of caustic soda, sp. grav., 1.12, four fluid ounces. Add water sufficient to make exactly six fluid ounces. 200 grains of this solution are exactly decomposed by one grain of sugar.]

The following are the details of the process for analysis:

(1.) 10 c. c. of clear urine are diluted by means of a measuring flask to 200 c. c. with water, and a large burette filled with the fluid; (2.) 10 c. c. of the copper solution (= ½ grain of sugar) are then measured into a flask, or white porcelain capsule, and 40 c. c. of distilled water added; (3.) The vessel is to be arranged over a spirit-lamp under the burette, and brought to boiling; (4.) The diluted urine is then delivered in cautiously from the burette until the last traces of blue color are removed from the copper solution, and the precipitate is of a distinct red color. (5.) It must be remembered that the urine has been diluted twenty times; so that the quantity used, divided by twenty, will represent the amount of the original urine used; and the estimate is to be made accordingly.\*

[The simplest, quickest, and most accurate method of determining the quantity of grape-sugar, is that proposed by Dr. William Roberts, of Manchester, and called by him the *differential density method*, founded on the diminution of density suffered by saccharine matter when fermented with yeast; experiments establishing the conclusion that the number of degrees of "density lost," indicated as many grains of sugar per fluid ounce (*Memoirs of the Manchester Literary and Philosophical Society*, 1860; *Edinburgh Medical Journal*, Oct., 1861; *A Practical Treatise on Urinary and Renal Diseases*, American reprint, 1860, p. 148).

In the practical application of the method, the ordinary urinometer may be used for taking the densities, so that it has a long scale. Greater pre-

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\* Haughton recommends the following formula: "Multiply the excess of specific gravity over 1000 by 20,—the result is grains of sugar in a pint of urine." The average of 234 determinations by Dr. De Chaumont gave 61.06 parts in 1000; the average by Haughton's formula gave 77.65;—error + 27. per cent. Dr. De Chaumont proposes to divide excess of specific gravity over 1000 by .54—result = parts in 1000. Haughton's factor, = + .4848, or  $\times 2.8$ . Dr. De Chaumont's factor, = + .54, or  $\times 1.89$ .

cision may be had by dividing the scale into two parts on separate instruments. Errors from variations of temperature are to be avoided, which is best done by putting up a few ounces of the unfermented urine in a companion vial, and to place this by the side of that set apart for fermentation, so that whatever the temperature of the fermented product may be when its density is taken, its unchanged *alter ego* stands near it for comparison at exactly the same temperature. The most convenient way of proceeding, is the following: Four ounces of saccharine urine are put into a 12 ounce bottle, and a lump of German yeast, about the size of a small walnut, is added. A great excess of yeast is used to hasten fermentation, but the result is not affected by a little more or less. The bottle is then stopped with a nicked cork (to permit the escape of carbonic acid), and set aside in a warm place to ferment. Beside it is placed a tightly corked 4 ounce vial, filled from the same urine, without any yeast. In about from eighteen to twenty-four hours, the fermentation will have ceased, and the scum have cleared off or subsided. The two vials should then be put in a cool place, and kept there for two or three hours to get the temperature of the atmosphere of the room. The fermented urine is then poured into a urine-glass, and its specific gravity noted; at the same time the density of the unfermented urine in the companion glass is taken, and the “density lost” ascertained. The two following examples will serve as illustrations of the method:

	I.	II.
Density before fermentation, . . . . .	1058	1038
Density after fermentation, . . . . .	1004	1018
Degrees of density lost, . . . . .	49	25
Grains of sugar per fluidounce, . . . . .	49	25

If it be desired to bring out the result at so much per cent., it can be done by multiplying the number indicating the “density lost” by the coefficient 0.23. Thus, in the first above examples,  $49 \times 0.23 = 11.27$ ; and in the second,  $25 \times 0.23 = 5.69$ , which are the amounts of sugar respectively per 100 parts.

The time actually necessary to determine the quantity of sugar in urine by this method need not exceed four or five minutes, but the result must be waited for until the succeeding day.]

7. **Estimation of Free Acid** is measured by a solution of carbonate of soda containing 530 grains in the 10,000 grain measure, = 53 grammes in the litre; and is represented by determining how many grains of crystallized oxalic acid a certain quantity of the soda-solution will neutralize. The details of the process are as follow :

- (1.) Take 50 or 100 c. c. of perfectly fresh urine;
- (2.) Add from a burette a standard solution of soda, in small portions at a time (say 5 c. c., or drop by drop);
- (3.) After every addition test the fluid by moistening a thin glass rod or feather with the mixture, and streak it across some well-prepared violet litmus paper; when the streaks cease to become red, the analysis is complete;
- (4.) Estimate how much of the standard solu-



tion has been used, and express the acidity as equal to so many grains of crystallized oxalic acid.

8. **Estimation of the Total Solid Matter.**—(1.) Measure 5 c. c. into a shallow platinum or porcelain capsule; (2.) Place it beside a vessel of strong sulphuric acid under the receiver of a powerful air-pump, and keep it *in vacuo* till all moisture is removed.

9. **Estimation of Total Saline Matter.**—(1.) Measure 10 c. c. into a small porcelain crucible; (2.) Evaporate to dryness; (3.) Add about ten drops of nitric acid; (4.) Heat the crucible to dull redness; (5.) Suffer it to cool, and add ten more drops of nitric acid; (6.) Heat it up again gradually to a moderately strong heat, until all the carbon is destroyed and the residue is white; (7.) Let it cool, and weigh.

10. **The Specific Gravity of Urine** is best taken by measuring 100 c. c. into a beaker or flask whose weight is accurately known. The increase of weight in grains will be the specific gravity, water being 1000. Instead of 100 c. c., 50 or 25 c. c. may be taken, when the weight, multiplied by 2 or 4, will be the specific gravity (SUTTON). The urinometer gives the least accurate of all results.

For further details regarding the processes of volumetric analysis the reader is referred to the excellent little treatise of Mr. Francis Sutton, already noticed.

## SECTION VIII.—THE MICROSCOPIC EXAMINATION OF THE URINE, AND THE PATHOLOGICAL RELATIONS OF THE DEPOSITS.

In perfectly healthy urine there ought to be no sediment whatever, unless it be the very merest haze of mucus, or the slightest precipitate of urates caused by a low temperature. Even these may be abnormal (PARKES).

The sediments have been broadly arranged by Dr. Parkes into the following three classes (*l. c.*):

### CLASS I.—SUBSTANCES SUSPENDED IN THE URINE WHICH HAVE NEVER BEEN DISSOLVED.

They commence to precipitate as soon as the urine is passed. The most important sediments belong to this class, and consist chiefly of organic bodies derived from the structures composing the urinary organs, or of the *productive* effects of disease upon the kidney, such as *inflammation*, tubercle, cancer. They often afford the only signs of kidney disease, or of the implication of the kidney in some general affection. They are made up of the following substances:

1. *Mucus and Epithelium from the Urinary Passages.*—In many diseases the quantity of epithelium from the bladder is increased, indicating catarrhal inflammation of the mucous membranes. The epithelial cells are of various sizes and stages of formation (Fig. 69); and frequently free nuclei are seen. In catarrh of the bladder the mucus, from its cohesion, is apt to form thin transparent flakes or cylinders, resembling casts from the prostate or kidney.

The epithelium-cells from the pelvis of the kidney are often triangular or caudate, with well-defined nuclei. They generally ad-

FIG. 69.\*



here together in groups of three to ten, when they appear to have an imbricated arrangement, and perhaps are more closely connected than natural, by adhesive mucus (Fig. 70). They are never found in healthy urine, but are present very commonly in catarrhal and calculous cystitis. Tailed or caudate cells are also sometimes present with pelvic epithelium.

FIG. 70.†

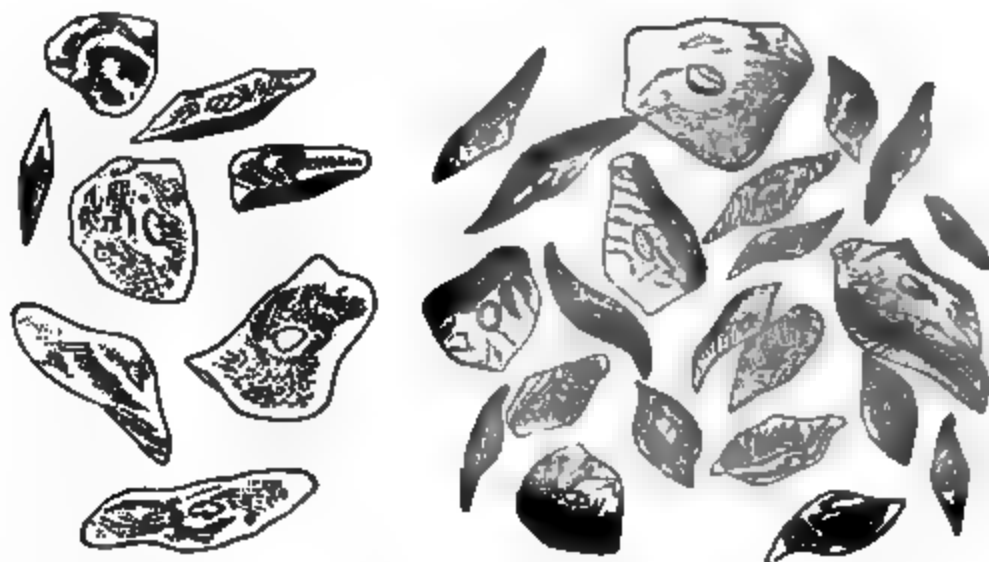


FIG. 71.‡



The epithelium from the ureter is columnar in its character (Fig. 71), and not unlike that found in the male urethra, which is mostly columnar, and is more flattened than that of the bladder, and less regular than that of the pelvis. Mixed

FIG. 72.§



\* Epithelium from the bladder. Many of the large cells lie upon the summit of the columnar and caudate cells, and depressions are seen on their under surface. One is seen near the centre of the figure (after DR. BEALE, *On the Urine*, p. 259).

† Epithelium from the pelvis of the kidney (after DR. BEALE, *l. c.*, p. 188).

‡ Epithelium from the ureter (after DR. BEALE, *l. c.*)

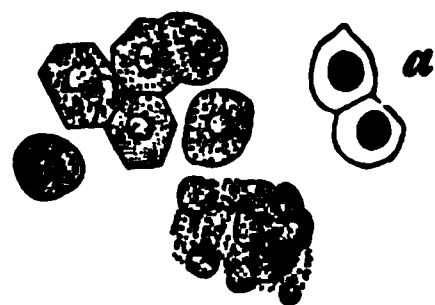
§ Vaginal epithelium from urine (after DR. BEALE, *l. c.*, p. 260).

with it there is a good deal of scaly epithelium, especially towards the orifice of the urethra.

Large cells of scaly epithelium are also often met with in the urine of females. They are derived from the vagina. They vary much in size and form, and are sometimes very irregular in shape, with uneven ragged edges (Fig. 72.)

Renal epithelium is only found in disease. It consists of round or slightly compressed cells, or masses of material, with well-defined central portions or nuclei, which are not cleft like the pus nucleus under the action of acetic acid, but become at first more defined and afterwards paler and smaller. In the urine they are less polygonal and more rounded than they are in the renal canals (Fig. 73). Their presence indicates more or less desquamation from the tubes; while their morbid condition and admixture with other products may indicate still greater disease. Thus it is sometimes fatty; the whole space between the nucleus and the cell-wall being filled with fatty globules; but these changes and their value have been fully considered under chronic Bright's disease, p. 153, *et seq.*

FIG. 73.\*



2. *Other Cell-Forms occur in the Urine, the productive results of inflammatory diseases* of the mucous membrane of the urinary passages. They are chiefly granules, singly or in clusters, and pus. The pus is known by its nucleus becoming cleft into two, three, or five divisions under the action of acetic acid. The amount of pus is generally far greater in *cystitis* than it is in *pyelitis* or *nephritis*; and wherever there is a markedly large quantity of pus it is generally from the bladder, unless there be a prostatic or other abscess communicating with the urethra.

3. *Cancer-Cells* occur in cancerous disease of the bladder, kidney, or urethra.

4. *Tubercle-Masses* may occur in tuberculous disease of the bladder or kidney.

5. *Cylinders* occur in the urine, and have various modes of origin: (a.) From the *bladder*, as long, flat, membraniform, twisted, or folded bodies. (b.) From the *prostate*, as coagula, two or three times as broad as renal cylinders. They are soluble in acetic acid. Amylaceous corpuscles may exist in them. (c.) From the *ureter* and *pelvis* of the kidney the coagula are cylindrical, pyriform, or globular. (d.) From the kidney-tubes cylinders or renal casts are formed in various diseases. They vary in breadth from  $\frac{1}{800}$ th to  $\frac{1}{1000}$ th of an inch—*i. e.*, from about the breadth of the straight renal tubes to half or a third of that size. Their length varies from  $\frac{1}{200}$ th to  $\frac{1}{50}$ th of an inch. The terms used by Dr. Geo. Johnson, to whose researches on diseases of the kidney we owe much, express very well the special character of the several varieties of casts. They are—(1.) Epithelial casts; (2.) Large waxy casts; (3.) Small waxy cases—the

\* Epithelium from convoluted portion of uriniferous tube.—(a.) Treated with acetic acid  $\times 215$  (after Dr. BEALE, *l. c.*, p. 187).

hyaline casts of Vogel and Basham ; (4.) Granular casts ; (5.) Oily casts ; (6.) Bloody casts ; (7.) Purulent casts.

Solution of iodine is the best reagent for making the waxy hyaline or transparent casts visible. The chemical composition of these casts is unknown. They are probably albuminous.

6. *Kidney-Structures* may occur in the urine if the structure of the kidney has commenced to break up.

7. *Blood-Corpuscles* indicate ruptures of vessels somewhere in the urinary passages. Copious hemorrhage is usually from the bladder, carcinoma of the kidney, or calculus of the pelvis. The corpuscles are often very much modified in form.

8. *Fibrine* is present in many cases of bloody urine.

9. *Corpora amylacea* may occur from the prostate gland.

10. Fat. 11. A fatty substance to which the name of uro-stealith has been given. 12. *Spermatozoa*. 13. *Sarcinæ*. 14. Hair. 15. Various *entozoa* have been occasionally found in the urine. (See under "Entozoa.")

## CLASS II.—SEDIMENT FORMING IN THE URINE AFTER SECRETION, BUT WHICH MAY DEPOSIT IN THE RENAL PASSAGES OR AFTER EMISSION, EITHER IN CONSEQUENCE OF CHEMICAL CHANGES OR FROM CHANGE OF TEMPERATURE.

1. *Uric Acid* in various combinations with bases, such as *soda*, *potash*, *lime*, or *ammonia*, and more or less colored with urine pigment. They are associated sometimes with phosphate or oxalate of lime. The uric acid sediments in combination with bases are generally associated with increased acidity of the urine, either by normal acids, such as uric, sulphuric, phosphoric, hippuric, or with the formation of acids developed after emission. They are known as "urates," and commonly spoken of as yellow, lateritious, "brick-dust," or "fever sediments." They dissolve when the urine is heated to 130° Fahr. They are also soluble in potash and liquor ammonia. Acids decompose them after warmth and liberate uric acid. Three forms are distinguishable—(1.) Irregularly formed or amorphous particles ; (2.) Round globules, of various sizes ; (3.) Fine acicular prismatic crystals. These forms may deposit at two different periods after emission—(1.) As soon as the urine has cooled down to the temperature of the atmosphere. In such cases the water is diminished—the urine being that known as febrile urine ; or it occurs when there is an absolute increase of uric acid. (2.) They deposit some hours after the urine has been passed, and long after the urine has been of equal temperature with the air. Under these circumstances the deposit betokens increased acidity from changes in the pigment or extractives, or uroxanthin. The acids so formed may be lactic, acetic, or butyric ; and a drop of acid added to such urine will anticipate the deposit.

2. *Uric Acid Sediments* in their pure state occur in the form of rhombic prisms, or rhombic plates, or of thin hexagonal plates like cystin ; but the most usual forms are referable to some variety of

the rhomb (Fig. 74). Such deposits often present the aspect of a granular sand of a golden lustre, sometimes mingled with blood-discs; and generally the deeper the color of the urine the darker is the uric acid sediment. Its appearance does not necessarily indicate that an excess of uric acid is forming in the body. The urine is generally yellow and transparent, and the acid is deposited slowly without admixture of urates. Liquor potassæ, and also nitric acid in excess, dissolve uric acid.

FIG. 74.\*



### 3. *Sediments of Hippuric Acid.*—

Owing to the solubility of this acid its sediments are rare. When it does occur, it is in the shape of long four-sided, acuminate prisms, or acicular needles fixed on uric acid crystals, with which they are sometimes confounded, as well as with phosphates. They are distinguished from phosphates by being insoluble in acids; and from uric acid they may be separated by boiling with strong alcohol (PARKES).

4. *Sediments containing Phosphoric Acid* are formed of the *ammoniaco-magnesian phosphate*, the *phosphate of lime*, and the *phosphate of magnesia*. The ammoniaco-magnesian phosphate occurs in the form of beautiful transparent prisms, or in foliaceous, penniform, or stellar prisms or crystals.

The *phosphate of lime* and the *phosphate of magnesia* form a white amorphous powder, or they occur as round small globules, or as prismatic crystals with oblique summits (HASSALL).

Acids dissolve all those sediments; heat has no effect upon them.

The *ammoniaco-magnesian phosphate* is almost always deposited as a result of the decomposition of urea, and the deposit usually commences on the surface of the fluid, where the urea is most exposed to the air.

The *phosphate of lime* and the *phosphate of ammonia* are thrown down if the urine becomes alkaline from fixed alkali; as after vegetable food or the carbonates of

FIG. 75.†



\* The most usual forms of uric acid sediment, with blood-corpuscles intermixed (after Dr. OTTO FUNKE).

† Ammoniaco-magnesian phosphate in prisms, mixed with amorphous granules, phosphate of lime, and granular urates (after Dr. THUDICHUM).



the fixed alkali, or after the salts which form them have been taken. Decomposition of the urea in these cases is generally rapid, and *ammoniaco-magnesian* phosphate is also produced. Such, also, is the case with urine of chronic diseases, or during convalescence from acute diseases, when the urine is feebly acid. The phosphates and urates occur together under the following conditions; (1.) When urates having been deposited, the urea decomposes to a slight extent—enough to form the ammoniaco-phosphate, and yet not enough to dissolve the urate; (2.) When crystallized uric acid has formed and been acted upon by the ammonia formed from the urea, the crystals of uric acid disappear, and are replaced by round globules of *urate of ammonia*, mixed with the precipitate of phosphates. These deposits are always white, and unchanged by heat; soluble in dilute hydrochloric acid, but insoluble in ammonia and in liquor potassæ. Mucus, pus, or blood may mask the chemical reactions. A small quantity of solution of sesquicarbonate of ammonia, added to a large quantity of healthy urine, will yield prisms of the triple phosphate.

5. *Sediments composed of Oxalate of Lime*.—Opinion is very undecided as to the significance of this deposit. It occurs in one out of every three cases, taking the cases indifferently as they occur in an hospital. It appears (Fig. 76) in four forms: (1.) As octahedra

FIG. 76.\*

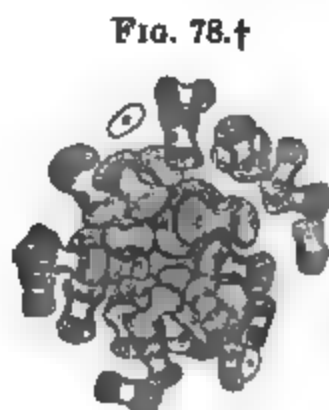


(a); (2.) As hour-glass, contracted, or dumb-bell-like bodies (b); and (3.) Compound octahedra (c) may also be seen; (4.) As small, flattened, bright discs, very readily mistaken for blood-discs.

Oxalate of lime, although found in the blood, is probably a result of chemical changes in the renal passages. The octahedra grow or increase in urine after it is passed; but the dumb-bell crystals are said not to do so. Dr. Beale considers that the dumb-bell crystals of oxalate of lime are mainly formed in the kidney-tubes. He found them most frequently in casts. Dr. Parkes considers that

\* (a.) Octahedra; (b.) Dumb-bell; (c.) Compound octahedra; circular and oval crystals of oxalate of lime (after Dr. BEALE, *l. c.*)

the oxalic acid of urine most probably results from uric acid; and may be a substitution for the excretion of carbonic acid of the lungs (SCHMIDT). In order that oxalic acid shall form in the urine, there must either be irritation from a calculus, or from some other cause, or there must be fermentive changes from mucus. If there be no evidence of any of these conditions, the oxalic acid of fresh urine may be presumed to come from the blood (PARKES). Crystals of oxalate of lime are insoluble in water—are unaltered by boiling either in *acetic acid* or in *liquor potassæ*; and are soluble in nitric acid without effervescence.



*Pathological Relations.*—Many articles of diet contain oxalic acid in abundance; but there are also certain articles of food and drink the use of which, when digestion is deranged, is followed by the formation of oxalate of lime and its presence in the urine. Sugar in excess, the use of frothy sparkling beer or wine, turnips, parsnips, carrots, cauliflower, asparagus, may all cause a "temporary oxaluria" (PROUT, ROBIN, and VERDEIL, ROSE, BIRD, HASSALL). When the respiratory functions are impeded, oxalates appear in the urine, and during convalescence from some severe diseases, as *typhus*.

The mineral acids are useful in correcting the dyspepsia associated with the elimination of oxalates. Water containing lime should be avoided, and distilled water used instead; and in many cases it will be necessary to empty the intestines by suitable purgatives.

6. *Sediments of Carbonate of Lime* are occasionally associated, in the form of dumb-bells, with the *ammoniaco-magnesian phosphate*.

7. *Sediments of Leucine* are precipitated in round corpuscles, sometimes with a concentric form, and look like heaps of fat; but when crystallized from pure solutions, it appears as fine, dark-colored needle-like crystals. To recognize it fully, it must be separated by careful sublimation (PARKES). The suspected leucine is to be placed on platinum, carefully moistened, and then dried with nitric acid. The almost imperceptible flake which is left is to be moistened with caustic soda, and evaporated carefully over a spirit-lamp. If leucine is present, it forms an oily-looking drop (SCHEBER).

*Pathological Relations.*—It is believed to be the result of the disintegration of certain of the nitrogenous tissues of the principal glands, and occurs particularly in *acute yellow atrophy* of the liver, where it is found in the substance of the liver itself.

8. *Sediments of Tyrosine* are of a greenish-yellow color, composed

\* Dumb-bell crystals of oxalate of lime, of very regular form, from the urine of a child two years of age, suffering from jaundice (after Dr. BEALE, *l. c.*, p. 800).

† Collection of dumb-bells, such as often forms the nucleus of a calculus (after Dr. BEALE, *l. c.*, p. 800).

of heaps of fine needles, which can be obtained on evaporation. It ought to be treated with nitric acid, like *leucine*, and then a little *liquor soda* used. The nitric acid gives a deep orange-yellow color, which becomes deep yellow on evaporation. The soda gives the

yellow flake a red tinge; and on heat and evaporation, a black-brown residue is left (SCHERER, quoted by PARKES).

*Pathological Relations.*—It is found wherever leucine is met with.

9. *Sediments of Cystin* form a white or light fawn-colored, amorphous, rather bulky precipitate, or they appear at once as six-sided plates (Fig. 79). In both cases ammonia dissolves it,—so do fixed alkalies and their carbonates; and from this solution it crystallizes on spontaneous evaporation. It does not disappear when the urine

is gently warmed; and it is insoluble in carbonate of ammonia, in dilute hydrochloric acid, and in acetic acid.

*Pathological Relations.*—It is associated with the excessive elimination of *sulphur*. Its persistence in the urine is often hereditary, and is generally associated with derangement of the functions, or organic disease of the liver. It occurs also in cases of chlorosis.

### CLASS III.—SEDIMENTS COMPOSED OF SUBSTANCES FOREIGN TO THE URINE, AND WHICH ACCUMULATE IN THE URINE ALWAYS AFTER EXPOSURE TO THE ATMOSPHERE.

1. *Fungi* in acid urine—*e. g.*, the *Penicilium glaucum*, its spores, thallus, and fructification.

2. The *Torulæ cerevisiæ* in saccharine urine begins to form in two or three hours after emission. It forms a gelatinous mass, composed of *sporules*, which subsequently develop into *beaded threads*; and in a few days aerial fructification appears.

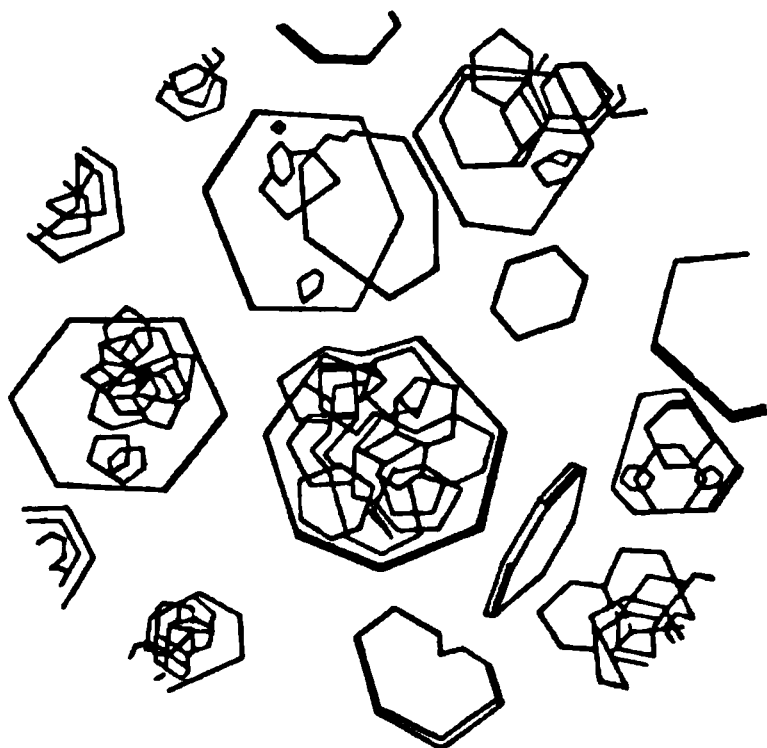
3. *Vibriones* and *Monads* occur in urine containing much mucus, and which is feebly acid or alkaline.

All urine should be collected in glass vessels, which are scrupulously clean; and the quantity operated upon should embrace the whole urine passed during the twenty-four hours. All sediments should be examined—(1.) Within a few hours after excretion; (2.) After twenty-four hours.

Of the substances dissolved in the urine, or becoming obvious only under special conditions, there are some which require more special notice, namely:

\* Cystin precipitated by acetic acid from its ammoniacal solution (after THURMICHUM).

FIG. 79.\*



1. *Hæmatine, or Blood-pigment*, occurs either as blood-corpuscles or separated from them. The corpuscles fall to the bottom of the tube or glass, and form a sediment. If the hæmatine is separate from the corpuscles, it constitutes a pigment completely dissolved in the urine, and to which it gives a brown or black color (see scale of color). It is always accompanied by albumen. Urine of a brown or black color, due to blood-pigment dissolved in the urine, is to be determined by the elimination process in part—bile and vegetable pigments being absent.

Urine containing blood in obvious quantity permits it generally to coagulate into blackish masses like pieces of black currant jelly; and when it partly coagulates in the bladder, linear masses of clot, of nearly the shape of leeches, are passed from the urethra. The urine has a port-wine hue, and abundance of entire red discs of blood-corpuscles may be detected with the microscope. These sink with readiness to the bottom of the vessel. Their non-granular surface, uniform size, and yellow color, are characters sufficient to identify them.

Blood-pigment, or hæmatine, dissolved in the urine, does not necessarily indicate local disease or rupture of vessels in part of the urinary organs. It must be regarded as indicating rather a specially morbid condition of the blood, as is associated with septic poisons, or with profound cachectic diseases (PARKES). It may be observed in *typhus fever, malignant variola, remittent fever, yellow fever, scurvy, and Bright's disease*. Albumen is said always to co-exist.

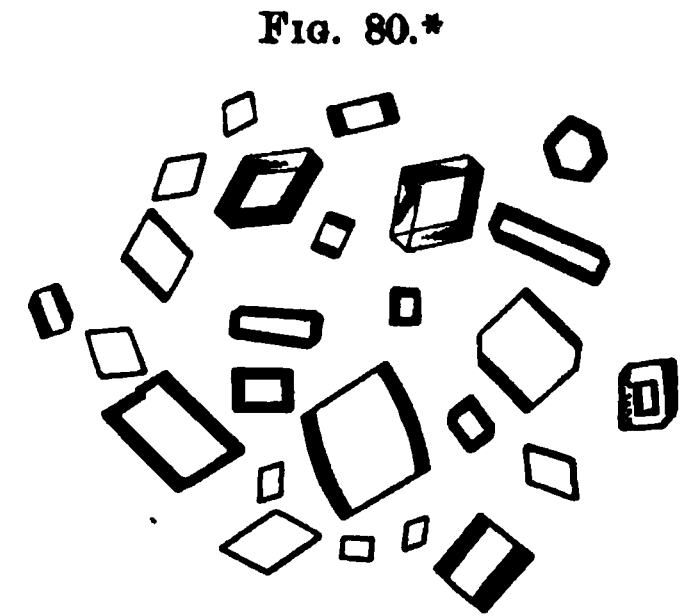
2. *Albumen*.—Its presence must be regarded as an indication of disease, although it has been observed to occur in small quantity, and temporarily, in the urine of some persons supposed to be at the time in a healthy state; although it is more than probable that slight disease is present, or that such grave affections as Bright's disease may be commencing, to terminate fatally perhaps two years later (Christison, *Monthly Journal*, Jan., 1851; PARKES, p. 184). Very slight disease—not more than what appears to be dyspepsia—may have albuminuria associated with it, of a temporary nature. It has been observed in *intermittent fever, typhoid fever, rubeola, variola, pneumonia, pleuro-pneumonia, scarlatina, bronchitis, pleurisy, peritonitis, intestinal catarrh, follicular enteritis, acute rheumatism, chlorosis, cardiac diseases, phthisis* (SOLON, FINGER, PARKES).

Hæmaturia has been found associated with albuminuria in *scarlatina, rheumatism, arthritis, typhus* (during the second week), *nephritis (calculous?) pneumonia, erysipelas, torpor, hepatitis, peritonitis, phthisis, chlorosis, intense oxaluria, phosphatic diathesis, last month of pregnancy, uterine disease, bladder-cancer, internal use of turpentine* (Heywood Thomson, *Lancet*, July, 1867; PARKES).

Dr. Parkes desires to draw a strong distinction between “temporary” and “permanent” albuminuria. The former ought to imply that the albuminuria, after lasting for some days, or even weeks, finally disappears entirely. The latter implies that albuminuria does not disappear. Dr. Parkes found temporary albuminuria, with the quantity of albumen large, in acute lobar pneu-

monic cases and acute renal cases, such as Bright's disease. He found albumen considerable in amount in *typhoid*, *variola*, and *scarlatinal cases*, and small in amount in *paraplegia (spinal)*, *hemiplegia*, *chronic phthisis*, *acute pleurisy*, *acute bronchitis*, in *hypertrophy*, *dilatation*, and *valvular affections of the heart*, in *acute and subacute rheumatism*, in *purpura hæmorrhagica*, in *typhoid* and *typhus fever* and in *erysipelas* (PARKES, p. 188).

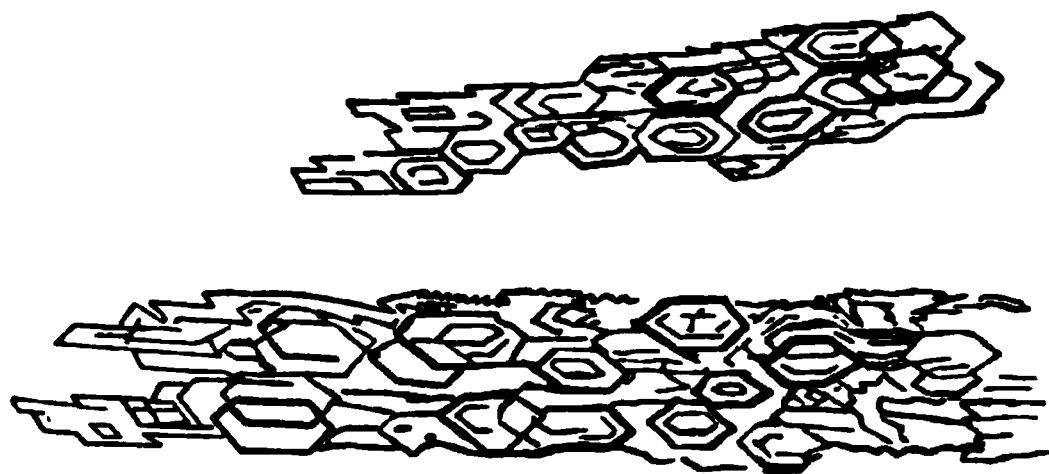
"Permanent albuminuria" he found associated with all forms of *Bright's disease*, with *encephaloid* and *cystic disease of the kidney*, with *leucocythæmia* and *presumed lardaceous disease of the kidney*, in *chronic phthisis*, and in *pleurisy*, where casts and kidney-structures were seen in the urine, in *hypertrophy* and *valvular affections of the heart*, in *cerebral softening of hemiplegia*, in *pancreatic disease*, in *purpura hæmorrhagica*, and in *typhoid fever*. The result is that if heart diseases be excluded, "permanent albuminuria" indicates kidney disease invariably, and the distinction between "temporary" and



"permanent" albuminuria is apt to be drawn with insufficient care; and in all cases where it occurs there is either congestion and increased lateral pressure on the vessels of the kidney, or there is absolute structural disease of their walls. In the Mauritius, and in Bourbon, a singular hæmaturia sometimes occurs, in which a varicose condition of the lymphatics has been supposed to cause the albumen (PARKES, p. 190).

3. *Urea*.—It is often of importance to determine the mere presence of urea in a small amount of fluid, as in the fluid of the ven-

FIG. 81.†



tricles of the brain. The process has been already explained at p. 280; and if, in place of nitric acid, oxalic acid is used, we obtain crystals of *oxalate of urea* (Figs. 80, 81).

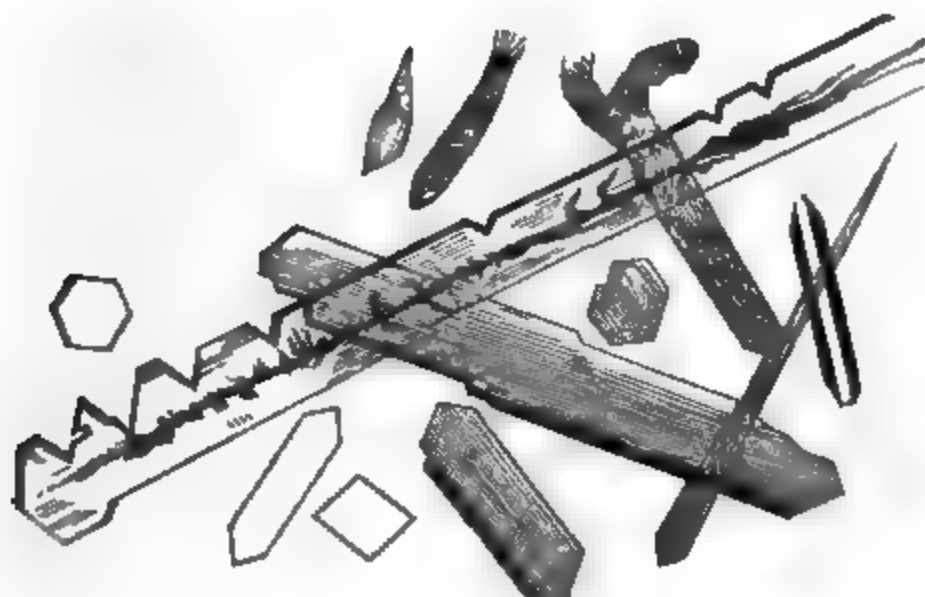
\* Oxalate of urea—perfect crystals (after BEALE). "Urinary Deposits," No. II, Plate IV.

† Oxalate of urea (after BEALE). "Urinary Deposits," No. II, Plate IV.



The alcoholic extract of urea leaves, on spontaneous evaporation, acicular crystals of the following form (Fig. 82):

FIG. 82.\*



100th of an inch  $\times$  42.

## SECTION IX.—DETAILED DESCRIPTION OF DISEASES OF THE KIDNEY.

**ACUTE BRIGHT'S DISEASE**—*SYN.*, ACUTE ALBUMINURIA; ACUTE DESQUAMATIVE NEPHRITIS; ACUTE RENAL DROPSY; [ACUTE TUBAL NEPHRITIS.]

**LATIN** *Eq.*, *morbis Brightii acutus*; **FRENCH** *Eq.*, *Maladie de Bright aiguë*; **GERMAN** *Eq.*, *Acute Bright'sche Krankheit*; **ITALIAN** *Eq.*, *Malattia del Bright acuta*.

**Definition.**—An intense febrile disease, which may come on after scarlatina, and other exanthemata, or independent of these, and which is marked by signs of intense congestion of the kidney, with exudation and hemorrhage into the tubes, and desquamation of the epithelium. The secondary phenomena are uræmic symptoms to a greater or less degree, and in the majority of cases general dropsy (FERRICHES, PARKES, JOHNSON, RAYER).

**Pathology.**—This affection seems to bear a similar relation to chronic Bright's disease that cases of acute phthisis bears to scrofula. It is described under the name of "*acute desquamative nephritis*" by Dr. George Johnson (whose investigations have especially elucidated this form of kidney diseases); and corresponds with the acute inflammatory dropsy of many writers. Elements closely allied to pus form in the kidney-tubes.

In the rapid forms of Bright's disease the products in the urine occur at first in the form of casts—a catarrhal process. These casts may accumulate and block up the tubes. The kidney is then enlarged, of a white color (the large white kidney of Bright). Acute

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\* Oxalate of urea from urin —extraction by alcohol, and an oxalate formed by the addition of oxalic acid.

dropsy is constant, often ascribed to cold, or as a result of scarlatina. Urine may be for a time suppressed, and the little which passes is of a red-brown color, generally from blood. The sediment is abundant and deeply tinged with blood. It contains albumen, blood-casts, and renal epithelium. The sediment is "composed of coagulated fibrine, blood-corpuscles, cells having for the most part the character of renal epithelium, and occasionally crystals of uric acid. Some of the fibrine is coagulated in irregular masses, having no definite form; this is always the case when the hemorrhage has been abundant and rapid, so that much of the blood has escaped from the kidneys before it has had time to coagulate; but with these masses there will be seen numerous cylindrical bodies composed of fibrine, which, having exuded from the Malpighian bodies, have coagulated in the tubes, and, escaping thence, present solid cylindrical moulds of the interior of the tubes, in which are entangled blood-corpuscles and epithelial cells, which have been shed by a process of desquamation from the surface of the tubes" (Johnson, *On the Kidney*, p. 89). To such casts, characterized by the presence of recently formed and entire epithelial cells, Dr. Johnson proposes the name of "*epithelial casts*"—their average diameter being about  $\frac{1}{16}$ th of an inch. Death, sometimes after only a fortnight's illness, discloses a large soft kidney, with swollen cortical substance of a dark color, on removal of the capsules, and the surface much injected. It exudes, on section, drops of blood. To the microscope the tubes are opaque, and are filled with lymph-corpuscles and granular matter. Some have lost their epithelium, and are filled with coagulated fibrine, which, if expelled, would form a fibrinous cast.

At a later period the pyramidal portions are still of a dark color; but afterwards the cortical part becomes paler and softer, the surface not smooth when cut, and giving out a turbid fluid when squeezed. The tubes are seen to be denuded of epithelium, and filled with masses of secretion, which take on the forms of the tubes. Scanty and bloody urine are conditions associated with engorgement of the Malpighian tufts. These are visible as red specks to the naked eye, and the tubes may be found filled with blood, or containing hæmatine. The morbid process continuing, secretion collects in the tubes and fills them to an extreme degree, and the more it accumulates, the larger and whiter the kidney becomes. The material can be seen with the naked eye in the cortical part. The surface is pale and smooth, with stellate or arborescent venation, surrounding numbers of opaque white spots. A section shows similar appearances, but the white material is now seen disposed in lines. If a congeries of tubes are swollen between the bloodvessels, granulations are thus formed. The new material consists of large masses of dark, granular, and fatty matter, which distend the tubes.

A section shows, microscopically, large portions of tubes quite opaque; their natural lining is gone, and contiguous tubes become united or massed together by the new material which fills them; and it is often difficult to determine whether the partition-membranes of the tubes exist or not (WILKS).

Granular matter may also be seen in some parts of the straight tubes. It is exceptional to find this material in the capsule of the Malpighian tufts.

[Dr. Dickinson gives the following description of the morbid appearances found in this disorder: The kidney becomes often more than double its usual weight from the great increase of blood. The surface is perfectly smooth, but there is much increase of vascularity; the vessels which divide the surface into lobules, and which in health are but faintly seen, become intensely injected, so as, sometimes, to give an almost uniform redness. The capsule is loose and thin, as in health. On section the inside presents a red or chocolate color, and drips with blood. The pelvis is injected. Underneath the blood, which obscures the tissue, there is a buff deposit, which becomes more evident after washing. The Malpighian bodies stand as red dots.

It may happen that though the disorder be no less acute, the congestion will be less conspicuous than the increase of bulk, the color being whiter than in health, though the cut surface exudes blood freely, and the whole organ is greatly injected, from the vascularity being masked by the opaque white epithelium which distends the tubes; and the cortex, which is much increased, looks as if it consisted of two materials—a red and a buff, coarsely intermingled. The cones are less changed than the cortex, being simply congested.

Under the microscope the cortical tubes are seen to be stuffed with an opaque brown material, which so long as it remains in the tube does not show any structure, looking uniformly granular. Spread out on the glass, it is seen to consist of cells of epithelium, not changed excepting that they may be stained of a brownish color, besides blood-corpuscles and indefinite granular matter, the latter probably resulting from disintegration of the epithelial cells. This condition is most marked in convoluted tubes, but the straight usually contain more or less of the same material. Beside the cell-growth, there is usually fibrinous exudation in the tubes, which, in some cases, is abundant.

The Malpighian bodies stand out prominently, and their vessel is seen to be distended with blood-corpuscles.

There are no other changes in the organ. There is no interstitial effusion, and, excepting the distension of the tubes and bloodvessels, the kidney is natural. The disorder is essentially a renal catarrh, depending upon a too prolific epithelial growth, and not upon any change of the cells, which, taken singly, present no tangible departure from their normal state.

The variations in the appearance of the organ depend upon the relative proportions of blood and epithelium—the more congestive varieties being produced by exposure to cold, while those in which the epithelial formation is more apparent are generally due to scarlatina. In the latter case the tubes contain little or no blood, but a profusion of natural gland-cells, with some granular material, probably from the breaking up of others (*Pathology and Treatment of Albuminuria*, p. 21–22).]

The *Urine* in the early stages and height of the disease presents intensely febrile characters. It is small in quantity, deeply pigmented, and deposits urates. It contains a variable but usually a large amount of albumen and blood. The sediments consist of desquamated kidney, ureter, and bladder-structures, and voided renal

cylinders, and sometimes large masses of coagulated fibrine, or partly decolorized clots. Urea is augmented[?]; and when it appears to be below the normal amount, its exit is impeded, and uræmic symptoms are present, which generally increase and prove fatal. When the urine is very scanty, it becomes almost solid by heat. When the disease is about to end fatally, the quantity of water and of the solids decrease, the diminution of the solids being more considerable than that of the water. The albumen decreases least, and is very abundant till the last. When recovery is about to take place, *diuresis* usually occurs; and often an enormous quantity of water is passed, containing much *urea* and *chloride of sodium*. The albumen at the same time diminishes and disappears, and the kidneys recover perfect health (PARKES, *l. c.*, p. 378).

[The state of the *urine* in acute Bright's disease may be thus summarized: It is scanty, invariably and largely albuminous from the beginning, and generally has an amount of blood to cause obvious discoloration. When acid, the blood gives a smoky tinge, and when alkaline or neutral, a pink or red hue. Average specific gravity 1019. There is a copious sediment on standing, which, in the absence of blood, consists of the contents of the tubes: (*a.*) Multitudes of cells of renal epithelium, natural or fatty, or pus-globules, into which the cells have been converted. (*b.*) Epithelial casts, of transparent fibrine, inclosing epithelial cells, or cells packed together in a plug, or later, granular casts of broken up epithelium, or transparent fibrinous cylinders, of large or small "waxy casts." When the epithelium of the cells is changed by disease, the contents of the cells may be fat- or pus-cells. If there is hæmaturia the casts may contain blood-corpuscles, or may be tinted brown by hæmatine. The chemical changes are: Diminution of all the constituents, the water, the urea, and the chlorides being lessened to a greater extent than in any other renal disease (DICKINSON). The phosphoric, sulphuric, and uric acids are reduced in a less marked manner; the phosphoric most, the uric least. The water, urea, chlorides, and, sometimes, the uric acid, are, during convalescence, increased beyond the normal amount.]

**Causes.**—The poisons of *scarlatina*, *cholera*, *measles*, and *erysipelas* [*diphtheria*, *pneumonia*, *pyæmia*, *typhus*]; intemperance in alcoholic drinks; the exhausting influence of previous disease; deficiency of food, with fatigue and mental anxiety; exposure to cold and wet.

**Treatment.**—(1.) Relieve the kidneys as much as possible from the labor of elimination, by avoiding exposure to cold, by keeping the patient at rest in bed, in a room of moderate uniform temperature. (2.) The food should be scanty, consisting of gruel, arrowroot, milk, or weak broth; pure water is the best drink, and alcoholic fluids are not to be taken on any account. (3.) Free action of the skin and bowels must be maintained. The *hot air bath* and antimonial remedies are the best agents to effect the first of these conditions, and free perspiration is to be encouraged by bedding the patient in blankets. *Antimonial wine* may be given in doses of from fifteen to thirty drops every four or five hours. The bowels are to be kept open by the *compound jalap powder*, in doses of twenty to sixty grains, repeated daily or on alternate days. It may be alternated

with *podophyllin*, or with extract of *colocynth*. Mercury is not to be given. (4.) Cupping over the loins relieves pain in the back, and the quantity of urine passed generally increases after eight or ten ounces of blood have been withdrawn in this way from an adult, or two or three ounces from a child three or four years old. (5.) When the tongue becomes clean and the general symptoms improve, mutton broth or good beef-tea may be indulged in; and, as the digestion improves, solid food may be eaten in small quantities, beginning with fish and fowl, and afterwards mutton or beef. (6.) Flannel must be worn next the skin. (7.) Iron is of great service during convalescence, for in such cases the anæmia becomes extreme. Phosphate of iron in the form of *syrup*, or *citrate of iron and quinia*, or the *ferrum reductum*, are the most digestible forms, and they ought to be given in small doses repeated after every diet. (8.) Diuretics are not to be given (see JOHNSON, *loc. cit.*, p. 125 to 138). In a case where the urine was suppressed, fomentations consisting of infusion of the leaves of *digitalis* were found by Professors Christison and Vogel to increase enormously the amount of urine. Dr. Parkes found the amount of the albumen to diminish markedly from the use of the tincture of the sesquichloride of iron (*l. c.*, p. 379). [See pages 158-9 of this volume.]

## SUPPURATIVE NEPHRITIS.

LATIN Eq., *Nephritis suppurans*; FRENCH Eq., *Néphrite suppurée*; GERMAN Eq., *Interstitielle Nephritis*; ITALIAN Eq., *Nefritide suppurativa*.

**Definition.**—“*Inflammation, with suppuration of the substance of the kidney.*”

**Pathology.**—The general nature of inflammation having been fully considered in volume first, it is of importance, in studying the inflammations of the kidney, to determine—(1.) The tissue or tissues affected—vessels, parenchyma, or interstitial connective-tissue; (2.) Whether only the cortical substance, or the pyramidal, or the pelvis of the kidney, is affected, or all parts together; (3.) Whether the affection is partial or diffused.

In the commencement of the disease a definite tissue can generally be indicated by microscopic examination after death, as the starting-point of the affection; but later it is not so easy. The several tissues are often consecutively attacked.

There are two essentially different affections,—(1.) Parenchymatous nephritis; (2.) Interstitial nephritis; and these may be complicated with the fatty or the amyloid degenerations about to be noticed. All forms of disease may be present at once, sometimes one and sometimes another occurring first; but *parenchymatous nephritis* is most generally the primary affection (Rasmussen, *Med.-Chir. Review*, July, 1863).

The cells of the convoluted tubes appear to be larger and richer in albumen than those in the straight ones; and any disease of the former, rendering them inactive, is of far more serious import than



disease of the latter, producing an actual change in the urine. Affections of the latter portion of the kidney only—namely, the pyramidal—are usually of a catarrhal nature, and are described by Rasmussen as “*papillary catarrh*,” or “*catarrhal nephritis*.” The catarrh affects principally the straight canals and papillæ, and is often continued from the bladder and urethra. Its exciting causes are stimulant, alcoholic, or terebinthine drinks, the use of cantharides, or of acid diuretics. It may be the starting-point of parenchymatous nephritis, and often complicates it. Such cases are generally suppurative, especially when secondary to bladder inflammation, or paralysis of the bladder in spinal affections. The suppuration is then generally most obvious and active in the cortical portion of the organ. The kidney is enlarged, and its surface covered with minute points of pus or small abscesses. A section of the whole kidney shows that these abscesses are diffused throughout the cortical, to the exclusion of the pyramidal part. The mucous membrane of the pelvis is inflamed, and is described under the name of pyelitis (WILKS). When the capsule is highly vascular and involved in the process, it indicates a chronic affection, or is a result of a former inflammation. In cases of uncomplicated catarrhal nephritis, where suppuration does not occur in the cortical portion, the lesions are confined to the canals and papillæ. They present a whitish or yellowish striation, with hyperæmia of the intervening vessels, and bloody ecchymosis may be present over the whole kidney, especially if acid diuretics have been largely given. If the disease continues long, the distended urinary canals press on the bloodvessels, when the hyperæmia generally ceases. The productive effects of the inflammation are limited to cell-growth (nucleated, club-shaped, fusiform, or ramifying), mixed with mucous catarrhal products (mucin), sometimes combined with fatty metamorphosis or other degeneration of epithelium.

*True parenchymatous nephritis* is an hypertrophy or “cloudy swelling” (see p. 153) of the large cells of the convoluted tubes. The cells take up large quantities of the *albuminates*, becoming distended, turbid, granular, and closely adherent to each other. Subsequently all the cell-forms vanish, and a large granular fatty mass is set free, which generally forms into “inflammatory globules.” It often co-exists with “*interstitial nephritis*.” When catarrh exists, it is usually associated with the symptomatic phenomena of Bright’s disease. Retardation of venous blood in the kidney is a common event in parenchymatous nephritis. Thrombi are then apt to form in the veins of the kidney, and being transported to the *vena cava* and heart, may find their way into the lungs. In the advanced stage of the disease, resolution or recovery corresponds to fatty degeneration of the cells, sometimes with loss of substance apparent on the surface, indurations, granulations, cysts, in connection with or separated from the urinary canals. The interspaces between the granular elevations consist of empty collapsed tubes. The granular cirrhotic appearance of the kidney is not similar in pathological origin to that of the liver. In the liver it is the interacinous connective-tissue which is first affected, and causes the cirrhotic granu-

lations; in the kidney, on the contrary, the parenchymatous cells of the tubes become first affected, and the lesion is afterwards complicated with *interstitial nephritis*. The Malpighian glanduli ultimately become small, corrugated, and surrounded by thickened capsules of connective-tissue. These capsules, with the epithelium, sometimes eventually become fatty, amyloid, or calcareous. With regard to *interstitial nephritis*, it has been doubted whether there is a connective-tissue or fibrous matrix to the kidney. I have never yet seen a human kidney in which it is not demonstrable. It becomes immensely hypertrophied, and minute cells grow up in its substance in large numbers, even to the extent of suppuration. Atrophy is also a usual result, first of all at the expense of the cortical part. On section it is seen to be diminished between the pyramids and the surface. The edge is irregular, and the surface is granular. The granulations are composed of bundles of tubes lying between veins, and are not to be confounded with white specks of deposit. The capsule separates with great difficulty, tearing the tissue which adheres to it. The glomeruli appear to be much closer together than natural (Dickinson, *Med.-Chir. Trans.*, 1863). Cysts are frequently met with on the surface.

Sometimes the whole of the new interstitial material undergoes the fatty degeneration. Granulations are also formed by the connective-tissue contracting round the canals and glomeruli, and circulation is more or less obstructed. The tubes become constricted in a bead-like manner, and the *tunica propria* is thickened and streaked. The glomeruli become small, homogeneous, and more or less fatty.

*Circumscribed interstitial nephritis* often affects the pyramids only, as a result of syphilis; nodes are the consequence, which, passing away or diminishing, leave cicatrix-like depressions not unlike those from hemorrhagic infarctions (RASMUSSEN, WILKS, VIRCHOW, DICKINSON).

The kidneys may suppurate—(1.) From morbid conditions of the blood; (2.) From external violence; (3.) From retention of urine; (4.) From calculi in the kidney (JOHNSON, *op. cit.*, p. 417).

**Symptoms.**—Uncomplicated acute nephritis has hitherto been considered so rare that its symptoms have not been well described. Those mentioned by Baillie are as follow: "When the kidneys," he says, "are inflamed, more or less pain is felt in the region of these glands, and the pain commonly shoots along the ureters. There is a sense of numbness down the thigh, and in the male there is often retraction of the testicle, or a feeling of pain in it. When one kidney is affected, these symptoms are only felt on that side. The urine is voided frequently, and is sometimes of a pale, but more commonly of a deep-red color. There is sickness and vomiting. The bowels are at the same time often costive, and subject to colicky pains. These symptoms are accompanied by more or less fever." "When pus is formed, the event may be known by the pus being mixed with the urine." Cases related by Mr. Stanley, however, by no means bear out this description. He gives the case of a man who had retention of urine in consequence of a gonorrhœal

discharge being stopped by injections. In this instance the kidneys were found extremely vascular and soft, with numerous minute depositions of pus throughout the cortical and tubular parts, and the infundibula and the pelvis were likewise filled with pus. The principal symptom was severe pain at the fifth lumbar vertebra. In another similar case, but not quite so acute, the kidneys were found so dark-colored as to be almost black, and at the same time remarkably flaccid. This patient died paraplegic, the loss of motion being complete, and that of sensation nearly so.

If nephritis passes to a chronic suppurative state, the pain in the loins is often severe and the appetite impaired, while pus is found often to a considerable amount in the urine, and if a calculus or gravel be the immediate cause, the urine may contain large portions of those substances mixed with blood. Dr. Johnson has clearly shown that the desquamative and suppurative processes in the kidney are much more closely allied than is generally supposed. They pass into each other by almost imperceptible gradations; the products appearing in the urine as purulent casts of the tubes.

**Prognosis.**—Acute affections of the kidney are in all cases of grave prognosis. The chronic forms of these affections are perhaps consistent with life, but in every case they greatly impair it, and are ultimately the cause of premature death. When pus-corpuscles take the place of renal epithelium the prognosis must always be unfavorable; and the suppurative process in the kidney is the most rapidly destructive of the lesions. Dr. Johnson has not met with a case of suppurative nephritis which has terminated in recovery (*op. cit.*, p. 440).

**Treatment.**—The treatment of acute nephritis must be according to the ordinary principles of the treatment of inflammation, by evacuants and opiates. Blisters in such cases are dangerous, and ought to be avoided. The neutral salts, with opiates, are admissible in some cases. Castor oil or other purgative substances which do not act so immediately on the kidneys, are more useful, such as compound jalap powder or podophyllin. The general plan of treatment is similar to that which has been already stated.

#### LARDACEOUS KIDNEY—*Syn.*, AMYLOID DISEASE; WAXY DISEASE.

*The Degenerations of the Kidney* are chiefly the fatty and the amyloid. (1.) *The fatty kidney* is large and flaccid, full of yellowish or whitish striæ and marks, combined or not with the characters of the parenchymatous inflammation just described. The calibre of the urinary canals is diminished, and they are separated by fatty masses. The glomeruli, and generally the walls of the vascular cells, are fatty or amyloid. (2.) *The amyloid degeneration of the kidney* begins with the arterial capillaries; especially—(1.) The loops of the *Malpighian* tufts; (2.) Their afferent vessels; (3.) The *vasa efferentia* and capillaries in the cortical part; (4.) The “*arteriola recta* ;” and after the lesion has advanced to a certain point the injection of the cortical substance becomes impracticable; the arteries

become impervious, the cortical substance anæmic, while hyperæmia increases in the pyramids; and hemorrhage is apt to occur at times, owing to the increased pressure on the inelastic vascular walls, giving rise to reddish-brown streaks or spots. Ultimately the Malpighian glomeruli become converted into solid, homogeneous, translucent globules; and the extension of the disease to the tubes and other tissue produces at length the condition known as waxy kidney. Sometimes the degeneration begins in the capillaries of the *arteriolæ rectæ*; but rarely are the large vessels affected, and it is long before the epithelial tissues degenerate.

The amyloid kidney must be distinguished from the large white and soft kidney described by Dr. Bright.

The amyloid kidney is much harder, more tough, and firmer than the white kidney of Bright. It is not easily torn, which is the case with Bright's white kidney. The surface appears uniform and smooth. The cortical substance is greatly increased in extent, pale, anæmic, and of a dim waxy appearance. The translucency, hardness, and uniformity are sufficient to identify the extreme cases; and in the more common and slighter cases the Malpighian glomeruli can be seen shining prominently, like glistening grains of a whitish-gray appearance, on the cut surface. The iodine test is absolutely necessary; and, unless a microscope has been used, it cannot be declared that amyloid degeneration is not present. Without any reagent, the Malpighian glomeruli appear under the microscope as globular transparent bodies, with a glistening aspect if they have suffered degeneration.

The capsule of the amyloid kidney is generally easy torn off, and loss of substance or atrophy may be present, as indicated by depressions or hollows of the surface beneath the capsules. The degeneration is most frequently combined with parenchymatous or interstitial nephritis, or the interstitial fatty kidney, especially in syphilitic cases. [See Dr. Dickinson's description of the morbid anatomy of this renal degeneration in this volume, p. 146.]

With regard to the amyloid kidney, Dr. T. Grainger Stewart, Pathologist to the Royal Infirmary, Edinburgh, has published (August, 1864, in *Edin. Med. Journal*) further observations, confirming those referred to in the previous volume (p. 134) of this edition.

In the cases of amyloid degeneration, Dr. Stewart has found great amelioration of the symptoms from the adoption of the following rules: (1.) To attend to the nutrition of the patient, giving good nutritive food in the form best suited to the individual tastes and powers of digestion; (2.) To give such tonic medicines as may improve the appetite; (3.) To give such hæmatic medicines as control the tendency to anæmia; and among these pre-eminently, the syrup of the *iodide of iron*. The *syrup of the phosphate*, as well as the *syrup of the phosphates of iron, quinia, and strychnia* (the formula for which is given at page 95, *ante*), are also, I think, most useful remedies; (4.) In all cases in which a syphilitic infection has been traced, and even in many others, *iodide of potassium* is to be given in

moderate and sustained doses. It diminishes the bulk of the liver in a remarkable manner (*Edin. Med. Journal*, August, 1864).

## SECTION X.—LESIONS WHICH TEND TO BE LOCALIZED IN THE SPLEEN.

### LARDACEOUS SPLEEN—SYN., AMYLOID DISEASE; WAXY SPLEEN.

LATIN Eq., *Lien lardaceous*; FRENCH Eq., *Dégénérescence lardacée*; GERMAN Eq., *Speckige Entartung der Milz*; ITALIAN Eq., *Milza lardacea*.

The chief part of the substance of this organ seems to be made up of distinct translucent Malpighian sacculi closely crowded together (BENNETT).

The amyloid degeneration of the spleen is frequently associated with a similar condition of other organs, especially the liver and the kidney. It is a condition characterized by great firmness of the spleen, peculiar waxy-like consistence, and with a distinctness and transparency of the Malpighian sacculi which are not usually very obvious (W. T. GAIRDNER). Of all the viscera and tissues of the body, the spleen is the one which is comparatively the most frequently affected by the lesion, although it was generally unknown up till 1853-4. Of the bodies examined in the Royal Infirmary of Edinburgh it was observed in 10 per cent. of all.

In the most extreme cases of the lesion the spleen is enlarged, and has a swollen aspect. Its weight and density are greatly increased. It feels to the touch like the consistence of wax and lead, and its section shows a dry and smooth surface. After exposure to the air its pulp may become of a bright red color; and then the Malpighian sacculi appear large and distinct, forming round, colorless, *transparent* granules about the size of a pin's head, slightly prominent, and so hard that they may be picked out by a knife. The pulp is greatly diminished in quantity, and seems in some to be entirely absent; so that the sacculi are crowded together, and the degenerate tissue in their vicinity becomes a continuous mass of a globular form. A small arterial twig can sometimes be seen passing into or through these Malpighian sacculi at their central part.

Microscopic sections are easily made, and their characters are even more striking than those presented to the naked eye. Under a low power (40 to 60 diameters) the Malpighian sacculi appear as large clear spaces of a circular or oval form, surrounded by the dark-red pulp. Under the higher powers (above 250) the nature of the alteration is seen to consist mainly of an alteration in the normal corpuscles of these sacculi, which are converted into and replaced by masses of a colorless, dense, highly translucent, and homogeneous material, and a careful examination discloses the outlines of irregular cell-forms. But these masses, owing to their roughness, are not easily broken up into the particles which com-



pose them (SANDERS). The translucent parts are very little acted on by reagents (acids, alkalies, alcohol). Its characters are marked and permanent, so that there is no difficulty at any time in recognizing or identifying the character of the degeneration.

Several varieties of the waxy spleen may be noticed. Two forms at least are distinctly indicated by Virchow, Sanders, and Wilks (although the latter observer says three):

1. *The Form in which the Malpighian Sacculi are the Structures prominently affected.*—They are sometimes large, and sometimes very small. In the former state we recognize the sago-like granules described by Virchow. In the latter case they are best seen after exposure to the air for a little time, or after having been well washed in water, and then put in alcohol, or into a solution of chloride of zinc, which brings out the waxy appearance very distinctly. In the most extreme cases they never occupy more than about half the bulk of the organ, the intervening pulp-substance being healthy.

2. *A Form in which the Pulp-Substance and Trabeculæ are mainly affected, leaving the Sacculi intact.*—It presents the same waxy consistence, the smooth dry section, and other characters of the lesion; but the sacculi are obscured by the peculiar translucent substance which pervades the pulp, and which looks as if melted tallow had been poured into the trabecular spaces (WILKS).

Histologically, there is no real difference between these two forms, because the corpuscles of the sacculi and of the surrounding pulp-substance are now known to be identical (BUSK and HUXLEY in Wedl, p. 247).\*

The degeneration seems to commence—(1.) In the arterial capillaries, where the little lateral bulgings are in connection with the Malpighian corpuscles. (2.) These sacculi contain at first normal splenic cells; but at a later stage the contents of the sacculi become irregular and granular, and lymph-corpuscles of a gelatinous appearance become changed into the minute masses of the waxy substance. (3.) Subsequently the trabeculæ and pulp-stroma become affected (BUSK and HUXLEY, as above).

Some cases of albuminoid disease related by Dr. Jenner are evidently of this second nature.

These lesions of the spleen seem to have arrested the attention of Drs. Bright and Hodgkin many years ago. They also recognized the frequent association of diseased lymphatic glands with it. Dr. Bright, indeed, refers to the circumstances as having been originally pointed out by Dr. Hodgkin in vol. xvii of the *Medico-Chirurgical Transactions*.

### SPLENITIS.

LATIN Eq., *Splenitis*; FRENCH Eq., *Splénite*; GERMAN Eq., *Milzentzündung*; ITALIAN Eq., *Splenitide*.

**Definition.**—*Inflammation of the substance of the spleen.*

**Pathology.**—A disease which is extremely rare in this country. It is usually limited to certain districts, as Cambridgeshire, Essex, or other paludal places.\* It is common in the East Indies, especially

in the low marshy districts of Bengal, and occurs in the paludal districts of other parts of the world. Now and then it is said to originate from a blow or other accidental violence. It is sometimes seen in children under ten years of age.

The few cases of disease of the spleen occurring in this country will account for its pathology having been little studied. In diffuse inflammation, however, of this viscus, we find it enlarged, of a deep venous color, and its tissue so softened as to be readily broken down, or even reduced to little more than the consistency of coagulated blood. Diffuse inflammation may terminate by resolution, or pus may form; and in this case one or more abscesses, often containing several ounces of pus, have been formed. The abscesses sometimes make their way to the surface. Dr. Baillie mentions that the spleen has been found in a state of gangrene.

**Symptoms.**—Acute inflammation of the spleen is seldom seen unless accompanied by ague; and the additional symptoms are probably tumefaction and some pain of the left side followed perhaps by dropsy.

In chronic affections even abscesses will sometimes form without any marked local symptoms. Dr. Abercrombie gives the case of a gentleman who was dyspeptic, but took a great deal of nourishment, who was much reduced in strength and flesh, but whose pulse was seldom more than 96 to 100; whose nights were good, though he was occasionally slightly feverish, and who was able, till within a few days of his death, to drive out in his carriage. At length he died, after suffering for two or three days from diarrhœa, but without any suspicion of the spleen being affected. On examination, however, the spleen was found enlarged, and in its centre an abscess containing several ounces of pus.

The more common form of diseased spleen is hypertrophy; and in these cases it can almost always be detected by palpation or percussion, sometimes extending low down into the pelvic region, well over on the right side of the *linea alba*, and extending backwards almost to the spine. In these cases the patient complains of weight and uneasiness, rather than of soreness; his pulse is natural, but the countenance extremely sallow, his person greatly emaciated, his bowels irritable; and these symptoms are for the most part accompanied by œdema of the lower extremities, or by ascites. The most remarkable part of the history of these cases, however, is, that notwithstanding the sallow and emaciated state of the patient, he is often seized towards the close of the disease with hemorrhage from the stomach and bowels, often so profuse that many pints have been passed or thrown up, greatly exhausting the patient, and rapidly hastening his dissolution.

The course of chronic splenitis is generally long, the patient usually surviving one or more years in the worst cases.

**Diagnosis.**—Enlarged spleen can only be confounded with encephaloid or other tumor of the abdomen.

**Treatment.**—Bleeding in *splenitis* has not been found to effect a cure, while mercury has been found for the most part, not only not to be useful, but even to be most pernicious. "I feel," says Mr.

Twining, "more anxious fairly to show the baneful effects of mercury in the disease now under consideration, because the instructions usually laid down in the best systems of medicine do not inculcate the avoidance of mercury in any case of enlarged spleen, nor do they advert to the pernicious effects of that state of disease which I have termed vascular engorgement." This gentleman, in further proof of his position, mentions thirteen cases in which the patient either died of mortification of the cheek, the nose, the upper lip, or after having lost all his teeth or a large portion of the jaw, in consequence of the use of mercury, and, if he survived the employment of this medicine, the spleen remained permanently enlarged. Dr. Voigt, physician to the Danish establishment at Serampore, writes that, although most authors recommend mercury, it is an indisputable fact that a very small quantity, even a few grains, generally occasion a profuse salivation, and so violent an affection of the mouth that mortification sets in, the teeth drop out, the bones become carious, and death ensues. Consequently, mercury and bleeding ought to be little used; and in India, in their stead, a *spleen mixture*, not very dissimilar to that recommended by Celsus, is most in vogue. The best, according to Mr. Twining, consists of the following:

R. Pulv. Jalap; Pulv. Rhei; Pulv. Calumba; Pulv. Zingib; Potass. Supertart., āā ʒj; Ferri Sulphat., ʒss; Tinct. Sennæ, ʒss; Aquæ Menthæ Pip., ʒixss. Of this mixture an ounce or an ounce and a half is to be taken twice a day, or such quantity as may produce three or four stools in the twenty-four hours.

The spleen mixture is, in some instances, efficacious, but in a much larger number of instances it entirely fails; and under these circumstances the *iodide of potassium* and the *bromide of potash* have been recommended. The dose of the bromide of potash is gr. v to x *ter die*, combined with *camphor mixture*. The marked influence of the *biniodide of mercury*, in the form of an ointment, rubbed into the skin over the surface of the spleen, in reducing its enlargement, has been already noticed (p. 853, *ante*). The invalid soldiers who suffer from enlarged spleens invariably apply for some to take away with them when they are discharged from Netley Hospital.

#### HYPERTROPHY OF THE SPLEEN.

LATIN EQ., *Hypertrophia*; FRENCH EQ., *Hypertrophie*; GERMAN EQ., *Hypertrophie*; ITALIAN EQ., *Iperetrofia*.

The spleen is more often hypertrophied, as a result of malaria or of *leucocythæmia*. In the *Medical Commentaries* an hypertrophied spleen is mentioned which weighed 11 lbs. Portal speaks of another that weighed 30 lbs; and Lieutaud met with one, in a woman who had been ill seventeen years, that weighed 32 lbs. It is singular that these large tumefied spleens sometimes subside very rapidly. Abercrombie mentions one that subsided in a week after the ague

on which it depended had been arrested. The hypertrophied spleen is generally more or less indurated.

The spleen is occasionally atrophied, so that little more than a rudimentary spleen remains. It is also found indurated, and often greatly softened. Hydatids have been found in the spleen. In a few instances small portions of the spleen, about the size of a nut, are found indurated and nearly white. These appearances are supposed to arise from slight effusions of blood into the substance of the spleen, which become organized, and the coloring particles being absorbed, leave the appearances in question; or they are due to *emboli*.

## CHAPTER XIV.

### DISEASES OF THE CUTANEOUS SYSTEM.

#### SECTION I.—GENERAL PATHOLOGY AND CLASSIFICATION OF DISEASES OF THE SKIN.

Diseases of the skin have been regarded too much as a specialty; and only now are they beginning to be looked upon as a class of diseases whose pathology is capable of being investigated and studied like other diseases. The expressions of skin diseases are undoubtedly various in appearance; and the same disease does not always exist in the same simple or elementary form.

The classification of skin diseases hitherto in use is that which is comprehended in the eight orders of Willan and Bateman; and the characteristics of these orders are embraced in the following definitions of terms in common use in the description of skin diseases:

**ORDER I. PIMPLES.**—*Papulæ* are simple solid acuminate elevations of the cuticle, resembling an enlarged *papilla* of the skin. They commonly terminate in a scurf, and sometimes, though seldom, in slight ulceration of its summit.

**ORDER II. SCALES.**—*Squamæ* consist of cuticle in patches, plates, or laminæ, in which the epidermic cells are morbidly adherent, hard, thickened, whitish, and opaque. These scales cover either small papillæ, red elevations, or larger deep red and dry surfaces.

**ORDER III. RASHES.**—*Exanthemata* are composed of superficial red patches of irregular size, and variously diffused. They disappear under pressure, and terminate by desquamation.

**ORDER IV. BLEBS.**—*Miniature Blisters*—*Bullæ*. They differ from vesicles in size, being larger. A large portion of cuticle is detached from the skin by the interposition of a watery fluid, usually transparent. The skin is red and inflamed beneath the blebs.

**ORDER V. PUSTULES.** *Pustulæ* consist in circumscribed elevations of the cuticle, and contain pus. They have red and inflamed bases, and are succeeded by an elevated scab, which may or may not be followed by a cicatrix.

**ORDER VI. VESICLES.**—*Vesiculæ*—small acuminate or orbicular elevations of the cuticle, containing lymph, which, at first clear and colorless, may become amber-colored, opaque, or pearl-like. Vesicles are succeeded by a scurf or a laminated scab.

**ORDER VII. TUBERCLES.**—*Tuberculæ*—small, hard, indolent elevations of the skin, sometimes suppurating partially, sometimes ulcerating at their summit.

**ORDER VIII. SPOTS.**—*Maculæ* are permanent discolorations or stains of some portions of the skin, often with a change of structure. They may be whitish, dusky, or dark.

Under these several orders Dr. Bennett proposes the following classification:

<b>ORDER I.—<i>Exanthemata</i>.</b> Erythema. Roseola. Urticaria.	<b>ORDER IV.—<i>Papulæ</i>.</b> Lichen. Prurigo.	<b>ORDER VII.—<i>Maculæ</i>.</b> Lentigo. Ephelides. Nævi. Purpura.
<b>ORDER II.—<i>Vesiculæ</i>.</b> Eczema. Herpes. Scabies. Pemphigus.	<b>ORDER V.—<i>Squamæ</i>.</b> Psoriasis. Pityriasis. Ichthyosis.	<b>ORDER VIII.—<i>Dermatozoa</i>.</b> Entozoön folliculo- rum. Acarus. Pediculus.
<b>ORDER III.—<i>Pustulæ</i>.</b> Impetigo. Ecthyma. Acne. Rupia.	<b>ORDER VI.—<i>Tuberculæ</i>.</b> Lepra Tuberculosa. Lupus. Molluscum.	<b>ORDER IX.—<i>Dermatophytæ</i>.</b> Achorion Schönlein (Favus). Achorion Grubii (Mentagra.)

These and similar classifications are mainly anatomical. They do not attempt to throw any light on the causes producing the various diseases, which are contemplated as so many distinct and individual "unities," their mutual relations being of secondary importance. The basis of classes, as proposed by Willan and Bateman, is only useful as furnishing outward marks or anatomical characters useful in describing the morbid anatomy of skin diseases; but it has no relation to the causes, to the pathology, nor to the treatment of such diseases. An affection which is papular to-day may be vesicular to-morrow, and pustular eventually. Under the same division, or class, maladies are brought together which Nature has stamped with broad and obvious marks of distinction. Febrile diseases are associated with non-febrile; and ailments which are local and trivial are associated with diseases of grave import and deeply rooted in the system. And, on the other hand, distempers which Nature has plainly brought together and connected by striking analogies and resemblances, the methodical arrangement of Willan and Bateman puts widely asunder (WATSON).

A purely anatomical classification of cutaneous lesions may be stated as follows, after the late Dr. A. B. Buchanan:

- I. **Deformities of the Skin** (mostly persistent and non-inflammatory).
- II. **Eruptions of the Skin** (mostly transient, and with the phenomena of inflammation).
  - (A.) **PRIMARY FORMS.**—(1.) *Macula*: (a.) Erythematous; (b.) Purpurous—comprehending stigma, petechia, vibix, ecchymosis. (2.) *Papula*. (3.) *Tuberculum*. (4.) *Pomphus*. (5.) *Vesicula*—comprehending vesicula miliaris and phlyctæna. (6.) *Pustula*—comprehending achor, psudracium, and phlyzadium. (7.) *Bulla*.
  - (B.) **SECONDARY FORMS.**—(1.) *Ercoriatio*. (2.) *Ulcers*. (3.) *Rima*. (4.) *Squama*. (5.) *Crusta*. (6.) *Cicatrix*.

Alibert conceived the idea of arranging skin diseases into natural families, of which he gives *twelve*. His classification presupposes a knowledge of the subject incompatible with teaching.

M. Hardy, of the Hôpital St. Louis, classifies skin diseases accord-



ing to their nature, into the following *ten* natural families: (1.) *Macules and deformities*; (2.) *Local inflammations*; (3.) *Parasitic diseases*; (4.) *Eruptive fevers*; (5.) *Symptomatic eruptions*; (6.) *Dartres, or Tetters*; (7.) *Scrofulides, or strumous eruptions*; (8.) *Syphilides, or syphilitic eruptions*; (9.) *Cancers*; (10.) *Exotic diseases*.

In an admirable paper "On the Theory and Classification of Inflammations of the Skin," by the late Dr. A. B. Buchanan, Physician to the Dispensary for Skin Diseases in Glasgow, it is shown that, in the present state of our knowledge, any classification resting on one principle of division only, runs the risk of being, to a greater or less extent, *artificial* and untrue; and to secure a *natural* system, several principles must be taken into account, though greater importance may be attached to some of these than to others (*Edin. Med. Journal*, January, 1863). Skin diseases, like all other diseases, ought to be classed according to their *nature* or *pathology*. The cause of a disease, when known, gives a more accurate indication of its true nature, and its means of cure, than a knowledge of its anatomical appearances or symptoms, which are mere effects of the cause. So far as can be done, therefore, Dr. Buchanan proposes that skin diseases should be classified according to their causes. Not knowing the cause, some other principles must be sought for under which groups may be formed; and he finds—in the pathological processes recognized as *inflammations, new formations, hemorrhages*—that skin diseases may be arranged into three or more groups.

*The inflammatory skin diseases* are those which affect the "*pars papillaris*" of the corium, as well as the "*pars reticularis*" and the subjacent connective-tissue; but the process is not confined to these, for it affects even the epidermis, whose cells are changed in formation and in structure in the same way as all cells are altered by the process of inflammation.

*The skin diseases characterized by new formations* may be regarded as—(1.) The productive effects of inflammation; or (2.) The productive effects of such constitutional states as cancer, scrofula, and Bright's disease.

*The hemorrhagic skin diseases* arise from the escape of blood in small quantities, owing to the rupture of capillary vessels, or to the escape of coloring matter along with the exudations. They are affections peculiar to the cutis; for, although blood may be effused among the epidermic cells, hemorrhage can only originate from the cutis.

On these principles a classification somewhat of the following form has been suggested:

## CLASSIFICATION OF SKIN DISEASES (DR. A. B. BUCHANAN).

### Class I. Inflammations.

#### GROUP I.—SIMPLE INFLAMMATIONS (ALLIED TO *Simple Dermatitis*).

- (1.) ERYTHEMA ([a.] *simplex*; [b.] *multiforme*; [c.] *chronicum*—comprehending *papulatum, nodosum, strophulus, squamosum, pityriasis furfuracea, membranacea, rubra*).

- (2.) HERPES (*simplex* and *zoster*).
- (3.) URTICARIA (idiopathic, from ingestion of particular kind of food; from uterine affections; or persistent).
- (4.) DERMATITIS (idiopathic, as from *burns*, or from *frost-bite*; or symptomatic, as of *erysipelas*; or phlegmonodes, as *furunculus*, *anthrax*, *Aleppo tubercle*).
- (5.) PEMPHIGUS (benign, persistent, and foliaceous).

GROUP II.—ECZEMATOUS INFLAMMATIONS (ALLIED TO *Eczema*).

- (1.) ECZEMA (*erythematodes*; *E. papulosum*, comprising *lichen simplex* and *prurigo*; *E. vesiculare*; *E. rubrum*; *E. pustulosum*, comprising *impetigo sparsa*, *figurata*, and *pilaris*; *E. lichen*; *E. squamosum*; *E. pityriasis*).
- (2.) ACNE (comprising *A. simplex*; *A. pilaris*; *A. rosacea*).
- (3.) ECTHYMA ([a.] *simplex*; [b.] *chronicum* = *rupia*; [c.] *gangrenosum*).
- (4.) PSORIASIS (*punctata*, *guttata*, *nummularis*, *circinata* [*lepra*], *gyrata*, *confluens*).

GROUP III.—ULCERS.

- (1.) IDIOPATHIC.
- (2.) SYMPATHETIC.
- (3.) CONSTITUTIONAL.

Class II.—New Formations.

GROUP I.—HOMOLOGOUS NEW FORMATIONS.

- (1.) EPIDERMIC (*epithelial growths*, comprising *callositus*, *clavus ichthyosis*, *cornu cutaneum*).
- (2.) PIGMENTARY (*lentigo*, *ephelis*, *moles*, *melanosis*, *chloasma*, *silver stain*, *leucopathia*).
- (3.) DERMIC (*cicatrix normal*, or *cheloid*, *cutaneous tumor* [*wens*], *multiple tumors* [*mycosis*], *molluscum simplex*, *condylomata*, *verruca vulgaris*, *verruca mollis*).

GROUP II.—HETEROLOGOUS NEW FORMATIONS.

- (1.) PSEUDOPLASMS (*lupus*, comprehending *maculosus*, *tuberculosis*, *hypertrophicus exedens*, *serpiginosus*; and *lepra*, comprehending *maculosa*, *tuberculosa anæsthetica*, *exulcerans*).
- (2.) NEOPLASMS (*epithelioma*, *carcinoma*).

Class III.—Hemorrhages—*e. g.*, *petechiæ*, *vibices*, *ecchymosis*, *purpura*.

Class IV.—Diseases of Accessory Organs—*e. g.*, *hair*, *nails*, *sweat glands*.

Class V.—Diseases Defined by Uniform Causes.

GROUP I.—PARASITIC DISEASES.

GROUP II.—SYPHILITIC ERUPTIONS.

GROUP III.—ERUPTIONS OF SPECIFIC FEVERS.

GROUP IV.—SCROFULODERMATA.

SECTION II.—DESCRIPTION IN DETAIL OF THE MORE COMMON DISEASES OF THE SKIN.

ERYTHEMATOUS INFLAMMATIONS.

1. *Erythema*.—Its expression is that of uniform redness simply, with puffiness of the skin, distributed in distinct patches of some size. It is accompanied by little constitutional disturbance; and if febrile phenomena are decided, it may betoken more severe areolar inflammation than *erythema*, and a more grave disease. It is thus apt to be mistaken for *erysipelas*. The varieties of *Erythema* are,—*E. læve*; *E. fugax*—syn., *E. volaticum*; *E. marginatum*; *E. papulatum*; *E. tuberculatum*; *E. nodosum*. Of these

varieties *erythema nodosum* is perhaps the most important. The indisposition which precedes the eruption is generally associated with a slight degree of fever. Red oval patches, considerably elevated and very tender, appear on the fore part of the legs, sometimes on the arms; and the long diameter of these patches is generally parallel to the axis of the limb. They sometimes form bumps an inch and a half long and an inch broad on the anterior aspect of the leg. After a few days the red color changes to a blue, the patches become soft, and although something like fluctuation may be felt, yet suppuration does not occur. Thus the bumps of erythema gradually subside. Sometimes the disease is seen in feeble boys; but it is most common in young women, in whom it seems associated with disordered menstruation, or with rheumatism (RAYER, WATSON). In the chronic stage desquamation invariably occurs; and to this stage of the disease the name *pityriasis* has been given.

**Treatment.**—*Rest* and *quinine*, after aperients.

2. *Herpes*.—This disease is expressed by red patches, of irregular form and variable size, upon each of which there arises a group, cluster, or crop of extremely minute vesicles. It is often preceded by considerable local irritation, or by cutaneous pain or neuralgia, which sometimes remains after the eruption has died away. The eruption runs a very definite course, and, when not interfered with, its development and defervescence is completed in about ten days. The vesicles ultimately rupture, and a gum-like scab forms over the group, which shrivels and contracts upon itself. The disease occurs sometimes—(1.) As one or two patches on the lips—*herpes labialis*; (2.) As a band of patches which encircles half the circumference of the trunk, or the whole circumference of a thigh—*herpes zoster*, *zona ignea*, *cingulum*, or *the shingles*.

[*Herpes zoster* is not a true skin affection, but a local expression of irritation of a nerve; most commonly an intercostal, though, sometimes, one of the cutaneous nerves of the abdomen, of the thigh, upper extremity, or of one of the branches of the superficial plexus of the neck, or of the facial, the eruption following the track of one of these nerves. The dorsal nerves are much the most frequently affected, especially the third and fourth; those of the forearm and thigh, least. Bateman held that its course was as regular as that of one of the eruptive fevers, having like them eruption, maturation, and decline, all in a limited period. It rarely happens twice in the same person; and seldom appears on both sides at the same time. Mr. Hutchinson thinks both sides are equally liable to be affected; Cazenave and Schledel say that in their experience it was on the right side in 19 cases out of twenty; while Rayer and Reil note the left as most frequently attacked. It rarely encircles the whole body, the zone being incomplete either in the centre, or at one or other extremity. A slight pricking or burning pain precedes its outcome, and often there is some constitutional disturbance. Severe dyspnoea, and a catching pain in the side (*pleurodynia*) are not unfrequently initial symptoms.]

(3.) A very common and troublesome form of the disease affects

the foreskin—*herpes preputialis*. [It occurs too in the female, on the labia, after menstruation, or during pregnancy.] Its effects are often mistaken for chancres, and by unprincipled persons may be treated as such. It has nothing to do with syphilis, and its history is a sufficient guide to diagnosis (see vol. i, p. 688). The disease may be prolonged by ulceration of the vesicles, or by irritant applications, or when the scabs are prematurely chafed off.

**Treatment.**—The state of the stomach and digestive organs, and diet generally, should be regulated. Malt liquors should be avoided; and, if pain is severe, opiate fomentations may be applied. *Herpes preputialis* requires no treatment beyond careful ablution with tepid water, and the interposition of a piece of wet lint between the prepuce and the *glans penis*; [or the lint may be soaked in lead water.] If there is much pain, the lint should be moistened with a watery solution of opium. [If the vesicles have ulcerated from irritation, it may be necessary to touch them with sulphate of copper, or nitrate of silver.]

3. *Nettlerash*, or *Urticaria*, consists of an eruption of little solid elastic eminences, roundish or oblong, pale in the centre, and red at the circumference. These are commonly called “wheals,” similar to what results from the mark of a lash. The eruption is attended with an intense heat, a burning, tingling, or pricking sensation, very much like that produced by the stinging of a nettle. There are several varieties of *urticaria*—(a.) *Urticaria acuta*; (b.) *Urticaria chronica*. The acute forms are generally connected with the ingestion of some kinds of food—oatmeal, bitter almonds, shell-fish, &c. The more chronic and intermittent forms are associated with uterine or other affections.

**Treatment.**—Emetics and purgatives in the first instance; afterwards the correction of faulty digestion. The surface of the eruption may be dusted over with flour; or the following lotion may be used:

R. Carbonatis Ammoniae, ʒj; Plumb. Acetatis, ʒij; Aquæ Rosearum, ʒviij.

4. *Pemphigus*, or *Pompholyx*, is characterized by an eruption of large vesicles, filled with serous fluid, known as *bullæ*. These *bullæ* vary in magnitude, are generally distinct, but numerous. They spring up in successive crops, generally in the [face, hands], fore-arms and legs, [soles of the feet], surrounded by redness of the skin. [Generally hemispherical, they may, by their confluence, assume great irregularity of shape, and attain a diameter of several inches in extent.] The vesicles, originally transparent, gradually become opaque, pearl-colored, and ultimately of a pale red-color. [The fluid at the beginning is clear and transparent, and may continue so throughout, but more frequently it becomes turbid or sero-purulent, or mixed with lymphic flakes. In very young, or old, or unhealthy subjects it may be tinged with blood.] Usually the disease is prolonged over weeks, months, or years, and is not attended by any decided febrile disturbances. A slight pricking sensation

indicates a red spot, where the eruption commences. In the centre of each of these spots the cuticle begins to rise like a blister; and as the circumference of the redness increases, the blister-like vesiculæ are so rapidly formed that, in a few hours, each vesicle may be as big as a hazelnut or walnut. The *blebs* are then apt to burst, when a straw-colored serum exudes; and the epidermis contracts into folds or wrinkles, the surface underneath being red, painful, and smarting. If the vesicles do not burst, small brownish, flat crusts form. The disease is usually associated with debility, intemperance, and bad or insufficient food. The following varieties are to be distinguished: (a.) *Pemphigus acutus*; (b.) *Pemphigus chronicus*; (c.) *Pemphigus solitarius*; and (d.) *Pemphigus foliaceus*.

*Pemphigus foliaceus* commences on the front of the chest, and when fully developed covers the whole body. It is almost always fatal.

**Treatment.**—Dietetic and tonic, especially quinine and the mineral acids. Arsenic is said to be of use in obstinate cases. Emollient lotions, with or without opium, are useful local applications.

[The irritation may be relieved by keeping applied a compress wet with a lotion composed of a drachm, each, of oxide of zinc and prepared calamine, two drachms of glycerine, and enough rose-water to make an eight ounce mixture.]

#### ECZEMATOUS INFLAMMATIONS.

*Eczema*, in some of its many forms, is one of the most common of skin diseases. The eruption, when at its height, is characterized by—(1.) Infiltration of the skin; (2.) Exudation on its surface; (3.) The formation of crusts; (4.) Itching. Eczematous skin feels thick when pinched up into a fold. It has a doughy feel, and on pressure the redness disappears, and shows a yellowish hue of the skin. The exudation is watery, sometimes purulent, and sometimes mixed with blood. It stains and stiffens underclothing with which it may come in contact. The exudation becoming concrete, forms crusts, and their appearances vary with the character and nature of the exudation. The itching is always aggravated by touching the part, and still, at the same time, an irresistible desire to scratch is excited. Scratching is sometimes thus persevered in till blood flows from the part. The disease is thereby greatly aggravated—a copious eruption of newly formed vesicles and fissures being apt to form.

The elementary lesions of the skin in eczema are—(1.) An erythematous state of the skin; (2.) Vesicles; (3.) Pustules; (4.) Papules; (5.) Fissures; (6.) A mixture of several or of all of those lesions; and in the fully expressed disease there may not be the merest vestige of either a vesicle, a pustule, or a papule, but the skin is red and smooth on the surface, having a brilliantly polished and shining appearance, the tissue being loaded with infiltration. The vesicles, when they occur, are usually developed at the orifices



of the cutaneous follicles. They are small and closely set together: they usually rupture early, and the serosity concretes into crusts. The more the skin is infiltrated the less likely are vesicles to be seen. In the absence of vesicles, when the disease is at its height, the infiltrated patches are red and inflamed. The redness is not uniform: it is studded with innumerable points of a deeper red, giving a punctated appearance to the part (DEVERGIE). These points correspond to the orifices of the glands, like the vesicles which may have preceded them; and the appearance is due to the greater congestion of the skin which surrounds the glandular orifices. Minute excoriations are apt to occur at these spots, the result of rupture of the vesicles. From these excoriated points serous fluid continues to exude, which afterward concretes into scabs (*eczema vesiculorum*). The *impetigo* of authors is the pustular form of *eczema*, and the pustules form upon erythematous patches of skin, just as the vesicles did before them. They are most common on the head and chin. Vesicles and pustules are often seen on the same patch (*eczema impetiginodes* or *eczema pustulosum*).

*Lichen* is the papular form of *eczema*, the eruption commencing as small red papulæ, either isolated or confluent. These becoming excoriated, give vent to a serous fluid in considerable abundance, which ultimately concretes into a crust. If the summits of the papulæ become torn by the nails in scratching, yellow or blackish crusts form, due to the mixture of blood and serum, and abrasions of the skin occur. The disease is then known as *prurigo* (*eczema lichenoides* or *eczema pruriginosum*). It occurs more or less in all chronic cases of *scabies* or *phthiriasis*, and sometimes in *urticaria*. The disease attacks by preference the scrofulous and the debilitated, and is especially associated with improper, insufficient or bad food. A too liberal diet, and stimulants in food or drink, similarly predispose to the disease. Exposure of the skin to the heat of the sun, or to acrid substances, also brings about the disease—*e. g.*, in cooks, grocers, bakers, and the like. Fissures of the skin are a most frequent complication, and occur in those situations where the skin is naturally thrown into folds, as at the arms, the angles of the mouth, the joints, the palms of the hands, and flexures of the fingers. They are apt to increase in depth as the disease continues, sometimes bleeding, and causing excruciating pain. When deep, the fissures are red and raw-looking, and serum or blood exudes from them, giving rise to crusts which partially fill them up (*eczema rimosum* of T. M. Anderson and Buchanan—the *eczema fendillé* of the French).

Any part of the skin may be the seat of *eczema*; but there are certain localities where it is most apt to occur. These are the head, the hairy portions of the face, the lips, the edges of the eyelids, the nostrils, the external auditory passages and ears, the hands, feet, legs, genitals, perineum, *mammæ*, umbilicus, and parts of the skin which are naturally in contact with one another.

On the head and hairy parts of the face the disease occurs most frequently in the pustular form (*impetigo capitis*); and collections of pus are apt to form beneath the crusts and scabs, attended sometimes by little cutaneous abscesses and enlargements of the lym-

phatic glands. Each pustule of eczema is situated at the orifice of a hair follicle; and the hair may be seen to pass through the centre of each pustule. These pustules dry up into small yellow crusts when the disease affects the hairy parts of the face; and the skin on which the pustules are developed assumes a dusky red tint, becoming gradually more thickened and infiltrated (*impetigo menti—eczema pilare faciei*). In the *eczema* of the internal auditory passages the disease usually affects the auricles, commencing on the skin of these parts, and gradually extending inwards. The calibre of the meatus is narrowed, and often so much so that it is impossible to see the *membrani tympani*. A milky or watery fluid exudes from the meatus, and moistens the pillow at night. The discharge is apt to have a very bad odor if the ear is not frequently washed out (Anderson, *Med. Times and Gazette*, June, 1863).

Treatment of eczema must be constitutional in the first instance; and local applications suited to the nature of the part affected are to be carefully used. Derangements of the digestive organs must be especially rectified. If the tongue is loaded, the appetite bad, the liver torpid, as indicated by light, clay-colored evacuations and costive bowels, small doses of gray powder in combination with quinine are indicated. Dr. T. M. Anderson suggests the following:

R. Sulphatis Quiniae, gr. xij; Pulv. Rhei, gr. xxxvj; Hydrarg. c. Cretâ, gr. xxx; Sacchari Albi, ʒj; misce, et divide in Pulv. xij.

Two of these powders are to be taken daily by an adult; but at any age the dose must be so adjusted that the patient has at least one full natural evacuation daily. If the patient is robust, but with the functions of the liver and bowels impaired, occasional doses of *calomel*, alone or in combination with *scammony*, will stimulate the torpidity of the digestive organs; and at the same time the cutaneous inflammation will diminish. If the patient is a full feeder, and will not be persuaded to live more sparingly, *one drachm to two drachms of the sulphate of magnesia* may be given twice daily, with *a sixth to half a grain of tartar emetic* added to each dose. The effect of this remedy will be to diminish desire for food, and at the same time keep the bowels freely open. If the patient is scrofulous, or debilitated from insufficient food, or food not nutritive enough, more nourishing food must be given, combined with tonics containing iron and cod-liver oil. Children a few months old, reduced to "skin and bone," recover wonderfully under the influence of twenty drops of the syrup of the iodide of iron in a teaspoonful of cod-liver oil repeated three times daily, the dose of the oil being gradually increased to a tablespoonful (ANDERSON, *l. c.*, June 27, 1863). *Syrup of the phosphate of iron*, or of the *phosphates of iron, quinia*, and *strychnia*, should be alternated with the *iodide of iron*.

The treatment must be steadily maintained for at least six weeks or two months. In severe cases the oil may be rubbed into the skin, in addition to giving it internally. Cod-liver oil and iron are almost equally serviceable to eczematous adults. When there is a difficulty of taking it in its pure state, the emulsion of it will often

be found useful ;—when the appetite is very deficient, a pure tonic, such as *quinine* and *aromatic sulphuric acid*, as in the following :

R. Sulphatis Quiniæ, gr. xvj ; Acid. Sulphurici Aromatici, ℥iv ; Syrupi Limonum, ℥ss. ; Infus. Cascarillæ, ad ℥vij ; *misce, et cola per chartam*. A tablespoonful to be taken three times a day half an hour before food.

If the eczematous patients are robust and plethoric, the local abstraction of blood by leeches or scarification is often beneficial ; but in general the action of *calomel purgation* is sufficient to reduce the inflammatory action, combined with a regulated abstemious diet of mixed animal and vegetable food simply dressed. Dishes of pastry, pickles, spices, strong tea, and coffee, are particularly to be avoided ; while the use of wine, spirits, and malted liquors must be entirely suspended.

If the patient has been much in the habit of using stimulants, the eczema will be much more difficult to subdue than if he had been an abstainer from these fluids. It is sometimes necessary to prescribe milk diet for a time, all animal food being proscribed.

Of internal medicines, arsenic, sulphur, and the alkalies are especially useful (STARTIN, ANDERSON). Fowler's solution (the *liquor arsenicalis* of the Edinburgh Pharmacopœia) is well suited. An adult may commence with *five* minims thrice daily ; and at the end of a week the dose should be increased by *one drop* every second or third day, till the disease begins to yield, or the medicine to disagree (ANDERSON). "In order to secure the virtues of arsenic as an alterative, it will be necessary to push the medicine to the full development of the phenomena which first indicate its peculiar action on the system. Arsenic as a remedy is too often suspended, or altogether abandoned, at the very moment its curative powers are coming into play. The earliest manifestation of its physiological action is apt to be looked upon as its poisonous operation ; and the patient declares that the medicine has disagreed with him. Forthwith the physician shares his fears ; the prescription is changed, and another case is added to the many in which arsenic is said to have failed after a fair trial of its efficacy" (Begbie, *Contributions to Practical Medicine*, p. 270). *Arsenical solutions* should be given immediately after food ; and in persons whose digestive organs are weak, a tonic infusion, such as the infusion of *cascarilla* or *gentian*, is the best vehicle for its administration ; and a few drops of morphia may sometimes be added, as in the following :

R. Solut. Fowleri ; Solut. Muriatis Morphicæ, āā ℥ij ; Syrupi Limonum, ℥ss. ; Tinct. Cocci Cacti, ℥ss. ; Infus. Cascarillæ, ad ℥xij ; *misce*. A tablespoonful of this mixture to be taken thrice daily immediately after food.

The *liquor sodæ arsenias*, in doses of five to ten minims, is said to cure eczema with less gastric disturbance and with less irritability of the conjunctiva than the *liquor arsenicalis*. As the disease yields, the dose may be diminished ; but the use of the remedy should not be suspended till some time after the complete removal of the erup-

tion. In the case of infants at the breast, arsenical solutions may be given to the mother; and *mercury may be combined with the arsenic, as in Donovan's solution, of which ten minims may be given thrice daily.* The use of the natural mineral waters which contain sulphur is the best method of securing the full effects of the alterative action of sulphur on eczematous patients. These waters are especially Harrogate and Moffat in this country, [and the Virginia springs, and those of Richfield, N. Y., in the United States. Sulphur may be efficiently administered in from one-half to one drachm doses of the precipitated preparation, with the same quantity of tartrate of soda or bitartrate of potash, and ten grains of the bicarbonate of soda or potash, in a cup of milk, every night.] *Alkalies* are most beneficial in those cases where the patient has been in the habit of taking stimulants; and when there is a tendency to acidity of the stomach, and to the deposit of *lithates* in the urine, or to gout, or to rheumatism, *aqua potassæ may be given, largely diluted with water, in doses of twenty minims three times a day.* Dr. Anderson recommends *sesquicarbonate of ammonia* (now called the *carbonate of ammonia* in the *British Pharmacopœia*), in doses gradually increasing from ten to thirty grains. If gout prevails, *colchicum* ought to be given with the other remedies; and in rheumatism the *acetate* or *bicarbonate of potash* in half-drachm doses should be added to each dose. All the alkaline remedies should be largely diluted with water.

With regard to local treatment, the first point is to remove all sources of irritation, and especially the crusts. A poultice composed of *crumbs of bread* and *hot almond oil* applied to the eruption at night will usually bring away the crusts in the morning. If they fail to be detached by this application, they must be again lubricated with fresh almond oil, and forcibly removed when they are thoroughly softened. If, after the crusts are removed, the surface is seen to be acutely inflamed, and if there is a *burning heat* in place of *itching*, local sedatives must be applied. For this purpose Dr. Anderson recommends a cold potato-starch poultice, a small quantity of powder containing camphor being sprinkled over the inflamed surface before the poultice is applied. The camphor powder may be composed of the following ingredients:

R. Camphoræ, ʒss.; Alcoholis, q. s.; Pulv. Oxydi Zinci; Pulv. Amyli, āā ʒiij. This powder must be kept in a stoppered bottle (ANDERSON).

Instead of poultices, a mixture of powdered oxide of zinc and glycerine, in the proportion of half an ounce of the one to two ounces of the other, will be found to be a soothing application; and to it a little camphor may also be added, as in the following:

R. Camphoræ, ʒij; Pulv. Oxydi Zinci, ʒss.; Glycerinæ, ʒij; Cochinillini, gr. ij; Spt. Rosmar., ʒj; misce. This mixture must be stirred before using it, a thin layer being then rubbed over the inflamed part twice or thrice daily (ANDERSON).

[When the itching is troublesome, stuping with decoction of bran, or applying an ointment composed of thirty drops of chloroform in one

ounce of cold cream, will generally relieve it. If there is much weeping, mucilage of starch should be applied morning and evening, and afterwards the strong tincture of iodine. A good ointment in eczema proper is:

R. Plumbi Iodidi, gr. xij-xx; Chloroform, ℥xl; Glycerinæ, f3j; Unguent. Rosæ, 3vij.]

When the disease passes into a chronic stage, as indicated by the disappearance of the burning heat and the supervention of itching, the local applications must vary with the condition of the parts. If there be much *infiltration* of the tissues, treatment by potash applications is the most efficient (HEBRA, ANDERSON). The more extensive the disease the *weaker* the solution ought to be for application over the large surface. If the infiltration is slight, common *potash soap* (soft soap, black soap—*sapo mollis*, *sapo viridis*), or a solution of *one part of it in two of boiling water*, a little oil of rosemary or citronella being used to conceal the odor, may be used. A piece of flannel dipped in this liniment should be rubbed as firmly as possible over the affected parts night and morning, the solution being allowed to dry upon them. It should, however, be washed off after each application. If the patient can bear it, a piece of flannel, saturated with the solution, may be left in contact with the part all the night.

[In inveterate cases Hebra uses his *Tinctura Saponis viridis cum pice*:

R. Saponis Viridis; Picis liquid.; Spir. methyl. (alcohol mixed with one-ninth of its bulk of pyroxylic spirit); āā partes equales.]

Another method of treatment is to paint over the eruption night and morning with a large brush, charged with the *aqua potassæ* of the *Edinburgh Pharmacopœia*, its irritant action being neutralized by means of cold water when the smarting becomes excessive. A solution of *potassa fusa* may also be employed of various strengths. If the case be mild, *two grains to an ounce of water*; if more severe then *five, ten, twenty, thirty, or even more grains to the ounce of water* may be used. *But all these stronger solutions must be washed off very speedily, and they ought not to be employed more than once daily.* Such remedies the physician ought to apply himself, and not intrust the control of their action to the patient. If there is a tendency to the formation of fissures, which are apt to result from the use of potash, *cod-liver oil* or *glycerine* should be applied to the parts every night. As infiltration subsides, the solution ought to be gradually diluted; and great caution is necessary in using such local applications upon infants, delicate females, and old or infirm persons. The affected parts should be bathed repeatedly during the day in cold water, during the use of these *potash* applications. The *cold douche* may be employed where it is practicable. If the itching is very intolerable, *prussic acid* may be mingled with the potash applications, as in the following:

R. Potassæ Fusæ, gr. x; Acid. Hydrocyan. Dil. (Ed. Ph.), ʒj; Aq. Rosarum, ʒij; misce. A little of this solution is to be rubbed firmly over



the eruption night and morning, and at any time when the itching sensation is severe.

*Tarry applications* are of great value in the local treatment of the *declining stages* of eczema, when infiltration and itching are subdued. Common tar (*pix liquida*) or oil of cade (*oleum cadini*, as manufactured at Aix-la-Chapelle) should be rubbed firmly over the eruption by means of a flannel cloth, and allowed to dry upon it. It should be applied thrice daily, and washed off with *soft soap* or *petroleum soap*. Dr. Anderson recommends that common tar should be combined with one of the potash solutions—for example, a mixture of *equal parts of common tar, methylated spirit, and soft soap*, used as above directed, will be found useful, or the following preparation, as less offensive:

R. Saponis Mollis, Spt. Rectificati, Olei Cadini, āā ʒj; Olei Lavandulæ, ʒiss.; misce. A little quantity is to be firmly rubbed over the eruption night and morning; and it must be washed off before each re-application.

[Or it may be combined with iodine: R. Iodinii, ʒi-ii; Olei picis (sp. grav., .853), fʒj. M. Apply with a camel's hair pencil.]

Of *mercurial applications* the *citrine ointment* (*Ung. Hyd. nit.*) is the best, or *the ointment of the iodide of mercury*. They may be used of their full strength, or diluted with lard, according to the indications of the case, care being taken that *mercurialism* is not induced. In using ointments, a small quantity should be melted on the finger, and rubbed firmly into the affected part; and none should be allowed to lie undissolved upon the skin; and the part should always be cleaned with soap and water before re-application. Epilation should be resorted to in cases of eczema of the hairy parts of the face. In very mild attacks, or with a view to prevent the recurrence of the disease, the skin may be washed occasionally with soft soap and water. Hendrie's "dispensary petroleum soap" is recommended by Dr. Anderson, to whose admirable lectures on "Eczema," in the *Medical Times and Gazette* for May, June, and July, 1863 (since published in a separate volume), the reader is referred for more detailed information regarding the nature and treatment of this inveterate skin disease.

*Acne* affects the sebaceous follicles of the skin generally, or those at the roots of the hairs of the beard. The disease generally appears as pimples; but suppuration usually very slowly follows: there may be persistent redness after suppuration is at an end, giving a blotchy appearance to the part. The parts most frequently affected are the temples, cheeks, nose, and forehead; but it also appears on the neck, shoulders, and front of the chest. The first occurrence of the disease is often indicated by the collection of morbid material in the sebaceous follicles, which open on the skin by a blackish point, to which the vulgar give the name of "*grubs*" or "*worms*" in the skin. The tubercles on which the classification of this disease is sometimes based are merely the terminations of the pustules, and not an elementary lesion. The accumulation of the matter secreted

by the sebaceous follicles tends to keep up the eczematous inflammation in them.

**Treatment.**—Local and general measures require to be combined. Diet, in the first instance, should be restricted. Wine, spirits, and coffee are to be refrained from. Milk is to be used as a drink, and as an article of diet, combined with light food, fresh vegetables, and succulent ripe fruits. Emollient applications, such as an emulsion of bitter almonds, a decoction of bran or quince seeds, and tepid milk are useful. Dr. Anderson has found one or other of the two following lotions particularly serviceable; and generally if one fails, the other succeeds:

R. Sulphuris, ʒj; Spt. Rectificati. ʒj; misce.

The mixture is to be shaken before using it; or,

R. Hyd. Corrosiv. Sublim., gr. ix–xij; Hydrochl. Ammoniaë, ʒss.; Cochinillini, gr. j; Aq. Rosæ, ad ʒvj; misce.

Whichever of these lotions is used let it be applied by dipping a piece of flannel into the lotion, and rubbing *very firmly* over the eruption night and morning.

To promote resolution of the induration, *iodide of sulphur*, in the proportion of *fifteen or twenty-four grains to an ounce of lard*, is of great service in dispelling the tumors. Drastic purgation ought to be avoided. Simple baths at a temperature of 88° or 90° Fahr. are of service.

[No treatment of acne is so promptly benefiting as Mr. Startin's,—touching the apices of the enlarged follicles with minute drops of the acid nitrate of mercury, applied by a fine glass-brush; a little roll of blotting-paper should be used to absorb all excess of acid. No soap should be used on the affected parts, which should be bathed with hot water, or with a solution of carbonate of soda, or aqua ammoniaë, one drachm of either to a quart of water; the following lotion to be used afterwards:

R. Olei Limonis, fʒij; Olei Rosmarini, fʒss.; Glycerinaë, fʒij; Alcoholis, fʒxiv. In chronic cases, half a grain of corrosive sublimate may be added to each half ounce. The constitutional treatment should be iron, bitter tonics, arsenic, and sulphur.]

*Ecthyma* is characterized by large round pustules, generally distinct, and seated upon a hard inflamed base. The pustules are succeeded by dark-colored scabs, which leave superficial cicatrices behind them, or red stains, which disappear after a time. The eruption of ecthyma is common on the neck, shoulders, buttocks, extremities, and chest; and is seldom developed on the face or scalp. The typical appearance of ecthyma may be seen in the pustules produced by friction with *tartar emetic* ointment. The pustules continue for several days, and are succeeded by scabs, which begin to form in the centre. The disease may occur at any age or season, but it most frequently appears during spring and summer in young adults. The *ecthyma cachecticum* of Willan prevails in old, irritable persons, much addicted to alcoholic fluids.

[*Rupia escharotica*, the most severe variety of ecthyma, is met with in youth or middle life, and in depraved constitutions, and begins as a dull red or livid spot, on which a bleb, which soon becomes filled with dark sanious matter, is soon formed. When this bursts, or is accidentally broken, a deep, ill-looking ulcer is seen, with an offensive secretion, and over which a dark-colored scab may form. In children its usual site is the genitals, legs, or scalp, and it may be attended with severe constitutional symptoms. In the adult it often appears on the nose, sometimes rapidly destroying an ala.

The crusts of pemphigus, particularly in early life, have a strong resemblance to ecthyma, but the latter begins as a tubercle, while pemphigus is essentially vesicular in its origin, and as its crusts fall off they are more apt to leave rough and reddish surfaces than an ulcer.]

**Treatment** is chiefly by diluents, simple and emollient baths, and regulation of diet. Moderate exercise should be taken, combined with the use of alkaline or salt-water baths. Mild laxatives are beneficial, and spirits, wine, and beer are to be refrained from. The food should be as nourishing as can be digested. Tonics, such as quinine and iron, are also indicated. A stimulating lotion composed of *muriatic acid* diluted with water, is of use to brush over the parts and promote cicatrization.

[In *Rupia escharotica* the system must be supported by full diet, stimulants, and quinine.]

*Psoriasis vulgaris*.—*Psora leprosa*, *Lepra vulgaris*, or *dry tetter*, [the *leprosy* of the Bible (Lev. 13: 2),] is characterized by the development of irregularly formed patches, slightly raised above the level of the skin, and covered with thin, dry, white scales. The patches may be distinct, small, and scattered; or larger, confounded together, and irregular; or they may be so extended as to make a continuous surface. Hence the names of varieties of psoriasis—*guttata*, *diffusa*, *gyrata*, *inveterata*. [Dr. McCall Anderson describes a variety which he calls *psoriasis rupioides*, because of the large conical crusts, marked by concentric rings.] The intense itchiness and the eruption are always preceded and accompanied by that form of dyspepsia, or impaired digestion, in which there is a superabundance of acidity, much formation of lithates, and an obvious constitutional tendency to gout. Anæmic persons, and those in whom the circulation is languid, with a dry skin, are those in whom the disease is prevalent. Sometimes it is localized in patches, as on the back, between the shoulders, the lips, the eyelids, the palms of the hands, the scrotum, or the pudenda. Sometimes it seems hereditary. Intemperance, the use of highly salted food, fish, or indigestible substances, are apt to induce an attack. The depressing passions, anxiety and grief, are also often followed by *psoriasis*.

[Mr. A. B. Squire has submitted that lactation is an exciting cause of psoriasis (*Med.-Chir. Trans.*, 1866). It is more common amongst the better than the poorer classes. Mr. C. Wilson believes that it is always due to syphilitic poison; whether this be true or not, it is a diathetic disorder, and most often associated with deficient vitality.

It would appear from the microscopical observations of Dr. J. Neumann, of Vienna, that psoriasis is essentially a nutritional change in the upper layers of the corium and papillæ, with an abundant cell-growth, causing hypertrophy of the papillæ. The excessive growth of epidermis is due to the active deposit of cells in the Malpighian layer, furnishing new material as fast as the epidermis scales off. (*Wochenblatt der Gesellschaft der Wiener Aerzte*, No. 30, 1867, and *Virchow's and Hirsch's Jahresbericht*, 2d band, 1868, p. 535).]

**Treatment.**—Preparations of arsenic are found to be of great service in this form of eczematous inflammation. A form of arsenical remedy known as the Asiatic pill is recommended by Cazenave. Its composition is as follows:

R. Arsenici Protoxidi, gr. j; Pip. Nig., gr. xij; Pulv. Acac., gr. ij; Aq. Destill., q. s.—Divide in pil. xij vel xvj. One pill to be taken once, twice, or thrice daily, after meals.

[Fowler's solution is probably the best mode of administering arsenic; but the doses usually given are too large. Three or four minims is a maximum dose, which should be reduced gradually to one or even half a minim. There is no doubt that arsenic is the most generally valuable of all drugs in the treatment of skin diseases, and in psoriasis it seems to have special efficacy. As the diathetic character of this class of affections is becoming recognized, the action of arsenic in their management is better understood. The general condition is improved, and the patient gets rapidly healthier. But the course must be a long one, extending over many months, and continued for some time after all eruption has disappeared; otherwise there is danger of relapse. To do this the doses must be small to begin with, and then decreased, and maintained at the point of toleration. The moment it disagrees it must be stopped, to commence again with a diminished dose, to be again intermitted for awhile, and so on. It should always be taken during, or immediately after, a meal. Used in this way it will rarely disappoint.

A good form for the administration of arsenic in psoriasis, particularly if there is a suspicion of syphilitic taint, is Neligan's *ioduretted solution of the iodide of potassium and arsenic*:

R. Liquoris Potassæ Arsenitis, fʒj; Potassii Iodidi, gr. xvj; Iodinii, gr. iv; Syr. Florum Aurantii, fʒij. M. From one-half to one teaspoonful to be given twice or thrice daily, at meal-time, in a little water, or bitter infusion.

The writer has found nothing to answer so well or so speedily in psoriasis palmaris, as corrosive sublimate in doses of one-sixteenth of a grain, alone, or with two to three grains of the iodide of potassium, and keeping the palms covered with glycerine.

Dr. T. W. Belcher, of Dublin, has called attention to the fact, in many instances a true one, that in the treatment of scaly diseases by arsenic or iodine, the local appearances are aggravated; the irritation is greater, and the scaly desquamation increased; but in a little while this passes away, and steady improvement follows.]

But it is of the greatest importance that the meals and the diet should be regulated. Small doses of *pilulæ hydrargyri*, or of *pilulæ*

*calomelanos composita*, or *hydrargyra c. cretâ*, may be given at bed-time for a few days, and followed each morning by a *drachm* of *magnesia*, given in combination with a teaspoonful of *lemon-juice*. The vascular excitement of the stomach is best subdued by dilute hydrocyanic acid, in the following formula (A. T. THOMSON):

R. Potassæ Carbonatis, ℥ss.; Succī Limonis Recentis, f℥iv; Acid. Hydrocyanici Dilutis, ℥iv; Vini Seminū Colchici, ℥xv; Aquæ Destillatæ, f℥vj; *misce. Fiat haustus 4ta; quâque horæ sumendus.*

The tone of the stomach is then to be restored by small doses of *bicarbonate of potassa*, with from twelve to fifteen minims of the *tincture of henbane*, in a fluid ounce and a half of the *infusion of cinchona*. The eruption and itching disappear as the mucous membrane of the stomach and bowels return to a healthy state. The diet should be absolutely free from stimulants; and tepid baths, at a temperature of 96° Fahr., used every morning for half an hour, are most serviceable. The bowels should be regularly relieved by such mild measures as the following formula for pills:

R. Pilulæ Hydrargyri, gr. vj; Pulveris Ipecacuanhæ, gr. vj; Extracti Colocynthis Comp., gr. xij; Ext. Hyoscyami, gr. xvij; *misce. Fiat pilulæ duodecem. Sum. ij, h. s. quotidie* (A. T. THOMSON).

Glycerine and emollient lotions are useful local applications. I have often seen the itchiness greatly relieved by the following lotion of bitter almonds rubbed night and morning over the parts affected:

R. Amygdal. Amar. Numero xx; Aquæ Rosæ, ℥vij. *Contunde et tere simul, dein cola, et adde Hyd. Corrosiv. Sublim., gr. xij; Ammonia Hydrochloratis, gr. xij; Spt. Vini Rect., ℥j.*

[Carbolic acid is now much used in this complaint.

R. Acid. Carbolici (Calvert's), gr. xiv; Alcoholici, f℥ss.; Glycerinæ, f℥j; Aquæ, f℥xivss. M.

A lotion of one drachm of dilute nitric acid in eight ounces of water will often prove useful. Itchiness may be abated by a mixture of borax, one drachm; alcohol, half a fluid ounce; chloroform, one-half to one drachm, and water enough to make eight ounces. A weak alkaline solution—soda or ammonia—is frequently relieving].

#### PHLEGMONOUS INFLAMMATIONS.

Of the phlegmonous inflammations of the skin, erysipelas may be considered the type. Its pathology has been already described (vol. i, p. 325).

#### NEW FORMATIONS.

*Warty Disease of the Skin.*—*Ichthyosis* involves the whole tissue of the skin, and is characterized by the growth of thick, dry, imbricated scales of a dirty gray color, resting upon a perfectly uninfamed surface, and never accompanied by pain, heat, or itching.



It appears principally on the external parts of the limbs, round the joints, on the knee and elbow, on the upper part of the back, and generally on those regions where the skin is thick and coarse. The disease is generally congenital, and lasts during life. It may not be strongly marked at the period of birth; but the skin appears dull, thick, and fretted, and the disease is developed as the infant grows older.

**Treatment.**—Remedial measures are only palliative, consisting mainly of mucilaginous and glycerine lotions, with vapor baths to mollify the roughness. Potash solutions or other preparations of potash are contraindicated as local applications.

*Cheloid, or Keloid.*—A tuberculated growth or swelling of the true skin, originally described by Alibert, subsequently by the late Dr. Addison, of Guy's Hospital, and Mr. Thomas Longmore, Professor of Military Surgery at the Army Medical School.

The keloid of Alibert is a fibrous tumor of the skin, often developed on a cicatrix; whereas true keloid, as described by Dr. Addison and Mr. Longmore, is a much more extensive lesion. The true keloid begins as a few prominent red tubercles, generally over the sternum—whence it may extend gradually to the sides of the body and the back. The red tubercles gradually increase in size, and coalesce into larger growths. These growths then send forth spur-like processes on every side; a slight puckering of the healthy skin surrounds the marginal limits of the bases of the tumors, and marks the advance of the disease. The spread of the disease is very slow and gradual, extending over months and even years. Intense itching and irritation accompany its advance, more particularly when warm in bed at night, or during hot weather. Continued cold causes the growths partially to disappear, leaving an old cicatrix-like condition of the skin (*Med.-Chir. Trans.*, 1863).

**Treatment** is not yet defined; but the history of the disease points to cold applications and tonic remedies as the basis of a rational treatment.

[Alibert defined and described **Keloid** as a cancerous affection, characterized by the presence of an excrescence—or, sometimes, of several excrescences—more or less prominent, hard and elastic when compressed by the fingers, cylindrical, or round, or quadrilateral, flattened at the centre, raised at the edge in the form of a rim, throwing out from its sides processes like roots, which are implanted in the skin, so as oftentimes to present the appearance of the cicatrix of a burn (*Monographie des Dermatoses*, 1835). Except that the disease is now known not to be of a cancerous nature, there is scarce anything to be added to this description. Alibert distinguished a *false* and *true* form of the disorder; the former originating spontaneously, and the latter growing upon a previously acquired cicatrix. Professor Longmore's case was of this latter kind (*Med.-Chir. Trans.*, vol. xvi, p. 105), and came on the exact spot, over the supra-spinous fossa, where the man had received nearly fifty lashes.

The keloid of Addison as described by him had its original seat in the subcutaneous areolar tissue. It was at first indicated by a white patch,

very slightly, if at all, raised above the level of the skin, and unattended by any inconvenience. Sometimes, when the patch was of much size, its surface was mottled by a faint yellowish or brownish tint. The disorder might here stop, the spot disappearing; or, after awhile, give rise to itching, pain, or tightness, at the same time hardness being traced along the course of the neighboring tendons or fasciæ. At length the affected part was hidebound, the tendons and fasciæ being rendered incapable of performing their functions, and to such an extent that the whole of a limb, but especially the fingers, might be permanently bent, and be as hard and immovable as a piece of wood. While these changes were going on, the cutis and cuticle became more affected, the skin shrinking, with a dry, smooth, or glistening aspect, and of a reddish, pinkish, yellowish, or a dead leaf, color. The cuticle exfoliated, and the cutis became liable to ulceration, with subsequent scabbing; or it was covered by obscure tubercular or nodular elevations—the whole appearance resembling the remains of an extensive and imperfectly cicatrized burn. On some part of the discolored and shrivelled skin, reddish, elevated, claw-like processes, from half an inch to two inches long, and penetrating the adjacent sounder tegument, occasionally appeared, having an exact resemblance to those described by Alibert as being characteristic of his true keloid. Frequently there are scattered over the surface oval, flattened, tubercular-looking elevations, hard to the touch, and without other discoloration than might be attributed to accidental irritation (*Med.-Chir. Trans.*, vol. xxxvii, 1854).

It seems very probable, from the recent observations of Dr. C. Hilton Fagge, of London, that "Addison's keloid," instead of being a distinct disorder, is, in fact, but a variety of the affection, or group of affections, known to the French and Germans as *Sclerodermia*, *Scleroma*, or *Scleriosis* (Virchow), and to which attention was first called by Thirial, under the name of *Sclérème chez les adultes*, in 1845 (see a translation of Rasmussen's paper on Sclerodermia in *Edin. Med. Journal*, Sept., 1867). Dr. Fagge had an opportunity of seeing a patient who had been under Dr. Addison's care with this disease, and of whom two drawings had been taken at that time, some twelve years afterwards, and he found the disease had to a very great extent disappeared. Many parts at which the skin had been much diseased, had almost become natural, the tegument being quite flexible and soft. At other parts distinct contraction had taken place. In another case, in which the affection existed in a marked degree, in eight months a great change for the better had occurred; at several points where the skin had been white, shining, and indurated, it had recovered its suppleness, and all that remained of the disease was a yellowish-brown discoloration. There was at these points no sign whatever of that cicatriform appearance on which so much stress has been laid. Dr. Fagge, from these cases, and from reading the histories of the similar cases recorded by other observers, is led to the conclusion that the natural tendency of the so-called keloid of Addison is to terminate in the way just described, thus making the prognosis much more favorable than it is generally given. With regard to the—

**Treatment**—Dr. Addison thought that iodine would most likely check its progress, but in three cases treated in Guy's Hospital, both iodine and iodide of potassium were tried without benefit; nor did liquor potassæ, arsenic, or mercurial inunctions seem to be of more use. Two of them bettered slowly when taking no medicine. Cod-liver oil, quinine, and iron, have been given in cases which improved or got well (MOSLER, SEDGWICK, HILLIER). Warm baths have been of service, as well as the

inunction of oily and fatty matters. Mosler in two of his cases used an ointment of two drachms of the black oxide of copper, one drachm of glycerine, and one ounce of lard. In one of Addison's, liquor plumbi subacetatis best relieved the itching.

With regard to "Alibert's keloid" Dr. Fagge states, that Mr. Hilton had known of at least one case where there was no recurrence after excision (*On Keloid, Scleriosis, Morphæa*. By C. Hilton Fagge, M.D. *Guy's Hosp. Reports*, 3d series, vol. xiii, 1868). Also the very full report of a case of Scleroderma, by Dr. Paulicki, of Hamburg, in *Virchow's Archiv*, for May, 1868, and *Edinburgh Medical Journal*, October, 1868, p. 366. The author compares at great length his case with those recorded by other writers.]

*Molluscum*.—This disease consists of numerous small tubercles, varying in size from that of a pea to a pigeon's egg. They are round, flat, and irregular in shape, and sometimes seated on a broad base, or attached to a peduncle. They grow very slowly, and may last through life, occurring chiefly on the face and neck, but may cover the whole body. Writers are not agreed as to the true nature of this disease. A contagious variety has been described by Bateman, Carswell, Dr. John Thomson, and Dr. Robert Paterson, of Leith, whose excellent and erudite paper, in the *Edinburgh Medical and Surgical Journal*, No. 148, will well repay a careful study.

**Treatment**.—The tubercles of molluscum seem to diminish under the use of arsenic. Sulphate of copper, solid or in strong solution, effects the destruction of the tumors with sufficient rapidity; but caustic potass is by far the most speedy and effectual method of cure (ROBERT PATERSON).

## [APPENDIX I.]

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### APPENDIX TO DISEASES OF THE NERVOUS SYSTEM.

(DR. CLYMER.)

#### PROGRESSIVE MYO-SCLEROSIC PARALYSIS—PSEUDO-HYPERTROPHIC MUSCULAR PARALYSIS.

Syn., LATIN, *Lipomatosis Luxurians Musculorum Progressiva; Atrophia Musculorum Lipomatosa*. FRENCH, *Paralysie Musculaire Pseudo-Hypertrophique; Paralysie Myo-Sclérosique*. GERMAN, *Progressive Muskelhypertrophie*.

**Definition.**—*A progressive disease of childhood, characterized at the outset by weakness of the lower extremities, and a peculiar wabbling gait; later there is more or less enlargement of the paretic muscles, particularly the gastrocnemii, followed at a variable period by atrophy and total palsy of the muscles. Until the last stage the general health is good; there are no symptoms showing lesion of the cerebro-spinal centres; the anatomical character is, probably, hyperplasia of the interstitial muscular connective tissue, with subsequent fibroid substitution.*

**History.**—On December 9th, 1851, a paper was read by Dr. Edward Meryon, "On Granular and Fatty Degeneration of the Voluntary Muscles," in which was detailed the case of a child, who, born healthy and well-proportioned, walked late, and then heavily and clumsily, until eight years of age, when his walking gradually worsened, and at eleven years he could not walk at all, and could scarcely stand. When he was twelve, the power of the muscles of the upper extremity was lessening also, notwithstanding that the muscular mass of the body and limbs did not appear to have diminished; on the contrary, the child had grown well and had gained flesh. He died, æt. 14, of a mild attack of fever, accompanied with nausea, giddiness, and a constant and profuse secretion of mucus in the larynx and trachea, and apparent paralysis of the pharyngeal muscles. On examination after death, the spinal cord and membranes were perfectly natural, but all the voluntary muscles were atrophied, soft, and, although the muscular fibres *appeared* to exist, they were almost bloodless. Under the microscope, the striped elementary fibres were found to be completely destroyed, the sarcous element being diffused, and in many places converted into oil globules and granular matter, whilst the sarcolemma was broken down. The degeneration was most advanced in the muscles of the abdomen and legs. Dr. Meryon goes on to state that three brothers of this patient, born later, were all similarly affected. He likens the affection pathogenetically to fatty disease of the heart described by Dr. Richard Quain. Dr. Meryon further mentions two cases related to him by Dr. W. Bullar, of Southampton, which from their clinical history, and the post-mortem appearances of one, were prob-

ably similar. The fact of "the calves being larger than natural," and having during the progress of the paralysis become permanently contracted, is particularly noted. He adds, moreover, that two more cases of a like kind had fallen under his notice during the year. Plates of the diseased muscle-tissue in the first case are given.\* Dr. T. K. Chambers reported a kindred case, in connection with mollities ossium, in 1854.†

In 1861, Dr. Duchenne (de Boulogne) published his first case, under the name of *paraplégie hypertrophique congénitale*, and observed by him in 1858 (*Electrisation Localisée*, 2d edition, 1861). He has since seen and reported‡ eleven cases; 1 in 1859, 1 in 1860, 2 in 1861, 4 in 1862, 1 in 1863, 1 in 1865, and 1 in 1867. Dr. Bergeron has reported 1 case under his care in the St. Eugénie Hospital, 1867.§ The following cases have also been published: Schutzenber, of Strasbourg, 1, 1862;|| Jaksch, 1, 1862;¶ Berend, 1, 1863; \*\* Eulenberg and Cohnheim, 1, 1863; †† Wernich, 2, 1864; ‡‡ Oppolzer, 1, 1865; §§ Griesinger, 1, 1865; ||| Heller, 4, 1865; ¶¶ Meryon, 2, 1866; \*\*\* Seidel, 3, 1867.††† At the meeting of the Pathological Society of London, November 27th, 1867, Dr. T. Hillier reported a case, since published in his work on the Diseases of Children, p. 254, and Mr. W. Adams one, to appear in the Proceedings of the Society for 1867-68.

**Symptoms.**—There are three stages of this disorder: (1.) Weakness of the lower extremities (paresis); lateral balancing in walking, keeping the legs widely apart; sacro-lumbar curvature, or arching forwards (cordosis), in standing and walking; bilateral equinism (equino-valgus of both feet), with persistent rigid flexion of the toes. (2.) Apparent hypertrophy of the muscles of the legs, particularly the calves, of the back, and the glutei; they are large, elastic, but very firm. This stage is stationary generally for awhile, and may last for several years. (3.) Generalization and progressive aggravation of the paralysis, with muscular atrophy. The following case, given by Dr. William Hillier,‡‡‡ is a good picture of the disorder in the third stage:

A boy, aged 11 years, of healthy parentage. He began to walk at 21

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\* Medico-Chirurgical Transactions, vol. xxxv, 1852. The chief points have been particularly given, because Dr. Duchenne (de Boulogne) scouts the idea of Dr. Meryon having first described this affection (la priorité de la découverte). "*L'honneur de cette découverte appartient tout entière à la France: (that is, to himself) c'est une question aujourd'hui jugée.*" In the same paper he says: "*la découverte de la paralysie pseudo-hypertrophique remonte à l'année 1858,*" (p. 6.)

† "The portion examined of the rectus muscle was, to the naked eye, of the natural color, but of too homogeneous an appearance. Under the microscope, it presented no appearance at all of fibrous structure, or even linear arrangement. It had become a mere congeries of fat-vesicles, the interspaces between which were filled up with globular granular corpuscles, estimated from 1 to 2-5000ths inch in diameter; many of the larger had a granular nucleus."—*Medico-Chirurgical Transactions*, vol. xxxvii, 1854.

‡ Archives Générales de Médecine, t. i, 1868.

§ Gazette Hébdomadaire de Médecine et de Chirurgie, No. 23, 1867.

|| Gazette Médicale de Strasbourg, No. 5, 1862.

¶ Prager Vierteljahrschrift, 1862.

\*\* Berlin Allgemeine Medicinische Central Zeitung, No. 9, 1863.

†† Verhandlungen der Berlin. Med. Gesellschaft, 1863; Berlin. Klin. Wochenschrift, 1866.

‡‡ Deutsch. Archiv für Klin. Med., 1863.

§§ Medicin. Jahrbücher, 1865.

||| Archiv. für Heilkunde, 1865.

¶¶ Deutsch. Archiv. für Klin. Med., 1865-67.

\*\*\* Medico-Chirurgical Transactions, vol. xlix, 1866.

††† Centralblatt, 1867. Die Atrophia Musculorum Lipomatosa, Jena, 1867, 8vo, and 2 Lith., p. 102.

‡‡‡ Diseases of Children, p. 254. London, 1868.



months of age; could never walk well, waddling very much with his legs apart; he has never been able to run or jump; frequently tumbling when attempting to go quickly. His calves were always large and out of proportion to his other muscles. The power of walking since he was three years old, instead of improving has gradually declined, till for six months past he has been quite unable to walk, and cannot now raise himself from his bed or stand unless supported. His heels are drawn up, and lower part of spine arched forwards when he attempts to stand. Lately his upper limbs are failing, so that he cannot cut his food or grasp anything firmly. He has complete control of his sphincters. The intellect is not very clear, nor yet very deficient, and is reported to be slow in learning. The upper limbs and muscles generally are very thin. The calves are still of fair size, but they have a peculiar doughy feel. There is no localized atrophy of any part.

The muscular hypertrophy appears at a variable period after weakness of the lower limbs, usually from a few months to one year. In Eulenberg's case it came on at the age of ten, first in the muscles of the thigh, then in those of the legs, particularly the gastrocnemii, and finally in the triceps brachialis, the affection having begun at five years of age by the usual symptoms of paresis. In Oppolzer's case, a considerable period from the outset elapsed before the muscular development was apparent; when the gastrocnemii contracted they formed two masses nearly the size of a man's fist. Though in most instances the hypertrophy is limited to the muscles already enumerated, it may involve many others; in Bergeron's case it invaded the muscles of the face, particularly the temporal, those of both extremities, and of the trunk, except the great dorsal, the pectoral, and the sterno-mastoid. The boy at ten years of age presented the appearance of an exaggerated Farnesian Hercules, weighing thirty-four kilogrammes, equal to about seventy-nine pounds. The hypertrophy is bilateral, most often beginning in the muscles of the calf of the leg, other muscles becoming subsequently affected. In one case, and that doubtful, the paresis and hypertrophy were seemingly coincident (DUCHENNE).

In the larger number of instances there are no morbid cerebral phenomena. One of Duchenne's cases was an idiot, and another nearly so. Yet often the intellect is sluggish, and the speech hesitating. The body-heat and circulation are unaltered; even the temperature of the affected limbs is natural, as well as the capillary circulation. No pain is complained of, and there is neither hyperæsthesia nor anæsthesia, deep-seated or cutaneous, of the paralyzed legs. The functions of the bladder and rectum are usually intact; in fact the patients appear to be in good health. Electro-muscular contractility is uncertain; it may be retained throughout, or it may be diminished, or it may vary at different times in the same case.

The course of myo-sclerotic paralysis is progressive; the functional troubles of the first stage worsen during the second—that of false hypertrophy; there is then commonly a pause for several years—two, three, or even more—when sudden and rapid aggravation happens, the paralysis extending to the upper extremities—if at first it was limited to the lower—the use of the arms is lost, the muscles generally become atrophied, the patient emaciates, and, having reached adolescence, is forced to keep a lying or sitting position. Finally, health fails, and some intercurrent disorder, as disease of the lungs or bronchi, kills before adult age is attained.

**Patho-anatomy.**—The brain and spinal marrow have been examined but once in this affection, and that by Cohnheim in Eulenberg's case (1865).

The microscope could detect no tissue-change. With regard to the muscle-structure, in Griesinger's case, Bilroth removed during life a small piece of muscle from the left deltoid, which was hypertrophied and paralyzed; the primitive fasciculi were whole, but they were shoved apart by a large quantity of adipose deposit. There was no trace of fatty degeneration in the fibre. Wernich took a small portion of the gastrocnemius from a lad, of eleven years, suffering from this disease, and examining it microscopically, saw nothing structurally abnormal, but large masses of fat were deposited between the fasciculi. Heller found the fatty vesicles in such quantity in the interstitial tissue, that he proposes to call the disease *lipomatosa luxurians*. Eulenberg and Cohnheim found the fibres in some of the damaged muscles much lessened, to the extent of from a fifth to a sixth of their natural size, and also little masses which seemed to be empty sarcolemmæ; throughout there was fatty matter, which in some places could not be distinguished from subcutaneous adipose tissue. In all of Dr. Duchenne's cases, in which the muscles were examined after death or during life, there was considerable hyperplasia of the connective tissue, along with fibroid substitution, whether the muscles were in the stage of hypertrophy or not. In the second stage, the muscles were very pale; the transverse striæ were extremely reduced, and sometimes had disappeared in part or wholly; the sarcolemma seemed to contain fatty vesicles, which in reality came from the interstitial connective tissue, and which were essentially different from the fatty granulations which characterize fatty muscular degeneration. From these observations Dr. Duchenne concludes, that the fundamental essential anatomical lesion of myo-sclerotic paralysis is, hyperplasia of the interstitial connective tissue, with more or less fibroid substitution, together with a variable amount of fatty vesicles. The increase of size of the affected muscles is due to this proliferation of the cellular and nuclear elements of the connective tissue, the one being in direct proportion to the other.

The difference between the observations of the German and French pathologists is, that the former found the sarcous elements but little changed, but large masses of interstitial fatty matter in the muscles; whilst the latter found the chief alterations in the intimate muscular structure itself. Very convincing evidence of the exactness of the microscopical observations of the French observers is given in Dr. Duchenne's paper.

**Causes and Pathogeny.**—This disorder is exclusively of childhood. Dr. Duchenne, in an experience of twenty years, during which his attention has been chiefly directed to this class of affections, has never seen any analogous disorder in the adult. If the paretic troubles did not exist in any degree at birth, in many of the cases they manifested themselves soon afterwards, or were first noticed when the child began to walk. In some of the recorded cases the first symptoms were observed at 5 years (DUCHENNE, HELLER, GRIESINGER), at 11 (WERNICH, HELLER), at 13 (DUCHENNE), and at 16 (HELLER). Males seem to be much more liable to it than females; of 12 cases 11 were boys (DUCHENNE). Hereditary influence is incontestable. Dr. Meryon has reported four cases in one family, and two cases each in two other families. Dr. Wernich mentions two brothers similarly afflicted; Heller four in a family; and Seidel three. In Heller's cases it followed exposure to cold and dampness, but in none of Duchenne's cases did this influence appear as even an exciting cause. In one of the latter's cases convulsions preceded the outset; and another happened after an attack of eruptive fever. None of the subjects seem to have had rickets.

The pathogeny of this affection is not understood. Up to the present time no appreciable lesion of the cerebro-spinal centres or nerves has been seen. The sympathetic does not appear to have been examined. The early muscular troubles cannot be attributed to tissue-changes; for they precede them, and are at no time in direct relation to them. Duchenne writes: The formative irritation, which produces the abundant nuclear proliferation and other changes in the interstitial connective tissue of the muscles, seems to be the probable cause of the paresis. But how explain the change itself in the connective tissue? This is still an unsolved problem (*loc. cit.*, p. 573). The Germans place it nosologically amongst constitutional disorders. As a disease involving an error of nutrition, it is probable that it is some portion of the sympathetic system which is at fault.

**Diagnosis.**—The following tabular statements, condensed from Duchenne's paper, show the diacritic symptoms of progressive myo-sclerotic paralysis, and those affections with which it may be confounded.

#### PROGRESSIVE FATTY MUSCULAR ATROPHY OF INFANCY.

1. Begins in the muscles of the face, particularly the orbicularis oris and zygomatic. After two or three years it invades the muscles of the trunk, and its course is the same as in the adult—from above downwards; affecting first the muscles of the upper extremities and of the body, and extending to those of the lower limbs later.

2. The muscles atrophy partially and irregularly, one after another; muscular debility in a limb is not general, but limited to the affected muscle or muscles, and is in direct proportion to the degree of atrophy; the consequence of which is, the several partial muscular paralyses and the various deformities in the muscles of the trunk.

3. The muscular fibre undergoes granular or fatty degeneration, and interstitial fatty substitution.

4. Muscular motility is not abolished until after the tissue-changes.

#### FATTY ATROPHIC PARALYSIS OF INFANCY.

1. Begins in a large majority of cases with febrile symptoms.

2. May be generalized, paraplegic, bilateral, or crossed, limited to one limb, or a portion of a limb at the outset.

When all the muscles are completely paralyzed from the outset, those whose innervation is the least damaged soon recover their power, whilst the others become greatly atrophied, or their texture is changed in different degrees.

3. In the first stage electro-muscular contractility is lessened or absent, although there may be no muscular tissue-change.

#### PROGRESSIVE MYO-SCLEROTIC PARALYSIS.

1. Begins, as a rule, in the lower limbs, whose muscular power is weakened; its course is from below upwards, the upper extremities and the muscles of the trunk becoming involved after a considerable interval of time.

2. Muscular weakness from the outset affects a large body of muscles, connected with several joints; later some of the parietic muscles, and more rarely all of them, become apparently hypertrophied.

3. The muscular interstitial tissue shows a remarkable proliferation of the elements of connective tissue, with abundant production of fibroid tissue, and more or less fat globules. Striation of muscular fibre generally exists.

4. Muscular motility is lessened or abolished.

#### PROGRESSIVE MYO-SCLEROTIC PARALYSIS.

1. Apyrexia throughout the course of the disorder.

2. At the outset, paresis affects the muscles of the lower limbs and the extensors of the spine, and their power is weakened.

At a later period the power of motion is totally lost.

3. In the first stage electro-muscular contractility unimpaired; (this is doubtful, and requires further observation to confirm it).

4. At an advanced stage the loss of motility is soon followed by atrophy, more or less rapid and complete, according to the degree of the nervous lesion.

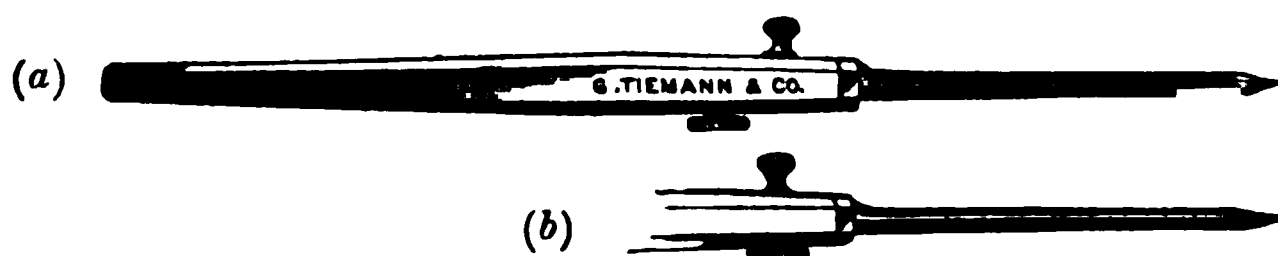
5. At an advanced period the loss of voluntary and electro-muscular contractility, shows that the muscles, whose innervation has been damaged, have undergone serious tissue-changes.

4. Paresis is soon succeeded by a considerable increase in the size of a greater or less number of muscles.

5. In the third stage of this disorder, if the enlargement of the muscles has disappeared and the limbs seem atrophied, a microscopic examination of a portion of an affected muscle taken from the patient, shows that the primitive fibres have, generally, preserved their transverse striation, and that the proliferated connective tissue is mingled with fibroid substitution and fatty vesicles, in greater or less quantity.

The symptoms of progressive myo-sclerotic paralysis would seem to be sufficiently distinctive to prevent an error of diagnosis by those practically acquainted with the different forms of infantile paralysis. A microscopic examination of the tissue of an affected muscle is, however, always desirable in this affection, and it can be readily done by the harpoon of Middeldorpf, or still better by the trocar (Fig. 83), (*emporte-pièce histologique*) recently (1865) devised and used by Dr. Duchenne.

FIG. 83.



This instrument is composed of a shaft, and of two half cylinders of steel, one of which is made to slide on the other by pushing a button forward. The fixed half cylinder has a small cavity near its pointed end. The skin is drawn tensely aside, and the trocar held by the three last fingers, the extremity of the index being placed so as to regulate the depth of the wound, it is plunged open (a) perpendicularly down to the muscle; the button at the under part of the handle is pressed forward; this forces the half-cylinder against the shoulder (b); a small portion of muscle is detached and caught in the cavity, and the instrument is withdrawn; the button is drawn back, and the bit of suspected tissue taken out for microscopical examination. The pain is slight, if the operation is adroitly done, and no ill consequences seem as yet to have followed its use.

**Prognosis.**—Of immediate danger to life in the early stages, there is little risk. In most instances the disorder pursues a regular course, the patient dying after two to five years from exhaustion and some acute intercurrent disease. All Duchenne's early cases perished in this way; but recently he has seen the disorder arrested in the first stage in two instances, and a cure follow. He argues that if it were more frequently detected and treated at the outset, which is not the case from the slightness of the early paretic symptoms escaping notice, the prognosis might be quite favorable. This is probable, as myo-sclerotic change has not yet happened to any extent.

**Treatment.**—Duchenne uses localized faradisation,\* shampooing (mas-

\* Faradisation is the local application of electricity, induced by a voltaic or a magnetic current passing through a coil of wire, and frequently interrupted. The best instrument is that of Stöhrer of Dresden.

sage) of the affected muscles, and hydrotherapy. In the second stage, when tissue-change had occurred, this treatment was only temporarily relieving, and the disease resumed its course, going on to a fatal ending. He also tried strychnia, ergot, iodide of potassium, but with no good result.

The paralyzed muscles should be galvanized for five minutes at a time, three or four times a week. The constant current of low tension should be used in connection with faradisation. Means with a view of improving general nutrition, regimenal and other, should not be neglected, and the writer would suggest the use of arsenic in the early stages.

### THE PHYSICAL DIAGNOSIS OF THE DISEASES OF THE CEREBRO-SPINAL SYSTEM.

It is generally conceded by those whose attention is directed especially to the study of the diseases of the nervous system, that the diagnosis of this class of affections may be materially helped from the investigation of physical conditions, existing in organs beyond the great nerve centres, and known to be present in many cerebro-spinal disorders, by means of certain instruments of precision. By the use of these, the relation of the observed phenomena to given morbid states of the brain and spinal cord is ascertained, and their symptomatic value estimated. The conditions to be particularly noted, are perversions of function in the skin, and in the muscles, altered thermal conditions of certain parts of the body, and tissue-changes occurring in the interior of the eye. These will be considered under the several heads of (1.) Perversions of Contactile Discrimination;\* (2.) Perversions of Muscular Power; (3.) Perversions of Temperature; (4.) Functional and Structural Perversions in the Eye.

(1.) **Perversions of Contactile Discrimination.**—The researches of Professor Ernest H. Weber† showed the discriminative power which the skin possesses of recognizing double impressions made simultaneously upon it within a given limit, but varying in different parts of the body. His mode of ascertaining this was to touch the surface with a pair of mathematical compasses, the points of which were protected with pieces of cork. The text-books on physiology give a summary of the results obtained by Weber. It will be enough to say here that, at the tip of the tongue and the palmar surface of the third phalanx of the fingers, the distance between the legs of the instrument seemed greater, though really so much less, than in less sensitive parts; in the tongue, at the point above mentioned, contactile impressions from the two applied points being distinctly appreciated if the average distance between them was only  $\frac{1}{2}$  of an inch; whereas at the middle of the forearm, thigh, and back they must be, in a natural condition, thirty lines apart, before being discriminated.

Weber's laws were first applied, as methods of detecting and measuring

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\* This term was proposed by Dr. John W. Ogle (*Beale's Archives of Medicine*, vol. i, p. 323), for Weber's expression, "tactile sense," as giving more understandingly the analysis of the so-called general tactile sense, and recognizing its division into four subordinate tactile senses: 1st. Temperature; 2d. Pain; 3d. Muscular action; and 4th. Contactility, as shown by Landry, and others.

† *De pulsû, auditû, resorptionê, et tactû.* Leipzig, 1834. At page 44 of a chapter, entitled *De subtilitate tactûs diversâ*, there is a section headed: *Experimenta de distantia duorum corpora organon tactûs simul tangentium recte perceptâ.*

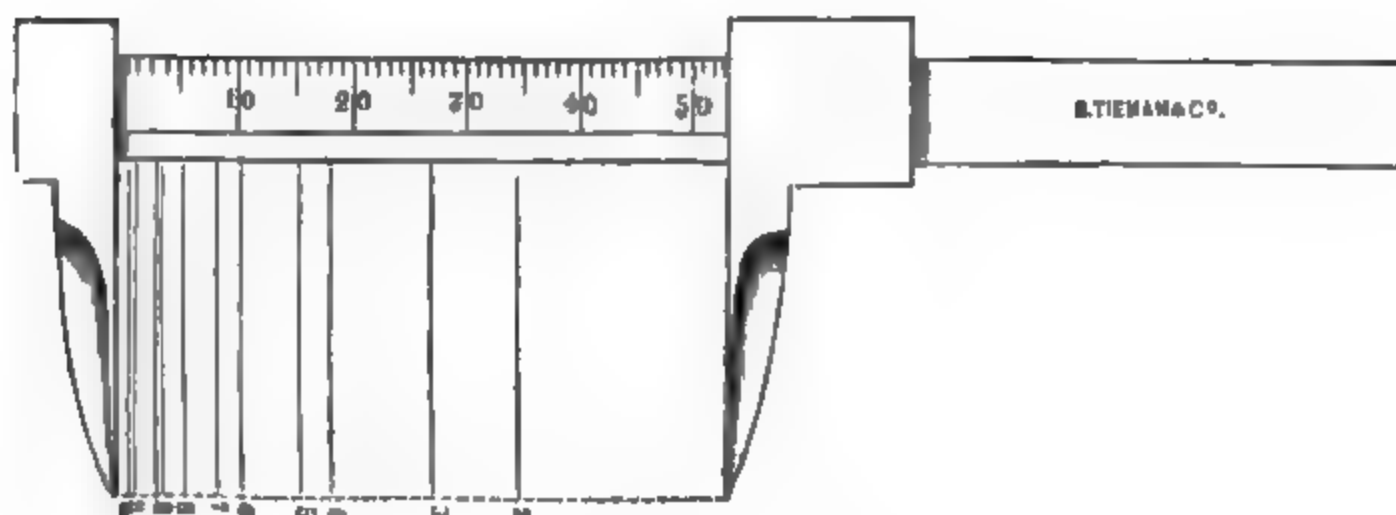


various degrees of contactile sensibility in diseases of the nervous system, by Dr. Brown-Séquard in 1849,\* and is called *æsthesiometry* (*αἰσθησις*, sensibility, and *μέτρον*, a measure). By the aid of this method (a) Any slight decrease or increase of contactile sensibility may be readily recognized, and which may not be discoverable by any other means; (b) The degree of decrease or increase may be exactly measured; and (c) Changes in the degree of existing hyperæsthesia or anæsthesia, may be accurately ascertained.

Dr. Brown-Séquard first used a pair of common mathematical compasses; but in 1858 he imagined and had made an ingenious instrument, which he called an *Æsthesiometer*.†

The *æsthesiometer* of Dr. Brown-Séquard (Fig. 84) is a decimetrical rule, graduated to millimeters; at one end is a fixed branch; another similar, but fixed, branch slides on the rule, marking the distance at which the points begin to be felt when applied to the skin. It is furnished with a handle, which may be used or not. To apply it, it is held by its flat sides, at a point about its centre of gravity, between the thumb and the three last fingers, whilst the index, slightly bearing on the back of the rule, regulates the contact of the points with the skin, which must be in accurate parallelism with the surface.

FIG. 84.



Dr. Sieveking, of London, in 1858,‡ devised another *æsthesiometer*, which is not dissimilar from the instrument, *stangenzirkel*, used by Weber in his physiological researches. The handle of Dr. Sieveking's *æsthesiometer* is shorter than that of Dr. Brown-Séquard's, though the principle is the same. Later (1859), Dr. John W. Ogle, believing that there would be several advantages in an instrument which combined the principle of the beam compass with that of the two-legged compass, had made his *aphemetric compass*.§

In Fig. 85 is represented another *aphemetric compass*, by Mr. Stohlman, of Messrs. G. Tieman & Co., surgical cutlers, of this city. It is the ordinary mathematical compass, provided with a brass scale attached to one of the legs. It is a convenient instrument, but has the fault of being too long. The best instrument is the compass of Dr. Jaccoud, made by Colin & Robert, of Paris, which is only nine centimeters long, with

\* Comptes Rendus de la Société de Biologie, t. 1er, p. 162, 1849.

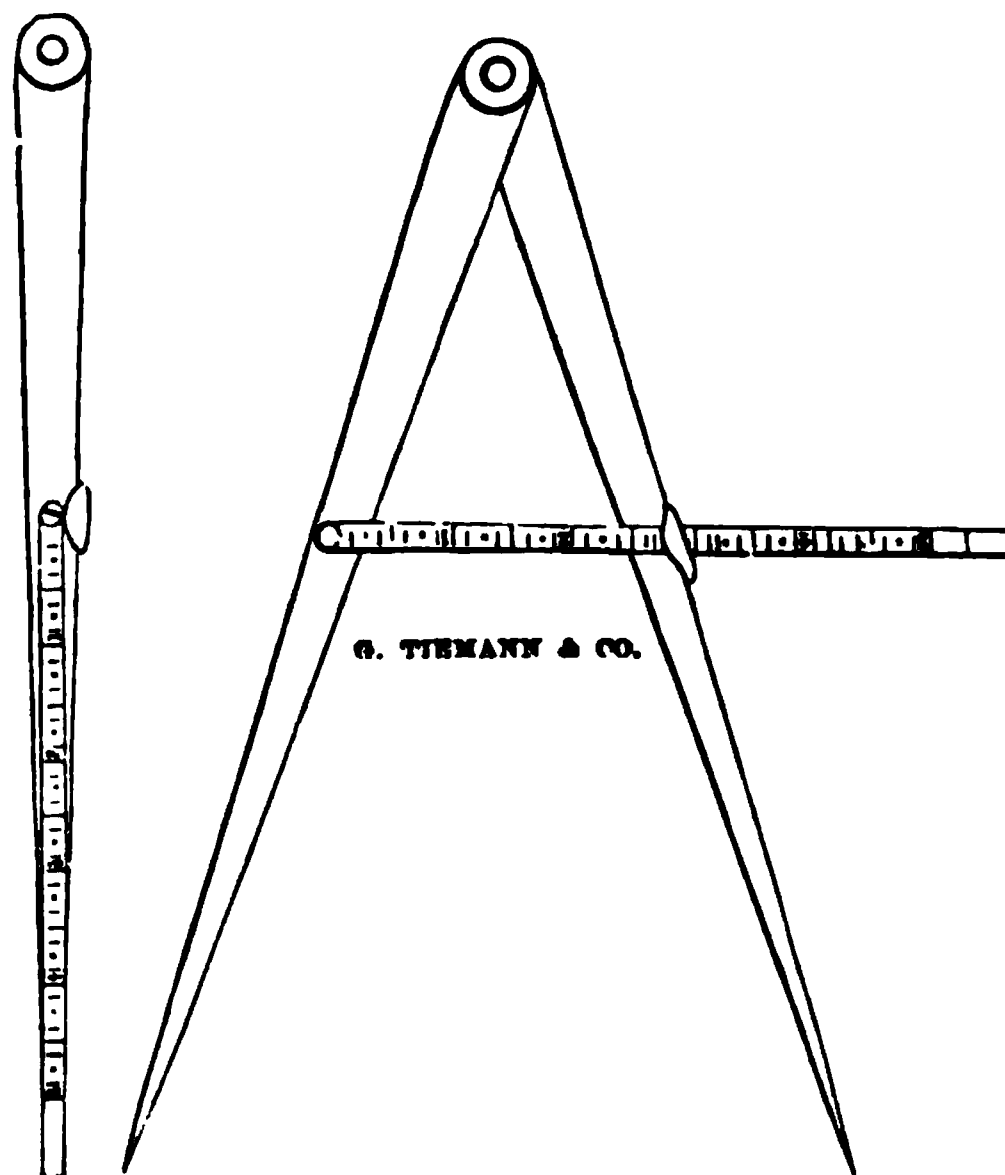
† Journal de Physiologie, 1858, p. 846.

‡ British and Foreign Medico-Chirurgical Review, vol. xxi, p. 280, 1858.

§ Beale's Archives of Medicine, vol. i, 1869.

a movable scale in the form of an arc of a circle, the whole folding up, and easily carried in the pocket.

FIG. 85.



Dr. Brown-Séquard remarks, that for those who treat nervous diseases, the æsthesiometer is as indispensable as is the stethoscope to those who treat diseases of the heart; and there is no doubt that it not only measures the degree of contactile discrimination, and ascertains its perversions, but often aids us in making out the precise site and nature of the disorder, particularly if the disease be at the base of the brain and in the spinal cord.

The following rules are given by Dr. Brown-Séquard in the use of the æsthesiometer (Art. *Æsthésiomètre*, *Dict. Encyclopédique des Sciences Médicales*, t. 2<sup>e</sup>, 1865).

1. The points of the instrument should be covered by pieces of cork, for the slightest prick will interfere with the accuracy of the perception of tactile sensation.
2. The points *must* be simultaneously applied.
3. Though the attention of the patient should be alert, he should not see whether one or both points are applied; nor should the full import of the experiment be made known to him, until after it has been tried. It is well for him to think that sometimes one point only, and again that both points, are applied, so that the degree of his contactile discrimination may be accurately determined.
4. Before beginning an investigation of the parts in which some modification of tactile sensation is suspected, the æsthesiometer should be applied upon a healthy portion of the skin, the two points being so separated that the patient can, at one time, have the impression of both points, and at the other, of one only.
5. It sometimes happens that the patient, knowing that both points are applied, imagines that he feels both, no matter how little the distance between the points. In such instances one point only should be applied, and after he

has asserted that he feels both, let him see that one only has been used. In this way we may be sure that he judges by his sensations, and not by a preconceived notion.

The normal distance-limit between the two points, that is, the limit within which these give only the sensation of one point and beyond it of two, is, as has been stated (p. 986), not the same in all parts of the body, but it scarcely varies for the same district of skin in different persons. This normal distance-limit is also the same, or nearly so, in corresponding parts of the right and left halves of the body.

We begin an exploration of tactile sensibility at the part where we wish to ascertain if it be changed, as well as the degree of change, by applying the two points of the *æsthesiometer* at the normal distance-limit. If the patient feels only one point, we gradually separate the two points from each other, and repeat the application until both are felt. By this means we recognize the existence and amount of *anæsthesia*. If the two points are felt at the normal distance-limit, they are gradually neared, and their application is to be repeated until there is the sensation of one point only. By this method the degree of *hyperæsthesia* is measured. The *æsthesiometer* should not be immediately reapplied upon the same point of the skin, for the feeling of contact often lasts for half a minute, and the patient may, after one or two applications, feel two points when only one is applied. In cases of *anæsthesia* or *hyperæsthesia*, limited to a very small region of the skin, the applications should not be repeated in less than half a minute.

In *anæsthesia* of considerable extent, the two points may be applied one after the other after an interval of forty or fifty seconds, and yet give the sensation of only one point: this is due to the slowness of the transmission of the impression; and the *æsthesiometer* in this way may mark the rapidity of the transmission of tactile impressions.

When the degree of *anæsthesia* is great, the two points of the *æsthesiometer* are felt only as one, no matter how great the space between them, provided that they are applied upon the same longitudinal line. Brown-Séquard has known one point to be put upon the wrist, and the other upon the forearm, and yet there was the sensation of one point only.

Cutaneous *hyperæsthesia* may be of such an amount that, however close the two points may be, both continue to be felt. In a case of chronic spinal meningitis, tactile sensibility was so much heightened that the patient felt both points applied to the thigh at the distance of one millimeter apart, whilst in health, there should have been a space of from five to six millimeters between them for both to have been distinctly felt.

To avoid the mistake of finding either *anæsthesia*, or *hyperæsthesia*, when there is really neither, it should be borne in mind that *strychnia* increases tactile sensibility, whilst *belladonna* lessens it. If then it is necessary to find out the exact state of the cutaneous sensibility of any one part of the body in patients using either of these drugs, the state of tactile sensibility in the healthy parts should be first explored, and the actual normal type of the individual under the influence of these drugs be ascertained.

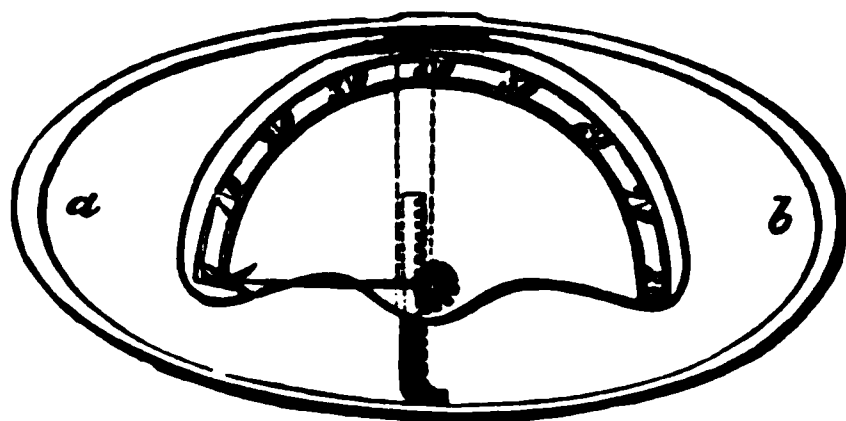
In certain cases when two points of an *æsthesiometer* are applied, there is a distinct sensation of three points; or two points may be felt when one only is applied. In the instances in which this perversion of contactile discrimination has been met with, there has always been inflammation, or congestion, sometimes the result of an intra-cranial tumor, at the base of the brain, and especially in one of the cerebral peduncles, or in one of the

lateral halves of the annular protuberance. The sites of this phenomenon are the face, neck, and the hand, but particularly the face, for in nine cases in which it was observed in six it was limited to the face. When the distance between the two points of the instrument was two centimeters, two, or, sometimes, only one, point was felt; but at two and a half or three, and even at four, centimeters, there was a distinct sensation of three points touching the skin (BROWN-SÉQUARD, *Archives de Physiologie*, t. 1<sup>r</sup>, 1868).

(2.) **Perversions of Muscular Power.**—Muscle is the instrument, and not the producer, of force, the genetic factor of all muscular power being in the nervous system. Hence damage to the nervous centres is followed by derangement of muscular function—diminished, or total loss of, power being one of the most common consequences of cerebral and spinal disease.

To measure the strength of paralyzed muscles of the upper extremity, the *dynamometer*, of Mathieu, a surgical cutler of Paris, will be found a useful instrument. The usual plan of making the patient grasp the physician's hand gives an imperfect indication, nor does it allow of any correct estimation of changes for better or for worse in the progress of the palsy. This simple instrument consists of an elliptical steel spring, to which is attached a semicircle of gilt brass, upon which a scale is marked. An indicator, with a small cog-wheel at one end, may be moved freely around the arc of the circle by a steel arm, upon one side of which cogs are cut, fitting into those of the indicator. The lower end of this arm touches the elliptical spring, when the indicator points to zero on the scale; a brass sheath on the under side of the scale keeps this arm in place, at the same time letting it move freely (Fig. 86).

FIG. 86.



When the dynamometer is taken into the hand and pressed, the two sides of the spring are approximated, and the steel-arm, the cogs being pushed by the lower side of the spring, turns the indicator. When pressure is taken off, the indicator does not return to 0, but remains at the point to which it has been carried by the muscular power of the patient.

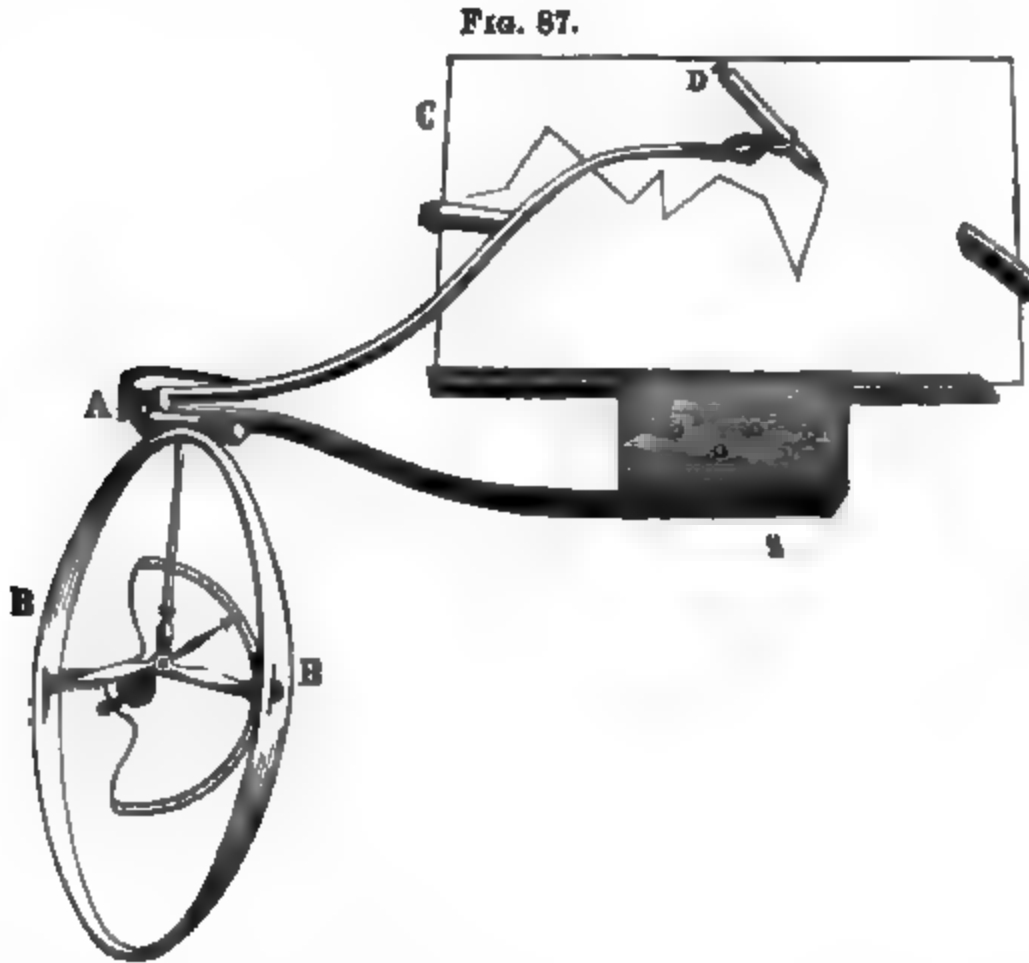
It may also be used to measure tractile force, for if two hooks with cords attached be fastened to the spring at the points *a* and *b*, traction on the cords will approximate the two sides of the ellipse, and thus push the steel arm so as to move the indicator.

Dr. W. A. Hammond has found it useful in determining the effect of electricity upon the irritability of paralyzed muscles, and thus enabling the physician to decide whether the proper time for its use has yet come, by observing whether the strength of the muscles is increased or diminished after the application of this agent.\*

\* *Quarterly Journal of Psychological Medicine*, vol. ii, p. 141, 1868.

The writer is indebted to the Editor of this Journal for the use of the cuts of Figs. 83, 85, 86, 87, 88, 89, 90, and 91, he having been the first to use and describe the dynamometer and dynamograph, of Mathieu, in this country.

By the *dynamograph* of Mathieu (Fig. 87), the degree of muscular power and tone are recorded.



A toggle-joint moving on a steel rod is attached to the dynamometer, B, B. This steel rod plays through a hole in the end of the elliptical spring, and moves the lever, which raises the pencil D. At A is a screw by which the point where the rod touches the lever is varied, thus increasing or lessening the delicacy of the indications. C is a silvered plate upon which the paper is fastened by clips. To the lower edge of this plate a strip of gilt brass, with cogs cut in it, is attached. E is a gilt brass box, containing a watch movement like that of a sphygmograph. A cog-wheel, which projects above the upper portion of this box, fits into the cogs on the plate, which carries the paper. The wheel for winding up the clockwork, and the lever for stopping it or setting it in motion, are not seen in the figure, they being on the opposite side of the box.

To set the instrument agoing, the sphygmograph movement is attached to the instrument at A; the clockwork is wound up, and the plate covering the paper placed in the groove on the top of the box E. The patient now seizes the dynamometer in his hand and squeezes it firmly; the lever is moved, and the plate with the paper is carried along by the cog-wheel. As it moves, the pencil traces a line on the paper, the height and regularity of which depend upon the firmness and steadiness with which the dynamometer is squeezed, and by this means the muscular power and tone are ascertained and recorded. The date and the name of the patient should be written on each piece of paper, which may be filed away for reference. The pencil ought to be of the softest lead, and the paper rough and unsized. The following illustrations of the action of the dynamograph are given by Dr. W. A. Hammond, of New York, and are the results of his personal experience with the instrument.\*

\* *Quarterly Journal of Psychological Medicine*, vol. ii, pp. 145, 146.



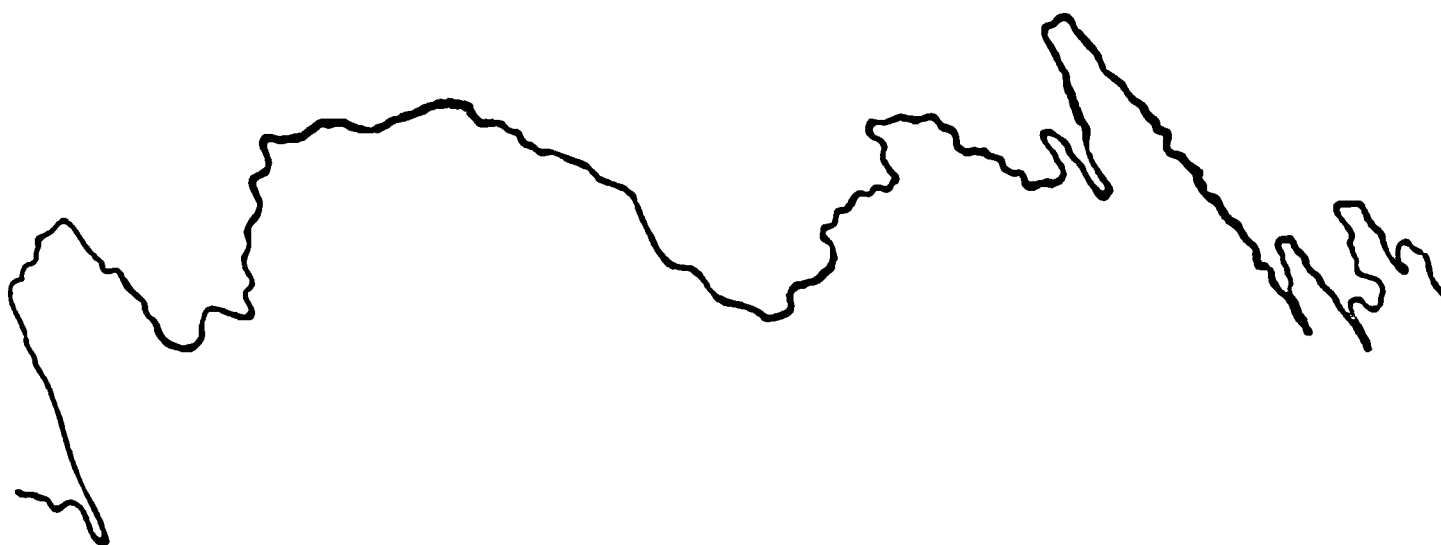
A tracing made by a person unaffected by any disorder of the nervous system involving loss of muscular power, is a straight line. Dr. Hammond found the same tracing made by a patient suffering from locomotor ataxia (Fig. 88).

FIG. 88.



Fig. 89 represents the tracing made by a lady suffering from right hemiplegia with aphasia. Though the line is irregular, there is no actual decline in it, the point of ending being really higher than the point of beginning.

FIG. 89.



A gentleman affected with congestion of the meninges of the spinal cord made the tracings in Fig. 90 with his left hand. There is a progressive fall in the wave line.

FIG. 90.



Fig. 91 shows the tracing made by the right hand of the same patient.

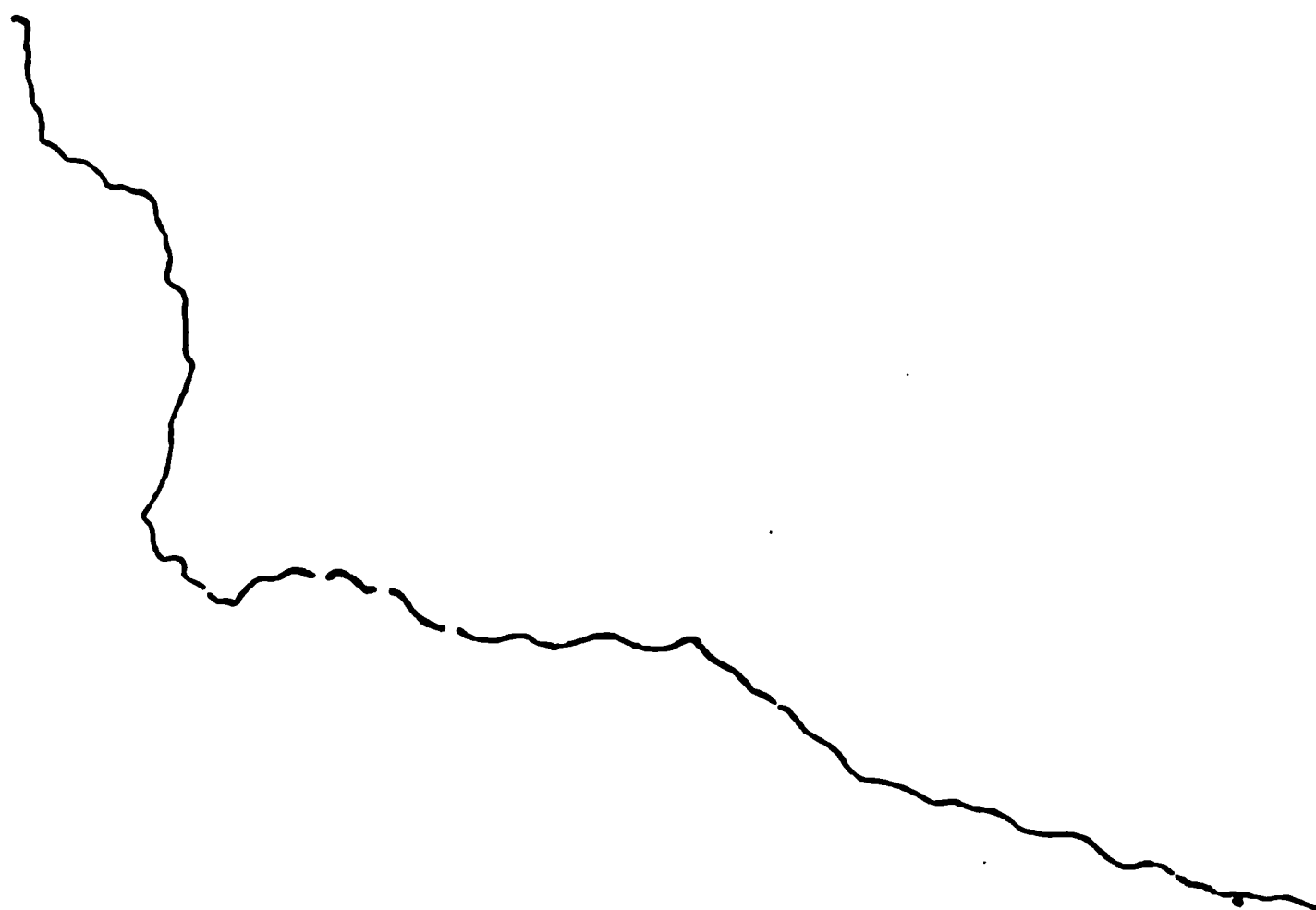
This line is uniformly descending. The right side was much more paralyzed than the left. Every time that the muscular power of this person was tested, the lines he made were similar in general features to the above.

In a case of softening of the brain, attended by general paralysis, the line was irregularly denticulated and descending.

(3.) **Perversions of the Body-Heat in certain parts of the body** — In most cases of hemiplegia, the temperature of the paralyzed side is raised, varying from  $\frac{1}{10}$ ths of a degree to 1 degree; it is rarely natural; and scarcely ever lessened. The difference of temperature may be present in all cases of hemiplegia symptomatic of various cerebral lesions, as hemorrhage, softening, &c. Whatever part of the brain is damaged increased temperature is always noticed, but whether the site of the lesion influences the amount of the increase, is not yet decided (FOLLET,

*Gaz. Hébdom.* t. 1<sup>er</sup>, 1867). The thermometric equilibrium is restored on the cure of the paralysis. The duration of the unilateral rise of the body-heat greatly varies in different cases. In some it persists for years, whilst in others, and particularly when the paralytic attack has not been

FIG. 91.



sudden, it may not last longer than two months. In long-standing cases, however, a time comes, sooner or later, when the thermometric equilibrium is restored, and this happens, Dr. Folet thinks, at the beginning of consecutive atrophy. When, after long-continued hemiplegia with persistent excess of body-heat, the other half of the body becomes paralyzed, the temperature of both sides becomes the same, or an increase takes place on the side last paralyzed. The presence or absence of muscular rigidity has no marked influence upon the thermometric markings. The general body-heat of hemiplegic patients is not as a rule increased, and does not exceed an average of  $92.5^{\circ}$  Fahr., except in the last hours of life, when it may rise to  $94.7^{\circ}$ , or even to  $96.5^{\circ}$ . Well-marked paralytic atrophy is always attended by variable lowering of temperature in the affected parts.

In the several local palsies, dependent upon diseases of the spinal cord, or of the spinal nerves, or connected with nutritional errors bringing on tissue-changes and causing muscular atrophy, the temperature of the affected part is always reduced; and in some cases a return of the temperature towards the natural standard is the first indication of improvement.

It is important then, that in these several nervous disorders, that the temperature be noted. Where the variation is considerable the thermometer will answer, but where it is slight it is important that slight deviations should be detected and accurately measured. For this purpose Becquerel's discs, in connection with a galvanometer, are the best means. They are very thin plates of copper, about the size of a half-dime, two in number, and soldered to a thin rod of bismuth, contained in a small tube of hard rubber, furnished with a handle. One disc is placed on the sound limb, and the other on the corresponding part of the paralyzed

limb. Both are in connection, by means of delicate silk-covered wires, with the poles of a galvanometer. If the temperature of both limbs be the same, the needle of the galvanometer remains at rest; if the heat of either is raised, the needle is deflected to the north or south pole, according as one or other limb has the highest temperature. By this apparatus less than the one-hundredth of a degree can be determined. (*Quarterly Journal of Psychological Medicine*, vol. i, p. 53.)

(4.) **Functional and Structural Perversions in the Fundus of the Eye.**—It has long been known that indications of disorders of the nervous system were to be found in the eye. Since the invention of the ophthalmoscope by Helmholtz, the changes in the optic nerve, the retina, and the choroid, happening in the course of cerebro-spinal disease, have been

FIG. 92.



studied particularly by Bouchut, Galezowski, Hughlings Jackson, Hulke, Allbutt, and others. The importance of ophthalmoscopic explorations, in all cases of cerebral disorder especially, when practicable, as an aid to diagnosis, is now recognized. Indeed, the inner eye of patients suffering from any acute or chronic affections of the nervous system should be examined whether they complain of defect of sight or not. The reader is referred to the various recently published works on the ophthalmoscope for practical directions in using the instrument.\*

The changes in the inner eye, which are known to accompany certain diseases of the brain and of the spinal cord, happen in the optic disc, the retina, the choroid, and their bloodvessels. The optic disc is liable to congestion, to congestion with effusion within or around it, to inflammation of its sheath, to inflammation in its substance, to anæmia, and to atrophy. Fibrinous or fatty patches may be seen in the retina, more

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\* The Lectures on the Use of the Ophthalmoscope, by Mr. Jonathan Hutchinson, in the *London Hospital Reports*, vol. iv, 1868, give a very clear summary by an expert. A valuable paper on the Modes of Examining the Optic Disc with the Ophthalmoscope, illustrated by chromolithographs showing the appearances of the disc in different colored eyes in health and in disease, by Mr. C. Bader, will be found in *Guy's Hosp. Reports*, Third Series, vol. xiii, 1868.

especially in the course of its vessels, and interstitial hemorrhage may occur. Hemorrhage and pigment changes occur in the choroid. And, lastly, the bloodvessels may be diminished, obliterated, or dilated, or be the site of blood-stasis, embolism, thrombus, or they may give way. These affections of the nervo-vascular parts of the eye may be included under the following heads: (1.) Simple hyperæmia of the discs and retinal vessels. (2.) Anæmia of the same parts. (3.) Ischæmia of the discs, and its consequences. (4.) Acute interstitial neuritis, and its consequences. (5.) Neuro-retinitis. (6.) Chronic neuritis. (7.) Simple progressive atrophy.

Dr. E. Bouchut, to whom the profession is indebted for being amongst the first to study and to call attention to the morbid appearances in the eye significant of cerebro-spinal disease (*Du Diagnostic du Système Nerveux par l'Ophthalmoscope*, Paris, 1866), recently communicated to the French Academy of Sciences (June 8, 1868), a paper, in which he has summarized the results of his own observations of the most important optic indications of organic cerebro-spinal disorders.

(1.) Optic neuritis and neuro-retinitis, choroiditis, and papillary atrophy accompany the greater number of acute and chronic affections of the brain and spinal-cord.

(2.) The law of coincidence of optic neuritis and organic lesions of the nervous system, may be explained by the anatomical and physiological relations of the eye with the brain and spinal cord.

(3.) Whenever any positive hindrance occurs in the cerebral circulation, in consequence of a lesion of the brain or spinal cord, there is papillary and retinal hyperæmia.

(4.) When there is acute or chronic inflammation of the brain, it may extend to the eye by following directly the course of the optic nerve.

(5.) Affections of the anterior spinal cord may, on account of the anastomosis of its nerves with the sympathetic, at a level with the two first dorsal pairs of nerves, produce the phenomena of papillary hypertrophy in the eye, followed later by atrophy of the optic nerve.

(6.) Optic neuritis and neuro-retinitis caused by acute and chronic diseases of the nervous system, are generally found in both eyes.

(7.) In affections of the brain and its meninges, optic neuritis is most often more marked in the eye corresponding to the hemisphere most severely affected.

(8.) Changes in the optic nerve and the retina, accompanying disorders of sensibility, of intellect, and of motility, always indicate organic disease of the brain.

(9.) These changes in the optic nerve and the retina should not be separated from other symptoms of the existing disease; their presence, however, is an element of diagnosis of positive certainty.

Ischæmia of the discs occurs in all intra-cranial affections which more or less directly distend the ophthalmic veins, as meningitis, hydrocephalus, and intra-cranial tumors. In meningitis, the exudation at the base of the brain may press upon, or the inflammation may involve, the cavernous sinus, in which case there is only ischæmia. Or the inflammation may creep down the nerve and cause neuritis optici, or it may mainly follow the sheath of the nerve and cause perineuritis; or it may both affect the sinus and the optic vein, and may creep down the nerve; in which case there will be both ischæmia papillæ and neuritis. Bouchut thinks that the changes in the eye often occur early enough to be the first certain signs of the existing disease. He relates a case of tubercular meningitis of the convexity of the brain, whose diagnosis by the general symptoms

The ages were :

1 to 2 years,	. . . . .	8 cases.
2 " 3 "	. . . . .	15 "
3 " 4 "	. . . . .	10 "
4 " 5 "	. . . . .	11 "
5 " 6 "	. . . . .	2 "
6 " 7 "	. . . . .	1 "
7 " 8 "	. . . . .	1 "
		—
		48

The ages of those who recovered were, respectively: 4 years 2 months, 4 years, 4 years 3 months, 3 years, 4 years, 2 years 4 months, 2 years 6 months, and 6 years 5 months.

Dr. E. Krackowizer has performed the operation for croup 55 times in New York, and once in Europe (fatal). Of the operations done in this country, the results are:

Years.	No. of cases.	Recoveries.	Deaths.
1852-60	28	5	18
1861	6	3	3
1862	4	1	3
1863.	6	3	3
1864	5	3	2
1865	6	1	5
1866	2	—	2
1867	3	—	3
—		—	—
55		16	39

In the Hôpital des Enfants Malades, during the year 1862, there were 136 operations of tracheotomy in croup, and 107 deaths. Excluding the St. Eugénie Hospital, its statistics having been already given (vol. i, p. 534), in the same year, in all the other hospitals of Paris, there were 6 operations and 6 deaths.

Dr. Barthez (Les resultats de la Trachéotomie dans le croup. *Gazette Médicale*, Août 1, 1868) gives the following facts with regard to this operation at the St. Eugénie Hospital. In 1862, there were 113 operations and 89 deaths, or 1 recovery in 5. In 1861, the proportion of recoveries was 1 in 6.90. In 1866-67, there were 223 operations and 79 recoveries, or 1 in 2.80. From 1861 to 1867, there were in all 755 operations and 222 recoveries, or 1 in 3.53. In 132 cases of croup treated during the same period, and not operated upon, there were 82 recoveries, or 1 in 1.60.

Dr. Barthez gives us the statistics of *all* the cases of croup admitted into St. Eugénie, from 1861 to 1867, 917 cases and 304 recoveries, or 1 in 3, about the same results as those of Rosen, and Rilliet of Geneva. He adds, "What gives the chief value to the statistics obtained by the results of the operation in the hospital is, that the largest number of the patients are brought there after the medical treatment has failed; and quite a number of them in such a condition that death is inevitable. The urgency is sometimes so great that there is hardly time to get ready the instruments."]







## PART IV.

### MEDICAL GEOGRAPHY; OR, THE GEOGRAPHICAL DISTRIBUTION OF HEALTH AND DISEASE OVER THE GLOBE.\*

#### CHAPTER I.

##### SCOPE AND AIM OF THIS BRANCH OF SCIENCE.

THIS department of the Science of Medicine treats of the manner, and endeavors to investigate the conditions, under which diseases are distributed over the world, or are confined to certain districts. It embraces a consideration of topics which constitute the basis of *physiology*, and which are of the greatest importance to Practical Medicine, of the utmost interest to Science, and of inestimable value to Political Economy. It embraces the medical application of the facts of *physical geography*, combined with those of *vital statistics*; and it has been variously named MEDICAL GEOGRAPHY or NOSO-GEOGRAPHY.

**Geographical Distribution of Disease-Realms.**—As the physiological conditions of plants and animals vary according to different degrees of latitude, or rather with the different lines of equal temperature and moisture north and south of the equator, so do the pathological characters of diseases differ; and races of men are influenced as

\* Our knowledge on this subject is as yet only beginning to assume a shape; and the limits of this text-book merely permit the most faint outline to be given. To Alexander Keith Johnston, F.R.S.E., the medical profession of this country is indebted for bringing the subject prominently forward, in a communication to the Epidemiological Society of London, published in their *Transactions* for 1856, p. 25, and also in his *Physical Atlas of Natural Phenomena*, where his observations at p. 117 are illustrated by a map. "No scholar out of the domain of medicine has contributed documents more valuable than these to medical literature." That map Mr. Johnston has reduced to a scale suited to this handbook, and thus liberally permits me to use it in illustration. The most important works or monographs which have been published on this subject are Mühry's *Outlines of Noso-Geography*, in two volumes; and Boudin's *Traité de Géographie et Statistique Médicales et des Maladies Endémiques*, Paris, 1857, 2 vols., 8vo. A paper on Acclimation by Dr. J. C. Nott, in a work entitled *The Indigenous Races of the Earth*; Sir Alexander Tulloch's *Army Statistics*; Marshall's "Sketch of the Geographical Distribution of Diseases," in the *Edinburgh Medical and Surgical Journal*, vol. xxxviii, p. 880, and vol. xliv, p. 28; Dr. A. S. Thomson's *Thesis on the Influence of Climate on the Health and Mortality of the Inhabitants of the different Regions of the Globe*, Edinburgh, 1837; and Sir Ranald Martin's work on the *Influence of Tropical Climate*, are the sources from which the outline here given has been compiled.

to health in proportion as they migrate from the land of their birth. It is the ascertained facts in meteorology and climatology of our globe which will help to explain the geographical limits of particular diseases, and their regulated distribution according to atmospheric temperature and moisture, the density and electricity of the air, and the vegetation with which they are surrounded. Such causes determine some of the laws by which diseases may be geographically distributed; but other concurrent causes must also be taken into account in considering the special diseases of countries. For example, topographical situation, geological nature and elevation of the soil, and state of the vegetation; in short, *physical climate*, generally and properly so called, combined with the habits of the people, their attention to personal hygiene and general sanitary arrangements, all concur to stamp the diseases of certain countries with a special character, and facilitate or retard their propagation.

There are facts which show that certain diseases are so completely under the influence of temperature that they are susceptible of being arranged systematically in zones of geographical distribution. The diseases susceptible of being thus classified are those of a communicable type, which require a certain range of temperature and concurrence of physical conditions for their prevalence and propagation.

They are chiefly *yellow fever, plague, typhus fever, typhoid fever, and cholera*. The geographical distribution of these diseases into zones, north and south of the equator, appears to be regulated, in a great measure, by relative degrees of temperature and humidity in the several places where they prevail, in America, Asia, Africa, and Europe. Such diseases as have been mentioned, and whose realms are bounded in a great measure by isothermal lines, are not only associated with locality, with characteristic vegetation, with heat and humidity, but they also follow the physiological habits of the several animal inhabitants of the different latitudes. *Malarious fevers, yellow fever, plague, typhus, and typhoid fevers*, have particular climates or zones where each predominates, and beyond the limits of which the disease is rarely if ever perpetuated when imported, unless change of seasons gives rise to a state of climate analogous to that in which the specific disease is known to flourish.

**Isothermic Zones** bear a most prominent part in relation to the geographical distribution of diseases. They connect the different places on the earth which have the same mean temperature, and which Humbolt was the first to indicate. *Insect-realms* have been similarly indicated by Latreille. The late Professor Edward Forbes described *homozoic belts* of marine life. Cuvier, Blumenbach, Morton, Latham, Prichard, and others, have indicated *homoecephalic zones or realms of men*; and, following out these ideas, we have realms of disease defined by Dr. Mühry and Mr. Keith Johnston.

**Realms of Disease and Description of the Map.**—These zones, belts, or realms of particular types of diseases, thus marked out on the globe by these observers, are intimately associated with temperature, and may be generally indicated by the regions of the *tropical, temperate, and polar* zones, distinguished on the map by the respective colors of *brown, green, and blue*.

I. The northern limit of the tropical zone unites with the southern limits of the temperate zones, and the lines of union of the two colors on the map (green and brown) indicate Humboldt's mean annual isothermal line of  $77^{\circ}$  Fahr. or  $19^{\circ}$  Reaumur. It passes through Cuba and Florida in America; skirts the Cape de Verd Islands to Africa, where, extending beyond the usual limits of the tropics, it passes the northern part of the great desert (Sahara) below Algiers, runs through Egypt, Northern Arabia, and Persia, into China, where it is lost in the Pacific Ocean, below the limits of the northern tropic. The limiting line of this zone ascends somewhat in summer, when the sun is north of the equator; and descends again in winter, when the sun is to the south of it. To the south of the equator the same *isothermal line* ( $77^{\circ}$ ) marks the southern limits of the tropical *disease-realm*, where it joins with the northern limits of the *south temperate* zone. It crosses South America near the Amazon district, and approaching southwards in Africa towards the Cape, crosses over and embraces the northern half of Australia. This is the *realm* of tropical diseases, and is colored *brown* on the map. The class of diseases which characterize this realm are the worst forms of *malarious* (*intermittent* and *remittent*) *fevers*, associated more especially with *dysentery*, *diarrhœa*, *malignant cholera*, *specific yellow fever*, *hepatic affections*, and *their results*. Our summer and autumnal affections, characterized by *biliousness*, *diarrhœa*, and *bilious*, *gastric*, or *typhoid fevers*, approach, by the phenomena they express, the type of the *tropical* disease. The paludal fevers of this tropical *disease-realm* prevail in their greatest intensity in flat, low-lying countries in the vicinity of marshes, the borders of lakes, shores of rivers and of the sea, and especially where the soil is damp underneath, and of certain geological formation. Sir Ranald Martin has clearly shown how the various soils affect powerfully the temperature and humidity of a place. Argillaceous and ferruginous soils appear in this realm to be especially insalubrious. The malarious fevers of this region make their appearance soon after the setting in of the rainy season, or when overflowed grounds, such as rice-fields, the partially dried-up beds and mouths of rivers, or irrigated plains, begin to dry up and leave portions of the surface of the land, whose subsoil is constantly wet, exposed to the rays of a tropical sun. From such a surface the belief is now universal that a miasm (of the specific nature of which we know nothing) emanates, and acts as a poison upon the blood; and is apt to be developed under similarity of climate, season, and soil, and to produce diseases whose symptoms and course express a constancy and similarity of type. The great centres of these malarious diseases in the different continents are,—(1.) In America—the shores of the Gulf of Mexico, the West India Islands, and the northern portion of South America; (2.) In Asia—India, China, Borneo, Ceylon; (3.) In Africa—the countries round the Gulf of Guinea on the west, Madagascar and Mozambique on the east, Algeria and the shores and islands of the Mediterranean on the north.

The whole mortality within this realm of disease amongst na-



tive troops (as estimated by Mr. Johnston) is about 18 per 1000 for all India, and about 75 per 1000 for the more unhealthy districts. An average mortality may therefore be stated of about 46 per 1000 for the realm of disease now under consideration, or 4.6 per cent. annually. Active disease and premature death have been such common occurrences in India, that people have been led to believe these results to be the price which we must pay for the tenure of that country. Because disease and premature death were so common, they ceased to arrest public attention, and the causes of them, so frequently pointed out by medical authorities in India, were generally disbelieved by the home authorities when reported to them. At last the *Report of the Royal Commission on the Sanitary State of the Army in India* has disclosed to the public the real state of the case, so long known to the intelligent but helpless medical officer. The state of affairs had been unceasingly represented by such men as Jackson, Sir Ranald Martin, Norman Cheevers, Morehead, W. C. Maclean, Macpherson, and many others. It has been promised by the Home Government that the sanitary reforms and improvements indicated by the Commission in their published Report shall be carried out. Several great measures advocated in the Report, and most ably urged by Miss Nightingale in her excellent pamphlet, *How to Live and Not Die in India*, have already been carried out. A Commission of Health has been appointed for each Presidency, and that for Bengal has already given public evidence of its zeal. These Commissions have been put into communication with the Barrack and Hospital Improvement Commission at the War Office, which now contains members representing the Indian Government; and by this time the Indian Commissions at the several Presidencies are in possession of all the more recent results of sanitary works and measures which have been of use at home. The condition of the soldier in India is being improved. The worst personal cause of ill-health to which he was in former times exposed have been, or are being, removed. The soldiers are allowed and encouraged to cultivate gardens and to work at trades. The regulation *two drams* of alcoholic spirits have been reduced to one, and that *one dram* is to be diluted with water. "But the main causes of disease in India—want of drainage, want of water-supply for stations and towns, want of proper barracks and hospitals—remain as before in all their primitive perfection."

The maximum intensity of *dysentery*, *yellow fever*, *diarrhœa*, *malarial fevers*, and *affections of the liver*, is observed in those countries which are situated under the line of greatest annual mean temperature—namely, 82° Fahr., which is the assumed equator of heat of the globe. This line of greatest heat also intersects the region of the earth where the greatest amount of water is deposited. Northward to 23° of north latitude, 53 per cent. of all the deaths are attributable to these diseases. In latitude 35° north, where the greatest annual temperature is only 77° Fahr., and just where the zone of the tropical disease-realm merges into that of the temperate realm of disease, these diseases cause about 14 per cent. of the whole mortality. To the southward, again, in the same latitude—namely,

35°, at the Cape of Good Hope—these diseases yield a loss of only 3 per cent. of the whole deaths.

II. The next well-marked *realm* is that in which varied forms of *continued febrile* disease take the place of the *malarious* or *paludal* fever of the torrid zone. The region where diseases of this type prevail embraces *realms* to the north and south of the equator, which may be generally described as in the north and south temperate zones. The southern boundary of the northern realm corresponds to the northern boundary of the tropical *disease-realm*, as already indicated by the isothermal line of 77°. In a northern direction it extends to latitude 60° north, and includes the British Isles, Norway, and Sweden; while in America its northern limit includes part of Nova Scotia and Newfoundland. Its boundary line to the north passes from 60° north latitude in a southeastern direction on both continents, till it gradually declines towards the borders of Asiatic Russia in the Old World, and to the district between Boston and Philadelphia on the eastern shores of the New, and corresponds nearly with the annual isotherm of 41°. In the southern hemisphere it embraces the southern two-thirds of South America, the district of the Cape of Good Hope, and the southern half of Australia. It embraces the most healthy regions of the world, in which the prevailing causes of ill-health are mostly due to the *condensation of people in towns*, and the insalubrious and depressing conditions which necessarily arise from that cause. Emanations from nuisances tend to accumulate, where mechanical and chemical arrangements of a sanitary kind do not remove the concentrated impurities. But where proper sanitary measures exist for the supply of pure water and free air, the artificial disadvantages inseparable from town life may be greatly modified in their influences, so much so, that in our country (the least unhealthy of all) the mortality may range from 1.7 to 3.6 per cent. annually (Reg.-Gen. Rep. for 1858, p. 15). Nearly every type of disease has a representative in this realm; but, generally speaking, the general and mixed diseases are the classes which furnish the greatest mortality. *Typhus* and *typhoid fever* prevail between the parallels of 44° and 60° in Western Europe; *yellow fever* has prevailed on the southern shores of Spain, the northwest coast of France, and Northern Italy; *intermittent fever* in the Netherlands, Sweden, and Central Italy, and generally where marshy, undrained lands exist. *Small-pox* especially prevails where vaccination has not checked its ravages; *leprosy* and *elephantiasis* prevail in Scandinavia; *pellagra* in Italy, France, and Spain; *plica polonica* in Poland and Tartary; and *consumption* and *rheumatism* everywhere. While, therefore, this temperate zone embraces, on the one hand, the extremes of temperature of the torrid and frigid zones in the seasons of summer and winter, it is also observed to have representatives of the types of disease which prevail in both these realms; and according as we approach its northern or its southern limits we find that the characters of disease become so modified and their types are found so to mingle together on the confines of these disease-realms that the *continued* type of febrile diseases peculiar to the temperate zones

tends, as we approach the tropics, to merge into and participate in those irregular intermittent characters which are peculiar to the type of the tropical malarious districts. It is only to a limited extent, however, that such modifications are found to occur; and there are some diseases which rarely tend to pass their geographical boundaries unless especially favored by *tropical* identity of climate on the one hand, or *temperate* identity on the other. Under the former circumstance yellow fever has been met with at Gibraltar, Cadiz, Lisbon, St. Nazaire, and even so far north as Plymouth Sound and Southampton Water; and it has also extended in America southwards beyond its usual limits; but so soon as the temperature falls below 55° Fahr., the importation of yellow fever into *this zone*, and its propagation or persistent existence in it, becomes impossible. Diarrhœa, also, and dysentery are apt to prevail during the summer and autumn months; while under bad diet, defective ventilation, and generally imperfect sanitary measures, *contagious typhus fever* is apt to be propagated in crowded localities of towns, and in huts, hospitals, and barracks. Typhus and typhoid fevers seem to have their special habitation in this zone, especially between 30° and 40° of north latitude, and likewise the true glandular plague; and which may be occasionally propagated beyond these limits by a secondary and specific poison, generated from human bodies and propagated by human intercourse. The zone, therefore, which we inhabit, while it may be said to exhibit the greatest variety of disease-processes and types of disease, may, nevertheless, be considered a highly favored district of the globe, inasmuch as the development and progress of disease and of epidemics are much more manageable, less intense, and on the whole less fatal, than similar types of disease in the tropical realm, or even than they were centuries ago. Under proper sanitary regulations *typhus fever* and many other diseases in this country are greatly under control. *Cholera* may be said to be the only scourge which occasionally, as an epidemic, becomes *rapidly* destructive to life. But, by scientifically directed sanitary measures, and the increasing experience of an enlightened age, the spread of the *plague* in this country has ceased. In this realm "history records the existence of diseases of former days now happily unknown; while, on the other hand, scourges unknown to ancient times devastate modern populations" (PLINY, SYDENHAM, BOUDIN). In the sixteenth century the Oriental plague, "like the destroying angel, spread its wings on the blast," and added to the miseries of that stormy and bloody time. Our physician-poet tells us that when the fate of England was about to be decided on "Bosworth's purple field," that plague, of most gigantic arm,

" Rushed as a storm o'er half the astonished isle,  
And strewed with sudden carcasses the land."

Then, as now, our standing armies and bodies of men congregated together especially suffered from pestilential disease. It so thinned the ranks of Henry's victorious army that few were left to see the conqueror crowned with Richard's diadem. Asiatic cholera may

now be said to have taken the place of the Oriental plague, and it is often no less destructive and important in its results—sternly demonstrating “how one dread year performs the work of ages, when the pestilence mocks in his fury the slow hand of time.”

III. To the northward of this temperate zone, in the northern hemisphere, there is another disease-realm, where *catarrhal affections*, *influenza*, *scurvy*, *erysipelas*, *diseases of the skin* and *digestive organs*, and various CONSTITUTIONAL affections more especially prevail, to the exclusion of *malarious febrile diseases*, except on very rare occasions in summer, and when *cholera* and *dysentery* may prevail. This *disease-realm*, in the polar isothermal zone, rejoices in a climate directly opposite to that of the tropical zone. Its southern limits are the northern boundaries of the previously defined region—namely, the isothermal line of  $41^{\circ}$  Fahr., or  $2^{\circ}$  or  $3^{\circ}$  of Reaumur. Commencing on the western coasts of North America above Sitka, it extends southward across the district of the Canadian lakes, sinking south and east into Canada and Newfoundland to Boston and New York or Philadelphia. Thence it continues northwest nearly on  $41^{\circ}$  Fahr. annual temperature, when it crosses to Europe, and ascends till near the borders of Iceland, whence it sinks towards Norway and Sweden, and, running above St. Petersburg and Moscow, crosses to Siberia. Iceland being the best-known locality of this district, Mr. Johnston takes its peculiar diseases as the representatives of this realm. Every year, in spring or in early summer, it is visited by *catarrh*; and at short intervals it is visited by *catarrhal fever*—a *true influenza*, which has usually a great effect on the mortality. The majority of Icelanders are said to die before the age of fifty, from *asthmatic* or *catarrhal affections*, which are also prevalent in Greenland and Labrador.

Dr. Lawson has attempted to establish (see vol. i, p. 224) the occurrence between 1817 and 1836 of a series of *oscillations of febrile diseases*, following each other over the world with amazing regularity. These he attributes to a cause or influence which, from its extent and progressive character, he names a “pandemic wave,” to distinguish the influence from that usually understood as “epidemic,” referring to a single form of disease, affecting a limited space. Under the influence of this pandemic wave he believes that there is a constant progressive tendency to the development of various endemic febrile diseases in the Atlantic and western parts of the Indian Ocean, from south or southeast to north or northwest.

Many of the facts and data on which he founds are, however, not sufficiently trustworthy to rest a judgment upon, and in some cases are susceptible of a totally different interpretation from that which Dr. Lawson has assigned to them. Although, therefore, his theory is “not proven,” yet the expression of it is calculated to do good, by drawing attention to this view of the subject.

## CHAPTER II.

## ON MALARIA AND PLACES KNOWN AS MALARIOUS.

GEOGRAPHICAL facts, collected by medical writers from Hippocrates downwards, show that every country is unhealthy in proportion to the quantity of marsh, or of undrained alluvial soil, it contains, the inhabitants of such districts dying often in the ratio of 1 in 20, instead of 1 in 38—the average mortality in healthy countries. The connection of a given class of disease—represented by remittent and intermittent fever—with marshy districts is now distinctly established and generally recognized.

Ancient Rome was once the seat of so many fatal epidemics that the Romans erected a temple to the goddess Febris. These epidemics were known to arise from the great masses of water poured down from the Palatine, Aventine, and Tarpeian hills becoming stagnant in the plains below, and converting them into swamps and marshes. The elder Tarquin ordered them to be drained, and led their waters by means of sewers to the Tiber. These subterraneous conduits ramified in every direction under the city, and were of such considerable height and breadth that Pliny terms them "*operum omnium dictu maximum suffossis montibus atque urbe pensili subterque navigata.*" This system of drainage, which was continued as late as the Cæsars, rendered Rome proportionably healthy, and the seat of a larger population than has since perhaps been collected within the walls of any city. On the invasion of the Goths, however, the public buildings were destroyed, the embankments of the Tiber broken down, the aqueducts laid in ruins, the sewers obstructed and filled up, and the whole country being now again overflowed, Rome has once more become the seat of an almost annual paludal fever, as in the times of her earliest foundation. The insalubrity of the Pontine Marshes, past or present, is notorious. Three hundred years, however, before the Christian era, Appius Claudius drained them, by making canals, building bridges, and by constructing that magnificent road, portions of which still remain and still bear his name. On the invasion of Italy by Theodoric, Cæcilius Decius gave a free course to the waters in the neighborhood of Rome, and the re-establishment of these immense marshes was one of the many disasters which resulted from the attacks of the Goths on Italy. Their present state is such that the Tuscan portion of Maremme, and indeed the whole of that district, may be said in summer to be absolutely depopulated, not a single house retaining an inhabitant, except the guard-houses, with a few soldiers and custom-house officers; and these are relieved twice or thrice during the summer, with the Maremme fever almost invariably upon them.

Many districts in the East and West Indies, in Pennsylvania, and Continental Europe, are known to be active in the evolution of *malarious influences*. Such places are generally the deltas, marshy



banks, and embouchures of rivers, in the plains extending from the bases of mountain ranges; partially inundated and irrigated lands, or such as are traversed by percolating streams or canals in wooded districts termed *jungles*; the seaboard, especially where there is jungle or salt marsh; and in the Bengal district the stations of Calcutta, Chinsurah, and Berhampore are highly malarious (MARTIN). The woods and marshes of the Sunderbunds, covering a superficies of more than 20,000 miles, and extending 180 miles south and east of Calcutta, composed of marshy land, covered with forest and underwood, together with the numerous embouchures of the Ganges, are well-known unhealthy districts. The partially dried-up marshes and beds of rivers have too often been fatal to our armies when imprudently and ignorantly encamped in their vicinity. In 1810 the plains of Spain, along the course of the Guadiana, with its "lines of detached pools" and its ravines always "half-dried," could tell of a fever-stricken army. The pages of history also remind us how our British soldiers perished on the low, dry-looking, sandy plains of Walcheren and of Rosendaal in 1794 and 1809-10. Our last war with Russia, during the campaign in Bulgaria, and especially at Varna in 1854, furnishes a no less melancholy record of the sufferings of British troops, and the persistent pernicious influence of a residence in malarious districts.

In China we know of the miasmatic nature of the deltas of the Blue and the Yellow Rivers. In Africa we know of the Zais, the Orange, and the Zambesa as unhealthy rivers. In America the Amazon, the Orinoco, and the Rio del Norte are similarly deleterious. In England we have the fens of Norfolk and Lincolnshire still a source of disease; in short, there is hardly a country which has not its marshy lands, so that abundance of work exists for chief commissioners of sewers to direct for good, where such "heads" of "*bodies*" exist; and the extent of disease proceeding from marshes has been shown in many places in Italy, Sicily, and Greece to be so great as to occasion more than two-thirds of the average mortality.

**Removal and Neutralization of Malaria.**—Of towns that have been drained and remain healthy there are many examples in ancient and modern history. Hippocrates tells us that the city of Abydos had been several times depopulated by fever; but the adjoining marshes having been drained, it became healthy. London, in the time of Sydenham, was infested with epidemic intermittent fever and dysentery, the mortality from the former alone averaging, in a comparatively small population, from one to two thousand persons annually. In the present day, owing to the formation of sewers and a general system of drainage, a case of ague contracted in London is hardly known. Many other towns, both of this country and of France, as Portsmouth, Rochefort, and Bordeaux, from being the constant seat of paludal fevers, have been, from the same causes, rendered in like manner comparatively healthy. Dr. Wood, of Philadelphia, relates an interesting fact regarding the neutralization of miasmatic effluvia. He tells us they are in some way rendered innocuous by the air of large cities. This fact is notorious in relation to the city of Rome; and it is abundantly con-

firmed in the larger towns of the United States, in the neighborhood of which these diseases have prevailed.

**Nature of the Noxious Agent.**—Thus the intimate connection between marshy districts and certain forms of disease is established by a great amount of direct and indirect testimony; but two questions still remain—namely, What is the nature of the noxious agent of malarious districts? and what circumstances are necessary to its formation or extrication? It seems certain that the deleterious agent is neither heat alone nor moisture alone, nor any known gas extricated from the marsh. It cannot be *heat* alone, for many of the hottest parts of the West Indies are free from fever. It cannot be *moisture* alone, for no persons enjoy better health than the crews of clean ships at sea, even when cruising in tropical climates, as long as they have no communication with the land. While *carbonic acid*, *azote*, *oxygen*, or *carburetted hydrogen*, the gases collected by stirring the bottom of marshes, have all been inspired without producing any disease similar to paludal fever, it seems to follow almost as a necessary consequence that the remote cause must be a *miasm*, *poison*, or *malaria*, whose presence is solely detected by its action on the human body; and two hypotheses have been imagined to account for its origin;—the one, that it is a product of vegetable decomposition—the other, that it is an exhalation from the earth, favored by the conditions of marshiness. A theory, often hinted at, is beginning to find expression—namely, that very minute fungi of rapid growth, like *smut*, *rust*, *mildew*, and the like, are active agents in the propagation of malaria. Inspector-General Reid has shown this in regard to the recent epidemic in the Mauritius, and Dr. Massey in regard to Ceylon (see *Army Med. Report* for 1867).

The general evidence in favor of vegetable decomposition (and therefore growth) being the remote cause is, that all countries are for the most part free from paludal diseases while the crops are growing, and only become unhealthy after the harvest, when large quantities of vegetable matters are left on the ground at the time the rain begins to fall. Marshes are in general healthy till the summer's sun, or other cause, has diminished their waters, and bared a greater or less portion of their bed. The part thus exposed almost always contains a large portion of vegetable matters, which, running into rapid decomposition, generates other vegetable growths of a fungoid nature especially, which may be or may convey the poison which gives origin to this class of disease. It is during the periods of the year when the drying process is in greatest activity that unhealthiness prevails with greatest severity in the East Indies—namely, before the commencement and after the termination of the rainy season. The particular evidence of vegetable decomposition being the source of the poison is of the following nature: Lancisi gives the history of an epidemic which for several summers infested, and almost depopulated, the ancient town of Urbs Vetus, situated on an elevated and salubrious part of Etruria, and which was traced to the circumstance of the peasants steeping their flax in some stagnant water in the neighborhood of the town. This practice was therefore prohibited in 1705, and the epidemic ceased

to appear. The steeping of flax being productive of paludal fever is a fact the knowledge of which is not limited to Italy; for the ancient as well as the new "*coutumes*" of almost all the provinces of France have proscribed the steeping of flax, "*la rouissage*," even in running waters, from the fear of infection. In the Netherlands the same belief has prevailed. In July, 1627, the King of Spain passed an ordinance prohibiting the steeping of flax in the streams and canals of Flanders. The prohibition may be explained on another ground—namely, because the flax poisons the water and kills the fish. In Ireland, and perhaps also elsewhere, steeping flax in a running stream is therefore forbidden by law.

The experience of the indigo-planter is to the same effect. In India, after the coloring matter has been extracted from the indigo plant, it was formerly the custom to throw the detritus into large heaps or masses in the immediate neighborhood of the works, and which, at the end of three or four years, becomes manure of an excellent quality. It was found, however, that these heaps, wetted from time to time by the heavy rains, and afterwards heated by the rays of a burning sun, rapidly decomposed, and at length emitted *miasmata*, which produced all the effects of those extricated from the marsh. The workmen who lived near, and more especially those to leeward of these masses, were found to be very commonly attacked by fever, chiefly of the remittent type, and similar to those which prevail in the paludal districts of that country. This consequence is now so well established that the most intelligent indigo-planters no longer allow these heaps to be formed either near the works or in the immediate neighborhood of the cottages of their workmen.

Ships also afford additional evidence of the truth of the hypothesis of vegetable decomposition and growth being the remote cause of malaria.

All intertropical regions, where the nature of the locality admits only of a rice cultivation, are well known to be unhealthy.

These facts render it highly probable that the noxious agent must be a product of vegetable development, growth, and propagation, evolved on the soil, and moving in the lower regions of the atmosphere. No analysis of the air has yet disclosed any immediate chemical principle to which the unhealthy influence of miasms may be ascribed. The atmospheric air collected at the embouchure of the Valtelline—a country where it is impossible to sleep without being attacked with fever—gives, on analysis, the same chemical constituent parts and proportions of gases as that collected at the summit of the Alps, or in the narrowest streets in London. But such places, on the other hand, give evidence of abundant minute fungoid vegetation, which may be active poisons, or active agents in the propagation of malaria.

If we consider the paludal poison to be a product of vegetable decomposition, or of vegetable growth, it follows that heat and moisture, quantity of vegetable matter, and nature of the soil, though not the essential agents, must have a sensible influence on its formation, must vary its intensity or quantity, and also must

limit paludal diseases to particular localities, seasons, and latitudes. A certain temperature, for example, under certain conditions as to moisture, is evidently necessary to its extrication and development.

It is certain also that a given quantity of moisture is as necessary to vegetable decomposition or growth as a given temperature, and that the extrication of the paludal poison will be most abundant from that soil which contains no more moisture than is necessary for that process; for an excess in quantity, by dividing and separating the particles, and by preventing the access of atmospheric air, will either retard or altogether put a stop to putrescency. Hence in some countries frequent and heavy rains will render marsh fevers prevalent, by saturating the whole of the open country; while privation of rain will in others produce exactly the same effect in other instances, merely by diminishing the superfluous quantity of water. Thus, in the West Indies, an uncommonly rainy season seldom fails, in the perfectly dry and well-cleared Island of Barbadoes, to induce for a time general sickness; while at Trinidad, whose central portions are described as a sea of swamp, and where it rains nine months in the year, an excess of moisture is a preservative from sickness; for should at any time rains fall only eight months in the year instead of nine, the swamps become dry and bared to the sun, and remittent fevers of the worst kind are sure to make their appearance. The same result follows on the subsiding of the waters of rivers that have overflowed their banks, as those of the Nile, the Rhone, the Danube, the Tigris, the Ganges, and many others.

It is evident from these data that the swamp, on its approach to dryness, is the source of disease and death; while an excess of rain has a preservative power so long as moisture is in excess. On the contrary, on the rich and dry plains, and even on the hills of tropical countries, rain is the cause not only of vegetable decomposition, but also of disease; while absence of rain tends to preserve health.

In estimating, however, the dryness of a country, its superficial appearance is often deceitful. In the years 1748 and 1794 the summers were dry, and our troops took up the encampments of Rosendaal and Ousterhout in South Holland. The soil in both places is a level plain of sand, with a perfectly dry surface; and where no other vegetation existed, or could exist, but a few stunted heath-plants; yet in both years fever became epidemic among the troops in each place. On digging for water the cause was discovered, for the soil was found to be saturated with water to within a few inches of the surface. It is probable, therefore, that this country was originally formed of vegetable and other detritus, brought down by the Rhine and the Waal, and afterwards covered with sand thrown up by the sea, and which, heated by the summer's sun, became the powerful cause of the extrication of marsh miasmata. From the exceeding malignity of the salt marshes, it has been supposed that a mixture of salt and fresh water renders a marsh more pernicious than either of them alone. It has been found, however, that on coasts where these marshes have been kept up to one uniform level by means of flood-gates, the surrounding



country is healthy; and it has therefore been inferred that the sickness produced is a consequence of the perpetual alteration of the level of the waters of the marsh, and not owing to the admixture of sea and spring water.

It is probably owing to a great excess of temperature that rocky countries, as Gibraltar and the Ionian Islands, are so often and so severely attacked with malarious fever. It is on the summit of these rocks that springs arise. The slightest frost produces fissures, into which fungi, as "*mould*," and other vegetable matters, insinuate themselves, while the bare rock becomes heated to an intense degree. Humboldt, on ascending the Orinoco, found the station at the great fall depopulated by fever, which the natives attributed to the bare rocks of the rapids. He determined the heat of these rocks to be  $118.4^{\circ}$  Fahr., while the temperature of the air immediately around was only  $78.8^{\circ}$  Fahr. Again, the rock of Gibraltar is known to be percolated with water, so that we can hardly conceive a more pestilential focus of disease when the causes necessary to the formation of miasm or fungi are combined. The existence of paludal fever in dry and rocky districts, therefore, although it may appear extraordinary and unexpected, is not necessarily an exception to the general law of paludal diseases being generated by miasmata, the result of vegetable decomposition or of vegetable development. In many climates the most deadly sites for encampments have been the dried up beds of rivers, or their immediate vicinities (MARTIN).

These facts seem, therefore, unquestionably to prove that heat and moisture, though not the primary cause of paludal disease, are conditions essentially connected with the development of vegetable growths, like fungi, of the noxious miasmata, and consequently furnish a strong additional argument in favor of the hypothesis of vegetable decomposition generating the remote cause which produces or propagates some miasmatic diseases. It is certain, however, even when the conditions of heat, moisture, and vegetable matter most abound, that paludal diseases do not always assume their severest forms; and there seems reason to believe that differences of geological formation, by favoring or otherwise influencing vegetable putrefaction and growth, may variously affect the health of countries similarly situated in other respects.

It is perfectly well known that different soils radiate heat with different degrees of intensity, and consequently are, under the same circumstances, of different temperatures, having very different powers of attracting moisture; and possibly, also, they may have other and more direct properties favorable to the generation of the paludal miasm. Nothing, for instance, is better determined in husbandry than that the *carbonate of lime*, mixed with the ordinary matters of a compost, greatly forwards the processes of putrefaction, so that the mass thus prepared is fit in a much shorter time for the purposes of manure.

There are some soils peculiarly favorable to the decomposition and growth of vegetable matters, and consequently to the more abundant extrication of marsh miasmata; and it is remarkable that those countries most celebrated for paludal fevers have been



found similar in their geological formation to each other, and to those artificial conditions which most favor rapid vegetable decomposition and growth.

To predicate all the facts connected with paludal diseases is not yet possible; for the variations of atmospheric temperature, the changes in the quantity and nature of the electric fluid, the quantity of water, the nature of the soil, the amount and character of the vegetable matters and their growth, form a problem extremely complicated, and one whose smallest variation as to quantity or time may occasion marked differences in the result. As a general rule, however, it may be stated, that in no climate do paludal fevers prevail to an equal degree all the year round. In the winter, much of the vegetable matter has already undergone decomposition and further growth, while the dryness of the season and the diminished temperature are little favorable to its further development. When the spring, however, arrives, and the rain falls, and the heat of the sun increases, the earth again evolves a miasm of mitigated intensity. In summer the products of vegetable decomposition are used up in affording nourishment to the growth of many vegetable forms; and this season, like the winter, is in general healthy. But in the autumn, and after the harvest has been gathered, when the ground is covered with vegetable debris, when the rain falls in torrents now and again, and when the solar heat has acquired its greatest intensity, all the conditions for the greatest amount of growth of vegetable matter, of moderate moisture, and of highest temperature, change and are united; so that the season which realizes the hopes of the husbandman is also the period of pestilence and of his greatest danger. There are two other facts, also, which are too prominent to be mistaken. The one is, that the miasmata vary greatly in intensity in different countries, and also in different parts of the same country; again, the diseases they produce, though annually *endemic* in given districts, yet become, in certain years, and from the action of causes not yet determined, *epidemic*.

In the same countries, also, it is determined that difference of altitude is equivalent to difference of latitude; and, as a general law, it may be stated that in the Antilles, on the continent of America, from Boston to Rio de Janeiro, and also on the continents of Asia and Africa, while in the low country severe remittent or yellow fever prevails, still in the higher country, though immediately contiguous, the type is changed to intermittent and mild remittent. The interesting fact stated by Humboldt, that the *vomito prieto* never appears on the table-lands of Mexico, is strictly in accordance with the observations made in every other equatorial part of the world at a similar elevation above the level of the sea. The circumstance of intermittents passing into remittents, and remittents into malarious yellow fever, and, conversely, of remitting and malarious yellow fever often terminating in intermittent—facts observed not only in the East and West Indies, but on the continents of America and of Africa—demonstrates a unity of cause as firmly as the best-established facts in medicine.

That paludal diseases, like many diseases produced by morbid

poisons, are annually endemic, and only occasionally epidemic, is unquestionable. A few years ago intermittent fever was epidemic in particular districts in this country; but of late years the cases of ague have been comparatively rare. In Demerara it has been observed that malarious yellow fever is epidemic about every seventh year. At Gibraltar, although sporadic cases of paludal fever occur annually, still malarious yellow fever is only occasionally epidemic, but so irregularly that it assumed that character in 1804, then in 1810, again in 1813 and in 1814, and from that period the garrison suffered no similar visitation till 1828. The physical causes on which this greater virulence and greater spread of the disease depend are not determined. In temperate climates it has been observed that paludal fevers have been most prevalent when a hot summer has succeeded a wet spring.

**Infecting Distance of Miasmata.**—As a general law, the danger of infection is in proportion to the proximity to a marsh. But there are many disturbing causes which produce remarkable exceptions to this law. These disturbing causes are, the extent of surface which generates the miasmata, their intensity, the direction of the wind, its force, the season of the year, the time of the day, and the attracting influence of the surface over which the miasmata pass.

**The Altitudinal Range.**—The Monte Mario, which adjoins Rome, is, according to Breyslack, about 165 yards perpendicular height above the Pontine Marshes, and is extremely unhealthy. Tivoli, which is about 230 yards above the level of the same marshes, is infinitely more salubrious; while at Serre, 340 yards perpendicular height, the inhabitants enjoy an entire exemption from the paludal diseases which prevail below. In Italy it is estimated that an altitude of 1400 to 1600 feet is necessary to assure an exemption from paludal disease; but in the West Indies, where the poison is of so much greater intensity than in Italy, it is estimated that an elevation of 2000 to 2500 feet is necessary to give a similar immunity. The different latitudes may account for this.

In towns partially freed from marsh miasmata by extensive drainage, the difference of a few feet perpendicular height makes an almost inconceivable difference in the liability of persons to paludal disease. The barracks of Spanish Town, the capital of Jamaica, for instance, consist of two stories, or of a ground floor and of a first floor; but it being found that two men were taken ill on the ground floor for one on the first floor, it was at length ordered that the ground floor should be no longer occupied. Dr. Cullen remarked a similar result at Portobello, Dr. Fergusson in St. Domingo, and Sir Gilbert Blane in the expedition to Walcheren. This law is so well understood in the West Indies that in Demerara, and in many other parts, the houses are built on dwarf columns, after the manner of corn stacks, in order that a stratum of air may be interposed between the house and the ground. In Rome, and in other towns of Italy, it is also so well known that the lower rooms of the houses are abandoned, the family occupying the upper rooms, as affording a greater protection from the paludal poison.

**The Lateral or Horizontal Spread** of marsh miasmata is a problem still more difficult than that of the altitudinal range. The least complicated cases are those when water alone intervenes between the marsh and the recipient. In the year 1746-47, while our troops lay in Zealand, the sickness was so great among four battalions quartered there that some of those corps had hardly 100 men fit for duty, or less than a seventh part of a battalion. In one corps, the Royals, only four men escaped. At the time, however, of this remarkable prevalence of fever on shore, Commodore Mitchell's squadron lay at anchor between South Beveland and the Island of Walcheren, and the fever raged at both places; but, nevertheless, in the midst of all the sickness that reigned around, the seamen were neither affected with fever nor flux, but continued to enjoy perfect health. These observations of Sir John Pringle were fully confirmed by those of Sir Gilbert Blane during the last disastrous expedition to Walcheren. "I had," says this physician, "the opportunity of observing the extent to which this noxious exhalation extended, which was found to be less than was generally known. Not only the crews of the ships in the Road of Flushing were entirely free from this epidemic, but also the crew of the guard-ship, which was stationed in the narrow channel between this island and Beveland. The width of this channel is about 6000 feet; yet, though some of the ships lay nearer to one shore than the other, there was no instance of any of the men or officers being taken ill with the same disorder as that with which the troops on shore were affected." It appears, therefore, that in Europe the horizontal spread of marsh miasmata over fresh water is less than 3000 feet. With respect to the spread of the miasmata over salt water, Sir Gilbert Blane wrote that in tropical climates ships at a distance of 3000 feet from a swampy shore—a distance to which the miasmata did not extend in Zealand—and even farther, were affected with the noxious exhalations. Dr. John Hunter considered a few miles to be a necessary interval for a ship lying to leeward of a swamp, in order to insure a complete exemption from the disease. When, however, the swamp or other source of the poison is of small extent, a much less space is sufficient to assure an exemption. In the epidemic on the coast of Spain, the fisherman living with his family on board his boat has been rarely attacked, though lying at anchor close in shore. Also, during the late epidemics at Gibraltar, it was not unusual for the richer inhabitants to hire a Moorish vessel and to live on board in the bay; and there was scarcely an instance of these persons having been affected, though keeping up a free communication during the day, either directly or indirectly, with the town.

The extent to which the marsh miasmata may spread from its source over land in a horizontal direction is a much more complicated question. The effect of trees in intercepting the paludal poison is remarkable, and appears to have been known to the ancients, who are supposed to have surrounded their temples with groves, on account of their protecting influence. Pope Benedict XIV ordered a wood to be cut down which separated Villatri

from the Pontine Marshes, and for many following years there raged throughout the whole country, and in places never before attacked, a most severe and fatal fever. The same effects were produced from a similar circumstance in the environs of Campo Santo. In the West Indies it is quite wonderful how near the marsh the planter, provided he is protected by trees, will venture to place his habitation.

Different soils also affect the transmission of the paludal poison. The spot, for instance, on which the new National Dock and Arsenal are built was a marsh of about 700 acres, and on either side of it are the villages of Greenhithe and of Northfleet. The peculiarity here is, that the inhabitants of these villages rarely suffer from intermittent fever, whilst those on the hills beyond are greatly afflicted with that disease. Dr. Maton mentions a similar fact in the neighborhood of Weymouth, and the same circumstance is observed in the neighborhood of Little Hampton and the marshy districts in Sussex.

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## CHAPTER III.

### ACCLIMATION, OR THE INFLUENCE OF CLIMATE ON MAN.

**Definition.**—*Acclimation consists in “a profound change in the organism, produced by a prolonged sojourn in a place whose climate is widely different from that to which one is accustomed, and which has the effect of rendering the individual who has been subjected to it similar in many respects to the natives of the country (indigènes) which he has adopted” (ROCHOUX). The process takes place to a certain extent so far as some individuals are concerned; but the ability to become acclimated is not possessed to the same extent by all nations.*

It is found that the *white races* reach their highest physical and intellectual development, as well as most perfect health and greatest average duration of life, above 40° in the western, and 45° in the eastern hemispheres; and whenever they emigrate many degrees below these lines they begin to deteriorate, from increased temperature, either alone or combined with other morbid influences, incident not less to change of climate than to habits of life—to evils social, civil, and more or less remediable by attending to the common principles of sanitary science.

The laws of climate show that each race of mankind has its prescribed salubrious limits. All of them seem to possess a certain degree of constitutional pliability, by which they are able to bear to a certain extent great changes of temperature and latitude; and those races that are indigenous to temperate climates support best the extremes of other latitudes. The inhabitants of the arctic regions, and of the tropics, have a certain pliancy of constitution; but while the inhabitants of the middle latitudes may emigrate 30° south or 30° north with comparative impunity, the Esquimaux in

the one extreme, or the Negro, Hindoo, or Malay in the other, have no power to withstand the vicissitudes of climate encountered in traversing the 70° of latitude between Greenland and the equator. The fair races of Northern Europe below the arctic zone find Jamaica, Louisiana, and India, to be extreme climates; and they and their descendants are no longer to be recognized after a prolonged residence there. When an Englishman is placed in the most beautiful part of Bengal or Jamaica, where malaria does not exist, although he may be subjected to no attack of acute disease, but may live with a tolerable degree of health his threescore years and ten, he nevertheless ceases to be the same healthy individual he once was; and, moreover, his descendants degenerate. He complains bitterly of the heat, and becomes tanned; his plump, plethoric frame becomes attenuated; his blood loses fibrine and red globules; both mind and body become sluggish; gray hairs and other marks show that age has come on prematurely—the man of forty looks fifty years old; the average duration of life is shortened (as shown in life insurance tables); and the race in time would be exterminated if cut off from fresh supplies of emigrants from the home country. The European in the Antilles *struggles with existence*—a prey to fever and dysentery. He is unequal to all labor, becomes wasted and wan, and finally perishes. His decay is premature; and, but for the constant influx of fresh European blood, he becomes rapidly extinct as a race. The European inhabitants of Jamaica, of Cuba, of Hispaniola, of the Windward and Leeward Islands, have made no progress since their first establishment there. They cannot execute labor—hence the necessity for preserving and maintaining the black population. Their offspring are pale, wan, and sickly, and in half a century cease to be productive. MORELL, p. 107. Our army medical historians tell us that our troops do not become acclimated in India. Length of residence in a distant land affords no immunity from the diseases of its climate, which act with redoubled energy on the stranger from the temperate zone. On the contrary, the mortality among officers and troops is greater among those who remain longest in these climates. JAMES A. MILLER, THOMAS, MACGREGOR, & KENNEDY. Dr. MILLER also makes the significant remark, that the small mortality among officers compared with soldiers in India is due to the greater facilities they enjoy of obtaining change of climate when they fall sick. Although the constitution of the man may be so debilitated that comparative health may be retained, yet there is a constant depletion of the physical and intellectual constitution. If, however, his descendants are taken back to their native climate, they may yet revert to the healthy standard of their original type. The good effects of leaving the period of service in our troops in the West Indies in three years has shown this in sustaining the average period the soldiers of the regiments in India have remained at the European regiments in India during the last year of the last disbandment was 18. European regiments in India have not been sent back the space of a year. It is now some time that the great majority of our troops are sent back to their native



new recruits are required to fill up the broken column; and, eight years having come and gone, not a man of the original thousand remains in the dissolving corps." With regard to the Bombay fusilier European regiment, for instance, Dr. Arnot has shown that its losses averaged 104 per 1000 per annum—a loss equivalent to the entire absorption of the regiment in nine years and seven months. In Bengal, also, it is an ascertained fact that a British regiment of 1000 men dissolved entirely away in eleven years, even in favorable times, and with all the improved conditions of the service. Dr. Arnot's statistics show that the Bengal army lost annually 9 per cent. of its numbers, giving a total loss in eight years of upwards of 14,000 men out of an army of 156,130 men. (See *Trans. Med. Ph. Society of Bombay* for 1855; *Indian Annals of Medical Science*; and *Sanitary Review* for October, 1857; whence these statements are quoted.)

In the *Statistical Reports of the Army Medical Department*, of which four annual volumes have now been published, for the years 1859 to 1862 inclusive, full information respecting the health of the army all over the world has been given by Dr. T. Graham Balfour, F.R.S., Deputy Inspector-General of Hospitals, to whom the science of medicine is so largely indebted in the direction of "Medical Geography." The leading facts are embraced in the following account of

*The Sickness and Mortality of British Troops at Different Places over the Globe.*

I. In the Stations of the United Kingdom the sickness is represented by 1025 admissions, and the mortality by 9.35 deaths per 1000 men.

The class of diseases which give rise to the largest proportion of cases is that of *Venereal*, by which one-third of all the admissions into hospital is caused. Next to it rank *miasmatic diseases*, *diseases of the integumentary system*, and *diseases of the respiratory system*. *Tubercular diseases*, chiefly *phthisis*, occasion upwards of one-third of the whole mortality, and diseases of the *respiratory system* stand next to them. The admissions by *tubercular diseases* average 19 per 1000, and the deaths 3.48; the admissions by diseases of the *respiratory system* amount to 98 per 1000, and the deaths to 1.47 per 1000.

II. The Mediterranean Stations.—1. *Gibraltar* The sickness here is represented by 894 admissions, and the mortality by 8.86 deaths per 1000 men. Of *miasmatic diseases* the *continued fevers* are the most prevalent, and at the same time the most fatal. The sickness from them is represented by 94 admissions, and the mortality by 2.62 deaths per 1000 men. The causes of such prevalence are represented to be—(1.) Overcrowding—the space in one of the barracks having been, during part of the time included in the Reports, only 322 cubic feet per man! (2.) Exposure to sun-heat; (3.) Imperfect drainage and sewerage. *Diarrhœa*, *dysentery*, and *cholera* are FOUR TIMES as prevalent as among troops at home, and occasion 73 admissions, *rheumatism* 38 admissions, and *ophthalmia* 41 admissions per 1000 men.

*Venereal diseases* give rise to nearly one-fourth of the admissions into hospital.

2. *Malta*. The sickness at this station is represented by 906 admissions, and the mortality by 12.31 deaths per 1000 of mean strength.

The diseases of the *miasmatic class*, in the order of their greatest prevalence, are as follow :

*Continued fever*, represented by 188 ; *ophthalmia*, by 81 ; *dysentery* and *diarrhœa*, by 73 admissions per 1000 men. Of these diseases *continued fever* has been the most fatal.

3. The sickness in the Ionian Islands is represented by 807 admissions, and the mortality by 9.11 deaths per 1000 of mean strength.

The most prevalent diseases are *continued fever*, *dysentery* and *diarrhœa*, and *ophthalmia*. The *continued fevers* are the fatal ones. Their prevalence and fatal character in Corfu are attributed, and apparently with justice, to the unhealthy situation of the Fort-Neuf Barracks, their overcrowded state and defective ventilation, and to the generally insanitary condition of the town.

III. The Stations in British America.—1. In *Bermuda* the sickness is represented by 637, and the mortality by 11.79 deaths per 1000 of mean strength. *Continued fevers*, *dysentery*, and *diarrhœa* are the most prevalent diseases, and those of the nervous system the most fatal, owing principally to the number of deaths from *delirium tremens*. Bermuda has been visited at irregular intervals by yellow fever in an epidemic form, but no cases of it have occurred since the commencement of the publication of the Army Annual Reports. In the year 1864 a deadly epidemic prevailed.

2. In *Nova Scotia* and *New Brunswick* the sickness is represented by 581 admissions per 1000 of mean strength, and the mortality by 7.43 deaths per 1000. The most prevalent diseases of the miasmatic class are *sore throat*, *influenza*, and *rheumatism*. The tubercular diseases are the most fatal, causing nearly one-third of the whole mortality. Next to them stand diseases of the respiratory organs—chiefly *pneumonia* and *bronchitis*.

3. In *Canada* the sickness is represented by 622 admissions per 1000 of mean strength, and the mortality by 9.07 deaths per 1000. The miasmatic diseases, in the order of their greatest prevalence, are as follow : *Dysentery*, *diarrhœa*, *cholera*, represented by 28 admissions per 1000 of mean strength ; *rheumatism*, by 28 per 1000 ; *sore throat* and *influenza*, by 26 per 1000 ; *continued fevers*, by 20 per 1000 ; *ophthalmia*, by 16 per 1000. Accidents, tubercular diseases, and those of the respiratory system, are the chief causes of mortality ; miasmatic diseases (chiefly *continued fevers*), and those of the nervous system, being next to them.

4. In *Newfoundland* the sickness is represented by 980 admissions, and the mortality by 6.72 deaths per 1000 of mean strength. The most prevalent diseases are *sore throat*, *influenza*, *bronchitis*, and *rheumatism*.

5. In *British Columbia* the sickness is represented by 702 admissions per 1000 mean strength, and the mortality by 14.52 deaths

per 1000 ; but of the eight deaths which occurred during four years, five were by drowning, and one that of a man frozen to death ; so that two only, or in the ratio of 0.36 per 1000 of mean strength, were the result of disease. *Influenza* and *venereal* are the prevailing diseases.

IV. **West India Stations.**—1. Among **White Troops** of the *Windward* and *Leeward* Command the sickness is represented by 1112 admissions per 1000 of mean strength, and the mortality by 10.91 deaths per 1000. The *miasmatic* diseases, arranged in the order of their prevalence, are as follow : *Paroxysmal fevers*, represented by 282 admissions ; *ophthalmia*, by 116 ; *dysentery*, *diarrhœa*, *cholera*, by 46 ; *continued fevers*, by 39 ; and *rheumatism*, by 26 admissions per 1000 of mean strength. *Yellow fever* sometimes prevails. *British Guiana* is the most sickly station ; *Trinidad* the most deadly.

Among **Black Troops** the sickness is represented by 915 admissions, and the mortality by 20.89 deaths per 1000 of mean strength. *Miasmatic* and *enthetic* diseases are the most prevalent, and *tubercular* and *miasmatic* the most fatal. The *miasmatic* diseases, arranged in the order of their prevalence, are as follow : *Paroxysmal fevers*, represented by 94 admissions per 1000 of mean strength ; *rheumatism*, by 86 ; *eruptive fevers*, by 28 ; *sore throat* and *influenza*, by 27 ; *ophthalmia*, by 25 ; and *dysentery*, *diarrhœa*, and *cholera*, by 18 admissions per 1000. *Tubercular diseases* are most fatal to this class of troops, causing nearly one-third of all the deaths ; next to these *rank diseases of the respiratory system*, *diseases of the nervous system*, *paroxysmal fevers*, and *dysentery* and *diarrhœa*.

2. The sickness in *Jamaica* among **White Troops** is represented by 896 admissions per 1000 mean strength, and the mortality by 14.08 deaths. The most prevalent of the *miasmatic* diseases are as follow : *Ophthalmia*, represented by 117 admissions per 1000 mean strength ; *continued fever*, by 74 ; *paroxysmal fevers*, by 63 ; *dysentery*, *diarrhœa*, and *cholera*, by 42 ; *rheumatism*, by 18 ; *sore throat* and *influenza*, by 9 admissions per 1000.

The most fatal diseases are *paroxysmal* and *continued fevers*.

Among the **Black Troops** in *Jamaica* the sickness is represented by 1090 admissions, and the mortality by 28.61 deaths per 1000 of mean strength.

The most prevalent *miasmatic* diseases are *paroxysmal fevers*, represented by 277 admissions per 1000 of mean strength ; *rheumatism*, by 64 ; *dysentery*, *diarrhœa*, and *cholera*, by 24 ; *continued fever*, by 22 ; *ophthalmia*, by 21 ; *sore throat* and *influenza*, by 18 admissions per 1000.

*Tubercular diseases* and those of the respiratory system are the most fatal, causing upwards of two-fifths of all the deaths. Next to them are *paroxysmal fevers*, and diseases of the digestive and nervous systems.

3. In the *Bahamas*, among **Black Troops**, the sickness is represented by 841 admissions, and the mortality represented by 29.08 deaths per 1000 of mean strength.

*Miasmatic diseases* are the most prevalent, and *tubercular* the

most fatal. The mortality is also high from those of the respiratory system.

4. In *Honduras*, among the **Black Troops**, the sickness is represented by 1164 admissions, and the mortality by 1823 deaths per 1000 of mean strength.

The prevalent diseases are *miasmatic*. Skin diseases also abound, especially *eczema*, *ulcers*, and *boils*.

V. **West African Stations**.—1. In *Sierra Leone* the sickness is represented by 740 admissions, and the mortality by 29.53 deaths per 1000 of mean strength.

2. At the *Gambia* the sickness is represented by 978 admissions, and the mortality by 33.74 deaths per 1000 of mean strength.

3. On the *Gold Coast* the sickness is represented by 624 admissions, and the mortality by 26.45 deaths per 1000 of mean strength.

The *Gambia* and *Sierra Leone* seem to be the most sickly; but the average duration of the cases of sickness, and the mortality compared with the admissions, is greatest on the *Gold Coast*.

The most prevalent miasmatic diseases in *Sierra Leone* are as follow:

*Paroxysmal fevers*, represented by 98 admissions per 1000 mean strength; *rheumatism*, by 56 per 1000; *dysentery* and *diarrhœa*, by 20; *eruptive fevers*, by 23 per 1000.

The most prevalent on the *Gambia* are,—

*Paroxysmal fevers*, represented by 294 admissions per 1000 of mean strength; *dysentery*, *diarrhœa*, and *cholera*, by 58 per 1000; *eruptive fevers*, by 44 per 1000; *rheumatism*, by 28 per 1000; *ophthalmia*, by 13 admissions per 1000 men.

The most prevalent on the *Gold Coast* are,—

*Dysentery*, *diarrhœa*, and *cholera*, represented by 31 admissions per 1000 mean strength; *rheumatism*, by 26 per 1000; *ophthalmia*, by 19 per 1000; *paroxysmal fever*, by 11 admissions per 1000. *Small-pox* and *measles* are sometimes epidemic; and *Guinea-worm*, among parasitic diseases, holds a prominent place. Indeed, in 1861, this affection was the cause of one-third of the admissions into hospital; and it is worthy of remark that in that year not a single case occurred among the troops at *Sierra Leone* and the *Gambia*.

VI. **St. Helena**.—The sickness is represented by 880 admissions per 1000 mean strength; the mortality, by 11.28 deaths per 1000.

The prevalent diseases are *dysentery* and *diarrhœa*, represented by 104; *continued fever*, by 124; *ophthalmia*, by 27; *paroxysmal fever*, by 32; *rheumatism*, by 26; *sore throat* and *influenza*, by 13 admissions per 1000 of mean strength.

The most fatal diseases are those of the nervous system, tubercular diseases, and *continued fevers*. *Intemperance* prevails to a great extent.

VII. **Cape of Good Hope**.—The sickness is represented by 873 admissions per 1000 mean strength; the mortality, by 10.87 deaths per 1000.

The most prevalent miasmatic diseases are *ophthalmia*, represented by 91 admissions per 1000 mean strength; *continued fever*, by 49; *rheumatism*, by 41; *dysentery*, *diarrhœa*, and *cholera*, by 45;

*sore throat* and *influenza*, by 20; *paroxysmal fevers*, by 9 admissions per 1000.

The most fatal of these diseases is *continued fever*. Intemperance prevails to a large extent, and is among the chief causes of the prevalence of diseases of the heart in this station (NICHOLSON, TAYLOR). *Ocular* and *cardiac diseases* are the cause of the discharge of half the invalids from the Cape. The mortality by diseases of the circulatory system amounts to 1.58 per 1000 of mean strength annually.

VIII. **Island of Mauritius.**—The sickness is represented by 915 admissions, and the mortality by 25.04 deaths per 1000 of mean strength. This ratio of mortality, however, may be considered as above the usual average, having been raised by the prevalence of a very fatal epidemic of cholera in 1862.—On the average of the three preceding years it only amounted to 17.39 per 1000.

The prevalent diseases are *dysentery* and *diarrhœa*, represented by 179 admissions per 1000 men; *continued fevers*, by 68; *spasmodic cholera*, by 25; *rheumatism*, by 25; *ophthalmia*, by 19; *sore throat* and *influenza*, by 10 admissions per 1000 men.

The most fatal diseases are *spasmodic cholera*, *dysentery* and *diarrhœa*, and *continued fevers*.

IX. **Ceylon.**—The sickness among **White Troops** is represented by 1513 admissions, and the mortality by 23.55 deaths per 1000 of mean strength.

The most prevalent diseases of the miasmatic class are *dysentery*, *diarrhœa*, and *cholera*, represented by 222 admissions per 1000 men; *paroxysmal fever*, by 121; *ophthalmia*, by 107; *rheumatism*, by 75; *continued fever*, by 44.

The fatal diseases are *dysentery*, *diarrhœa*, and *cholera*; diseases of the digestive system, particularly *hepatitis*, *tubercular diseases*, and those of the *nervous system*.

Among the **Black Troops** the sickness is represented by 1064 admissions, and the mortality by 11.97 deaths per 1000 of mean strength.

The prevalent diseases are *paroxysmal fever*, represented by 391 admissions; *continued fever*, by 60; *ophthalmia*, by 75; *rheumatism*, by 46; *dysentery*, *diarrhœa*, and *cholera*, by 56; *eruptive fevers*, by 10 admissions per 1000 men.

The fatal diseases are *spasmodic cholera*, *dysentery* and *diarrhœa*, and *paroxysmal fevers*.

Trincomalee is the most sickly station. Ague prevails there from the marshy soil of *paddy* fields, and cocoanut gardens.

X. **Australasia.**—1. *Australia and Tasmania.* The sickness is represented by 726 admissions per 1000 of mean strength, with a mortality of 15.51 deaths per 1000.

The miasmatic diseases, in the order of their greatest prevalence, are *dysentery*, *diarrhœa*, and *cholera*, represented by 87 admissions per 1000; *rheumatism*, by 51; *continued fever*, by 21; *sore throat* and *influenza*, by 50; *ophthalmia*, by 47 admissions per 1000 men.

The most fatal diseases in this command are the *tubercular*, which caused 4.67 deaths per 1000 of strength. Diseases of the *circulatory*



- system gave rise to 2.34; and those of the nervous system to 2.13. Of the miasmatic diseases, *continued fever* is the most fatal, the deaths by it amounting to 1.06 per 1000 of the strength.

2. *New Zealand*. The sickness is represented by 595 admissions per 1000, and the mortality by 13.25 deaths. But of these 4.16 were the result of wounds in action, leaving a proportion of only 9.09 per 1000 resulting from the ordinary contingencies of colonial service.

The miasmatic diseases, in the order of greatest prevalence, are *ophthalmia*, represented by 50 admissions per 1000 men; *dysentery* and *diarrhœa*, represented by 44; *rheumatism*, by 37 admissions per 1000; *sore throat* and *influenza*, by 36; *continued fever*, by 17; *paroxysmal fever*, by 8 admissions per 1000 men.

The most fatal diseases are those of the *tubercular class*. *Continued fever* gave rise to only .53 deaths per 1000 of the strength; and *dysentery* and *diarrhœa* caused exactly the same proportion.

XI. *China*.—The sickness among **European Troops** is represented by 1968 admissions, and the mortality by 57.81 deaths per 1000. The sickness among **Native Troops** is represented by 1724 admissions, and the mortality by 33.53 per 1000.

1. In *Southern China* the sickness among **European Troops** is represented by 2340 admissions per 1000, with a mortality represented by 35.49 per 1000.

The miasmatic diseases, in the order of greatest prevalence, are as follow: *Paroxysmal fevers*, represented by 571 admissions per 1000; *continued fever*, by 236; *dysentery* and *diarrhœa*, by 211; *ophthalmia*, by 109; *rheumatism*, by 75; *sore throat* and *influenza*, by 16 admissions per 1000 men.

The fatal diseases were *dysentery* and *diarrhœa*, *paroxysmal continued fevers*, and *spasmodic cholera*.

The sickness amongst **Native Troops** in *Southern China* is represented by 1286 per 1000, and the mortality is represented by 30 per 1000. The diseases arranged in the order of greatest prevalence are as follow: *Paroxysmal fevers*, represented by 603 admissions per 1000 men; *dysentery*, *diarrhœa*, and *cholera*, by 166; *rheumatism*, by 89; *continued fever*, by 25; *sore throat* and *influenza*, by 13; *ophthalmia*, by 12 admissions per 1000 men.

The fatal diseases were *dysentery*, *diarrhœa*, *cholera*, *paroxysmal continued fever*, and *rheumatism*.

2. In *North China* the results are so much affected by the peculiar circumstances of field service in the campaign of 1861, and the military operations around Shanghai in 1862, that they would only mislead if stated as the effect of climate. In 1861, at Tein-tsin, 19 deaths were recorded from *sunstroke* between the 17th and 23d of July, the *maximum* temperature during that period ranging from 95° to 108° Fahr. in the shade—giving a maximum average of 104° 6'; and the minimum temperature ranged from 73° to 83° Fahr. in the shade; the average being 79° 2' (Dr. LAMPREY). Information on this important point is exceedingly meagre. The experience of Surgeon Butler at Meean Meer led him to write that "when the

thermometer ranges beyond 98° Fahr., in crowded barracks, cases of heat apoplexy almost invariably occur." (See p. 379, *ante*.)

In 1862 cholera broke out in the field force in the neighborhood of Shanghai, and cut off 58 per 1000 of the strength. It also attacked the small garrison left at Taku, and the mortality in it amounted to 40 per 1000.

**XII. Stations in India.**—The prevalence of sickness, on the average of the three years 1860–62, as shown in the *Army Medical Department Reports*, is represented by 1818 admissions per 1000 mean strength, and the mortality is represented by 32.45 deaths. The relative sickness and mortality are distributed over the three Presidencies as follow :

The sickness in *Bengal* is represented by 1944 admissions, and the mortality by 37.43 deaths per 1000 ; the sickness in *Madras*, by 1342 admissions, and the mortality by 19.82 deaths per 1000 ; the sickness in *Bombay*, by 1844 admissions, and the mortality by 27.15 deaths per 1000.

In all three Presidencies *paroxysmal fevers* form the group of miasmatic diseases which gives rise to the highest proportion of admission ; but in Madras these are scarcely one-third as prevalent as in Bengal and Bombay. *Continued fevers* are very common in Bengal, and *dysentery* and *diarrhœa* are the cause of the admission into hospital of between 150 and 160 per 1000 of the strength in each of the Presidencies. *Spasmodic cholera* has not only been the cause of the highest rate of mortality in all three Presidencies, but has been a source of much greater mortality in Bengal than in the others. Next to it come *dysentery* and *diarrhœa*, and after them *paroxysmal fevers*.

*The lives wasted in India in each year* of service (according to the Commissioners' Report) appear to have been greater in the Queen's regiments than in the Company's regiments. Half of the deaths take place during the first five years ; and the numbers invalided increase slightly in each quinquennial period. The decrement year by year of the force in India has been such that 1000 effectives are reduced to 96 men in 20 years (that is, by death alone, to 344—by death and invaliding, to 216—by death, invaliding, and other causes, to 96). The mean term of service in India has been 8.6 years, and 11 recruits are required annually to every 100 men ; and to maintain 85,856 men 10,000 annual recruits are required ; and these are reduced to less than half their original numbers in *eight* years. The half of a regular army so constituted consists of men who have served less than six years ; and not more than a fourth of the men are veterans of ten years' standing, on whom the discipline and solidity of an army greatly depend.

*The endemic diseases of India* in the order of greatest prevalence are *paroxysmal fevers*, *continued fevers*, *dysentery*, *rheumatism*, *ophthalmia*, *spasmodic cholera*, *sore throat*, and *influenza*. Arranged in the order of comparative mortality, the diseases are as follow : *Spasmodic cholera*, *dysentery*, *diarrhœa*, and *continued* and *paroxysmal fevers*. These are especially the diseases of the sultry plains of India.

*The Fevers* are especially the paludal fevers; but both typhus and typhoid fevers are now known to occur in India.

*The Dysentery of India* is most prevalent in the plains during the hot and rainy seasons; and amongst British soldiers it is computed that eleven cases of dysentery occur to one amongst the native soldiery. The cases of dysentery present "a spectacle of distress of as pitiable a kind as can be found in the history of human suffering."

*Diseases of the Liver.*—No statistics can give any idea of the extent of these diseases; and in acute inflammation, so prevalent in the plains, the danger to life is imminent from the first, and, in the event of recovery, impairment of health more or less permanent is certain.

*Cholera* is the most acute of acute diseases. Troops, both European and Native, while on the march, are more liable to seizure by this disease; and when the attacks have occurred in cantonments after the march, the prevalence of the epidemic is generally in proportion to the length of march.

The annual rate of mortality among soldiers is 10 per 1000 in England; in India, according to the *Commissioners' Report*, it is 67 per 1000 (Bengal), of which 58 per 1000 is due to zymotic diseases, the fevers killing 17 men in 1000; dysentery and liver disease, 20; cholera and diarrhœa, 18 men in 1000. *Delirium tremens*, *catarrh*, *syphilis*, *rheumatism*, and *scurvy*, are much more fatal in India than in England.

Nearly all the diseases fatal in India are accompanied by profuse discharges, with which the air, water, linen, bedding, closets, walls of hospitals, and barracks become more or less infected; so that the *materies morbi* come into contact with all the inmates of buildings where the disease prevails. In India the soldier's sickness is doubled. As to how far this sickness and mortality are aggravated by the unsanitary state of the towns and the mode of life in India, the reader is referred to the *Report of the Royal Commission on the Sanitary State of the Army in India*, and to the second edition of Miss Nightingale's pamphlet, entitled *How to Live and Not Die in India*.

As legitimate deductions from the preceding statements, the following summaries of results are here given:

(A.)

Stations of the British Army, arranged in the Order of the GREATEST NUMBER OF ANNUAL ADMISSIONS per 1000 of Mean Strength.	Annual Admissions per 1000 Mean Strength.	Annual Mortality per 1000 Mean Strength.
<b>WHITE TROOPS.</b>		
1. South China, . . . . .	2340	35.49
2. Bengal, . . . . .	1944	37.43
3. Bombay, . . . . .	1844	27.15
4. Ceylon, . . . . .	1513	23.53
5. Madras, . . . . .	1342	19.82
6. Windward and Leeward Command, .	1112	10.91
7. United Kingdom, . . . . .	1025	9.35
8. Newfoundland, . . . . .	980	6.72
9. Mauritius, . . . . .	915	25.04
10. Malta, . . . . .	906	12.31
11. Jamaica, . . . . .	896	14.08
12. Gibraltar, . . . . .	894	8.86
13. St. Helena, . . . . .	880	11.28
14. Cape of Good Hope, . . . . .	873	10.87
15. Ionian Islands, . . . . .	807	9.11
16. Australia, . . . . .	726	15.51
17. Bermuda, . . . . .	637	11.79
18. Canada, . . . . .	622	9.07
19. New Zealand, . . . . .	595	13.25
20. Nova Scotia, &c., . . . . .	581	7.13
<b>COLONIAL CORPS.</b>		
1. South China,* . . . . .	1820	34.83
2. Honduras,† . . . . .	1164	18.23
3. Jamaica,† . . . . .	1090	28.61
4. Ceylon,‡ . . . . .	1064	11.79
5. Gambia,† . . . . .	978	33.74
6. Windward and Leeward Command,† .	915	20.89
7. Bahamas,† . . . . .	841	29.08
8. Sierra Leone,† . . . . .	740	29.43
9. Gold Coast,§ . . . . .	624	26.45

\* Asiatics.

† Africans and Colored Creoles.

‡ Cingalese and Natives of India.

§ Africans.

(B.)

Stations of the British Army, arranged in the Order of the GREATEST ANNUAL MORTALITY per 1000 of Mean Strength.	Annual Admissions per 1000 Mean Strength.	Annual Mortality per 1000 Mean Strength.
EUROPEAN TROOPS.		
1. Bengal, . . . . .	1944	37.43
2. South China, . . . . .	2340	35.49
3. Bombay, . . . . .	1844	27.15
4. Mauritius, . . . . .	915	25.04
5. Ceylon, . . . . .	1513	23.53
6. Madras, . . . . .	1342	19.82
7. Australia, . . . . .	726	15.51
8. Jamaica, . . . . .	896	14.08
9. New Zealand, . . . . .	595	13.25
10. Malta, . . . . .	906	12.31
11. Bermuda, . . . . .	637	11.79
12. St. Helena, . . . . .	880	11.28
13. Windward and Leeward Command, .	1112	10.91
14. Cape of Good Hope, . . . . .	873	10.87
15. United Kingdom, . . . . .	1025	9.35
16. Ionian Islands, . . . . .	807	9.11
17. Canada, . . . . .	622	9.07
18. Gibraltar, . . . . .	894	8.86
19. Nova Scotia, &c., . . . . .	581	7.13
20. Newfoundland, . . . . .	980	6.72
COLORED CORPS.		
1. South China,* . . . . .	1820	34.83
2. Gambia,† . . . . .	978	33.74
3. Sierra Leone,† . . . . .	740	29.53
4. Bahamas,† . . . . .	841	29.08
5. Jamaica,† . . . . .	1090	28.61
6. Gold Coast,† . . . . .	624	26.45
7. Windward and Leeward Command,† .	915	20.89
8. Honduras,† . . . . .	1164	18.23
9. Ceylon,‡ . . . . .	1064	11.97

“ Dr. Balfour’s valuable reports (at the period of their earliest appearance) shows at a glance the great diversity in the amount of sickness and mortality to which British troops were liable (in former times) at these stations, while serving in our widely extended colonial possessions; and it will be seen how great has been the reduction, especially in the mortality of the troops, during the four years comprised in the preceding tables, as compared with the rates when Dr. Balfour and those with whom he labored first brought them to the notice of the Secretary of State for War. Leaving out of consideration Sierra Leone as an extreme instance of unhealthiness, and from which it has been deemed expedient altogether to withdraw European troops, the admissions into hospital have ranged, during the twenty years subsequent to 1836, between 529 per 1000 of the

\* Asiatics.

† Africans and Colored Creoles.

‡ Africans.

§ Cingalese and Natives of India.



strength in New Zealand and 2117 per 1000 in Bombay; while the mortality has ranged between an average of 11 per 1000 in Newfoundland and 76.2 per 1000 in Bengal. Not only, however, do these great differences exist in the amount of sickness and mortality at different stations on the average of a series of years, but they occur to as great an extent in different years upon the same station. Thus, in the period of 1817 to 1836, the sickness ranged in the Windward and Leeward Command between 1512 and 2365 per 1000; in Jamaica, between 1389 and 2423; in Ceylon, between 1216 and 2895. At Gibraltar it ranged between 631 and 1498; and even in Canada it varied from 847 to 1409 per 1000. But the difference in the mortality in different years is even more striking: thus, in the Windward and Leeward Command it varied between 43 and 162; in Jamaica, between 61 and 307; in Ceylon, between 34 and 218; in Gibraltar, between 8 and 128; in Canada, between 9 and 48; and in Nova Scotia between 7 and 40 per 1000. These facts strikingly illustrate the necessity for a prolonged period of observation to obtain a fair average, and for great caution in making deductions from observations extending over limited periods and insufficient numbers.

“Another very striking fact shown by Dr. Balfour’s tables is the great reduction which has taken place during the last twenty years in the mortality at some of the stations—a result which seems fairly attributable, in a considerable degree at least, to the removal of those causes of disease which were brought to light by the Statistical Reports, and to the adoption of the measures then recommended for improving the health of the troops. The most striking examples of this are to be found in Jamaica, where the deaths have fallen from an average of 128 to 60.8 per 1000; in Newfoundland, from 37.7 to 11; in St. Helena, from 25.4 to 12.3; and in Ceylon, from 74.9 to 38.6 per 1000.

“But, with a view to judge by what measures the health of the troops was likely to be improved, it was necessary to ascertain the diseases to which the sickness and mortality were attributable. Abstracts were accordingly prepared, showing the diseases by which every admission into hospital and death had been occasioned for a period, in most of the colonies, of twenty years. These were again grouped in classes, and the results are as follow:

“Fevers will be found the greatest cause of mortality in Jamaica, the West Indies, and Bermuda, and, prior to 1837, in Ceylon, Gibraltar, and the Ionian Islands. Diseases of the lungs have been most fatal among the troops serving at home, particularly the Foot Guards, as well as in the West Indies, Jamaica, Bermuda, Canada, and Nova Scotia; those of the liver in Ceylon and India, and also in Mauritius and St. Helena, prior to 1837; while diseases of the stomach and bowels have been a source of considerable mortality in India, Ceylon, West Indies, Mauritius, and, prior to 1837, also at St. Helena. A comparative exemption is enjoyed by the black troops from the fatal forms of fever by which the white troops have suffered so severely; but there is a great liability among them to pulmonary disease in the West Indies and Jamaica. A comparison of the rates of mortality, prior and subsequent to 1837, shows that the improvement in the health of the troops has been attributable to different causes in different colonies. Thus, in the West Indies and St. Helena it has shown itself chiefly in the reduction of deaths by *diseases of the bowels*; in Jamaica, Gibraltar, and the Ionian Isles, by *fevers*; and in Ceylon and Bermuda, by both these classes.

“The influence of age upon mortality in the army is a question of much

importance with reference to the expediency of exacting from the soldier a long continuous service in our colonies, or of shortening that service, and by frequent reliefs affording him change of climate.

“The most striking feature in this particular is the rapidly progressive increase in the mortality with advancing years in tropical climates—a result completely at variance with the prevalent opinion of the advantage of long-continued residence in such climates, and of much importance in determining the question of frequent reliefs.”

Although military statistics may not be proper *criteria* of the healthiness of a place, nor of the diseases affecting the civil community, yet the information is of great value, and the following tables, kindly prepared for this edition by Dr. T. G. Balfour, F.R.S., show at a glance the *sickness* and *mortality* of the British army at all the stations occupied by it, and the classes of diseases by which *sickness* and *mortality* are caused; and, if compared with previous statements, will show the great reduction which has taken place in the amount of sickness and mortality compared with former times:

TABLES SHOWING THE PROPORTION OF ADMISSIONS INTO HOSPITAL, AND DEATHS PER 1000 OF MEAN STRENGTH, AT EACH OF THE FOLLOWING STATIONS, ON THE AVERAGE OF THE PERIODS NOTED.

## I.—EUROPEAN TROOPS.

Stations and Period of Observation.	United Kingdom, 1860-62.		Gibraltar, 1860-62.		Malta, 1860-62.		Ionian Islands, 1859-62.		Hermode, 1860-62.		Nova Scotia, &c., 1860-62.		Canada, 1860-62.		Newfoundland, 1860-62.		Windward and Leeward Command, 1860-62.		Jamaica, 1860-62.	
	Admissions.	Deaths.	Admissions.	Deaths.	Admissions.	Deaths.	Admissions.	Deaths.	Admissions.	Deaths.	Admissions.	Deaths.	Admissions.	Deaths.	Admissions.	Deaths.	Admissions.	Deaths.	Admissions.	Deaths.
Miasmatic Diseases, . . . . .	223	1.06	284	4.82	420	5.98	328	4.68	188	.91	120	.69	135	1.09	376	...	536	4.45	339	6.55
Enthetic Diseases, . . . . .	352	.12	213	...	111	...	120	.06	61	...	131	...	133	.05	44	...	104	...	103	...
Dietic Diseases, . . . . .	7	.06	13	.18	6	.09	5	.13	26	.23	12	.57	19	.16	36	1.12	26	.20	26	.39
Parasitic Diseases, . . . . .	36	.01	6	...	13	...	5	...	6	...	10	...	7	...	6	...	3	...	3	...
Mathetic Diseases, . . . . .	2	.14	3	.05	3	.04	1	.19	2	.23	1	.23	2	...	7	...	6	...	2	...
Tubercular Diseases, . . . . .	19	3.48	7	1.15	11	2.14	10	1.27	9	2.04	14	2.29	7	1.41	11	1.12	10	1.21	5	1.96
Diseases of the Nervous System, . . . . .	20	.68	19	.23	16	.56	17	.63	23	2.94	14	.46	16	1.09	54	2.24	33	2.21	15	1.57
“ “ Circulatory System, . . . . .	9	.73	6	.64	6	.51	6	.44	5	.91	3	.91	3	.73	26	...	4	.81	6	.78
“ “ Respiratory System, . . . . .	98	1.47	56	.99	47	.90	46	.57	64	...	65	1.03	66	1.46	123	2.24	32	.40	48	...
“ “ Digestive System, . . . . .	38	.49	39	.23	43	.60	42	.32	62	1.13	32	.34	38	.42	58	...	75	.61	92	1.17
“ “ Urinary System, . . . . .	3	.10	3	.23	2	.21	2	...	1	...	3	...	2	.16	2	...	1	...	2	...
“ “ Reproductive System, . . . . .	7	...	16	...	10	...	5	...	10	...	3	...	6	...	4	...	13	...	2	...
“ “ Locomotive System, . . . . .	3	...	5	.09	3	...	3	.06	3	...	2	...	3	...	11	...	5	...	1	...
“ “ Integumentary System, . . . . .	121	.68	137	...	136	...	136	...	163	...	73	...	85	.16	66	...	161	...	132	...
Diseases of Nutrition, . . . . .	2	.93	2	...	1	...	4	...	2	...	...	...	1	...	...	...	2	...	3	...
Accidents, . . . . .	80	.59	77	.41	71	1.07	74	.51	78	2.04	95	.69	97	1.86	145	...	96	.61	131	...
Battle, . . . . .	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...
Homicide, . . . . .	...	.02	...	.14	...	...	...	.06	...	...	...	.11	...	.21	...	...	...	...	...	.30
Suicide, . . . . .	...	.26	...	.46	...	.17	...	.13	...	1.36	...	.11	...	.26	...	...	...	.40	...	1.17
Execution, . . . . .	...	.02	...	.14	...	.04	...	.06	...	...	...	...	...	.05	...	...	...	...	...	...
Corporal Punishment, . . . . .	2	...	3	...	3	...	2	...	1	...	1	...	1	...	3	...	...	...	3	...
Disease not specified, . . . . .	2	...	3	...	3	...	2	...	2	...	2	...	1	...	6	...	4	...	2	...
No appreciable Disease, . . . . .	...	...	1	...	...	...	...	...	1	...	...	...	...	...	...	...	...	...	...	...
Total, . . . . .	1025	9.35	894	8.86	906	12.31	807	9.11	637	11.79	581	7.43	623	9.07	980	6.72	1112	10.91	896	14.08

I.—EUROPEAN TROOPS—Continued.

Stations and Period of Observation.	St. Helena, 1860-62.		Cape of Good Hope, 1860-62.		Mauritius, 1860-62.		Ceylon, 1860-62.		Australia and Tasmania, 1860-62.		New Zealand, 1860-62.		South China, 1860-62.		Bengal, 1860-62.		Madras, 1860-62.		Bombay, 1860-62.	
	Admissions.	Deaths.	Admissions.	Deaths.	Admissions.	Deaths.	Admissions.	Deaths.	Admissions.	Deaths.	Admissions.	Deaths.	Admissions.	Deaths.	Admissions.	Deaths.	Admissions.	Deaths.	Admissions.	Deaths.
Miasmatic Diseases, . . . . .	356	2.61	283	2.17	355	17.16	597	9.15	227	2.13	206	1.06	1236	24.21	1114	24.84	568	8.12	1049	16.82
Exanthetic Diseases, . . . . .	97	...	182	.06	78	...	233	.27	121	...	30	...	465	...	341	.32	269	.35	317	.31
Dietic Diseases, . . . . .	43	.43	11	.53	17	.14	18	.27	24	...	11	.60	30	.36	9	.06	17	.12	16	.19
Parasitic Diseases, . . . . .	3	...	15	...	10	...	4	...	1	...	13	...	5	...	7	...	9	...	12	...
Diathectic Diseases, . . . . .	5	.43	3	.12	4	...	4	.27	6	.42	6	.23	5	...	6	.22	5	.18	7	.19
Tubercular Diseases, . . . . .	3	1.74	9	1.18	12	1.69	18	3.08	18	4.67	8	2.04	9	.95	11	2.48	16	1.88	9	1.53
Diseases of the Nervous System, . . . . .	27	2.17	24	1.35	25	1.13	64	2.50	24	2.13	22	.60	32	2.37	34	2.28	33	1.59	33	1.53
"  "  Circulatory System, . . . . .	8	.43	22	1.58	11	.56	17	.83	13	2.34	7	.98	13	.59	10	.62	15	1.27	12	.53
"  "  Respiratory System, . . . . .	47	.87	57	.94	62	.56	86	1.10	52	1.06	64	.76	105	1.66	57	1.04	49	.59	51	.96
"  "  Digestive System, . . . . .	55	.43	46	.94	89	1.41	177	3.88	46	.64	33	.68	117	2.26	146	3.65	120	3.41	122	3.64
"  "  Urinary System, . . . . .	4	...	2	.18	3	...	6	.27	4	.64	2	.23	2	.12	2	.14	2	.18	3	.96
"  "  Reproductive System, . . . . .	3	...	9	...	11	...	15	...	1	...	4	...	6	...	11	.02	12	...	6	...
"  "  Locomotive System, . . . . .	2	...	4	...	5	...	4	.27	3	...	3	...	5	...	5	.03	5	.03	4	...
"  "  Integumentary System, . . . . .	78	...	103	...	136	...	145	...	71	...	81	.33	150	.12	108	.07	107	.18	102	.66
Diseases of Nutrition, . . . . .	...	...	1	...	16	.14	8	...	...	...	1	...	3	.71	8	.14	6	.21	6	...
Accidents, . . . . .	143	2.17	89	1.52	76	1.27	127	1.30	99	1.06	86	1.44	94	1.66	74	.73	91	1.27	89	1.68
Battle, . . . . .	...	...	...	...	...	...	...	...	...	...	10	4.16	...	...	...	...	...	...	...	...
Homicide, . . . . .	...	...	...	.66	...	...	...	...	...	...	...	.08	...	.12	...	.03	...	...	...	...
Suicide, . . . . .	...	...	...	.18	...	.98	...	.27	...	.31	...	...	...	.13	...	.29	...	.06	...	.34
Execution, . . . . .	...	...	...	.66	...	...	...	...	...	...	...	.08	...	...	...	.01	...	...	...	.03
Corporal Punishment, . . . . .	1	...	4	...	3	...	3	...	2	...	3	...	...	...	1	...	1	...	1	...
Disease not specified, . . . . .	5	...	10	...	8	...	8	...	14	.21	6	.68	32	.24	6	.36	8	.38	2	.22
No appreciable Disease, . . . . .	...	...	...	...	1	...	...	...	1	...	...	...	...	...	...	...	...	...	...	...
Total, . . . . .	800	11.28	673	10.87	913	25.64	1513	23.35	726	16.51	596	13.26	3346	25.49	1944	37.43	1242	19.82	1944	27.15

## II.—COLONIAL TROOPS.

Stations and Period of Observation.	Windward and Leeward Command, 1859-62.		Jamaica, 1859-62.		Bahamae, 1859-62.		Bonaire, 1859-62.		St. Pierre, 1859-62.		Gambia, 1859-62.		Gold Coast, 1859-62.		Ceylon, 1859-62.		China, 1859-62.	
	Admissions.	Deaths.	Admissions.	Deaths.	Admissions.	Deaths.	Admissions.	Deaths.	Admissions.	Deaths.	Admissions.	Deaths.	Admissions.	Deaths.	Admissions.	Deaths.	Admissions.	Deaths.
Miasmatic Diseases.	300	5.	437	6.55	388	5.54	567	3.64	227	4.92	458	4.60	103	4.96	653	5.48	1275	23.98
Enthetic Diseases.	201	...	284	1.04	173	.69	98	...	142	...	153	1.63	41	...	80	...	95	.73
Dietic Diseases.	1	...	3	...	3	...	3	2.19	1	.70	3	...	2	...	1	...	3	...
Parasitic Diseases.	1	...	3	...	6	...	12	...	19	...	14	...	223	...	69	...	43	...
Dysenteric Diseases.	6	.44	6	1.04	4	.69	6	...	4	...	9	2.30	1	...	4	.60	13	.36
Tubercular Diseases.	16	5.86	15	6.20	19	10.39	5	2.19	16	7.74	14	9.20	6	3.31	3	1.	3	.72
Diseases of the Nervous System.	17	1.96	24	2.07	16	2.08	18	1.46	14	4.22	15	1.63	12	3.31	9	.83	20	.80
"  "  Circulatory System.	4	1.09	2	.69	2	...	4	2.91	1	...	2	1.53	...	83	2	1.	2	.48
"  "  Respiratory System.	83	2.39	73	6.20	55	6.23	65	.73	67	4.92	94	8.43	29	4.13	43	.66	84	2.63
"  "  Digestive System.	37	.65	41	2.42	18	2.08	49	.73	26	4.32	38	.77	24	2.47	32	.50	65	.84
"  "  Urinary System.	3	.65	4	...	2	...	3	...	1	...	4	.77	3	...	...	...	2	...
"  "  Reproductive System.	11	...	9	...	19	...	15	...	5	...	3	...	6	...	5	...	2	.12
"  "  Locomotive System.	3	...	2	...	1	...	4	...	13	.70	6	.77	12	...	1	...	1	...
"  "  Integumentary System.	140	.22	119	.84	73	.69	198	.73	124	...	96	...	99	...	136	...	197	.24
Diseases of Nutrition.	2	.44	...	...	2	...	1	...	1	...	1	...	...	...	1	.17	31	2.99
Accidents.	73	.65	60	.69	57	...	106	1.46	56	...	57	...	56	4.96	74	.50	71	.96
Battle.	...	...	...	...	...	...	...	...	16	2.11	6	...	...	...	...	...	...	...
Homicide.	...	.65	...	.34	...	...	...	.73	...	...	1	.77	...	...	...	...	...	...
Suicide.	...	.66	...	.69	...	...	...	.73	...	...	...	.77	...	.83	...	...	...	...
Execution.	...	.22	...	.34	...	...	...	...	...	...	...	.77	...	1.65	...	...	1	...
Corporal Punishment.	6	...	6	...	2	...	9	...	6	...	5	...	6	...	1	...	1	...
Not specified.	14	...	4	...	6	...	2	.73	2	...	5	...	3	...	1	...	2	.34
Total.	916	36.89	1000	28.61	841	20.08	1164	18.23	740	29.53	978	33.74	624	26.46	1664	11.97	1920	34.83



The natives of this country who are now scattered throughout Hindostan and the Indian Archipelago, on both sides of Africa, a few hundred miles north of the Cape, along the southern shores of the Mediterranean, in the West Indies, South America, and elsewhere, also illustrate the necessity of a change of climate by their habits. Few of them ever return to their native land with constitutions unimpaired; and in no case do the British natives, whose means are not absolutely insignificant, attempt to rear up their children in any of those tropical regions. If they do so, "parents soon mourn over the graves of lost offspring, or sigh on beholding the sickly appearance of those who survive." Of them the adults, and especially the females, suffer under hourly increasing morbid influence, and are destined at length to succumb, far within the average limits of longevity that would have been accorded them by any insurance agency in England. On the contrary, when means permit, every sacrifice is made by parents in India, under the name of "*education*," to send their children homeward, that their constitutions may become *retempered* before they are once more exposed to deleterious intertropical influences. It is a significant fact, says Nott, on whose authority these statements are made, that the oldest purely English regiment in India, the "Bombay Tufts," notwithstanding that marriages with British females are encouraged, has never been able, from the time of Charles II, to the present hour, to rear, from births in the corps, *boys* enough to supply its musical band with drummers and fifiers. The same rule holds good with the Dutch in Batavia and other Indian islands. In Algeria, also, the French are beginning to find out that, unless the indigenous Arab or Kabyle will plough the fields for them, *colonization* is hopeless (BOUDIN). At Madagascar the Frenchman, and at Sierra Leone the Englishman, die off in any prolonged attempt at *colonization* (BRYSON). With the Negro races the same phenomena are observed. The Negro is evidently killed by cold. He is the native of the hottest region of the globe, where he goes naked in the scorching rays of the sun, and can lie down and sleep on the ground in a temperature of 150° Fahr.; but if removed from such latitudes to places beyond 40° north, he steadily deteriorates, and ultimately becomes exterminated. The statistics of New England, New York, and Philadelphia prove this, where the mortality is double that of the white population (NOTT). The acclimation of the Negro has been overrated. They never, according to Dr. Nott, become proof against intermittents and their sequelæ in America.

There is, however, an acclimation, although an imperfect one, against moderately high temperature; and it is also equally true that persons who have gone through this process, and more especially their children, when grown up, are less liable to violent attacks of paludal fevers, when exposed to them, than fresh immigrants from the temperate regions. The course of disease upon fresh arrivals—new-comers—is more rapid, as a rule, and more severe.

The statistics of Sir Alexander Tulloch and Dr. Graham Balfour show very strongly that certain races cannot become acclimated in certain realms, though they may in others, far removed from

their original birthplace. British soldiers and civilians, for instance, enjoy even better health at the Cape Colony than in Great Britain; while the Negro in most regions out of Africa, whether within the tropics, as in the Antilles, or out of the tropics, as at Gibraltar, is gradually being exterminated; and "before a century has passed, the Negro race will almost have disappeared from the British Colonies in the West Indies." In 1817 one of the West India regiments, composed of Negroes, was sent to Gibraltar, and formed part of the garrison there for twenty-two months; the mortality in it was at the rate of 62 per 1000 of the strength annually, or above four times that of the European troops in the same garrison during the same period (BALFOUR). The American statistics of the United States confirm these conclusions (CARREY, DE BOW, NOTT).

Individuals of races pre-eminent among all others for capacity for colonization and acclimation are seen in all parts of the world combating successfully climatic influences, mainly by adapting themselves to new conditions, carefully regulating their mode of life, manners, and customs, so as to suit the climate in which they live. This is the first and main point to be attended to in attempts at acclimation. Food, clothing, and habits must be regulated so as to conform to the wants of the climate of adoption. The *Caucasian* races, particularly the *Semitic*, exemplified in the *Jewish* tribe, appear to be capable of enduring all, or almost all, varieties of climate from Sweden to Aden. The *Gypsies* are another remarkable instance; but it is difficult to say whether they should be classed as *Caucasian* or not. Probably they are not, but are *Turanians* Caucasianized in language merely (BEDDOE).

Of the *European* races the most southern, who are generally darker, seem to have a wider range of acclimation than the *Teutonic*; and of races *not Caucasian*, the *Chinese* appear to be highly favored in this respect. The facts most favorable to the acclimation of the *Teutonic* races are the rapid multiplication of the *German Colonies* in Southern Brazil, and of the *Cape Dutch* in the hot and dry interior of South Africa. It is still doubtful whether our race will not degenerate in Australia, though at present it is remarkably healthy there, owing principally to the absence of malaria. Most of the *Dark races* suffer greatly from *phthisis* when removed to this country. Some of them seem to be inferior to the *Negroes* themselves in this respect (BEDDOE).

In the Island of Ceylon, during a series of years, the comparative ratio of mortality has been noted among five different races of which the troops are composed; and the following are the significant results, illustrating the statements now made from the Statistical Report on Ceylon, by the late Sir Alexander Tulloch and Dr. Balfour:

	Annual deaths per 1000 men.
Natives of Bengal and Madras,	40
Troops recruited on the Coast of Ceylon,	23
Malays,	25
Negro Troops,	50
English Troops,	69

The most minute and reliable information we possess regarding the influence of tropical climates on European races is to be found in the Statistical Reports of Colonel Sir Alexander Tulloch and Dr. G. Balfour, the works of Sir Ranald Martin, already frequently noticed, and lastly, the *Report of the Royal Commission on the Sanitary State of the Army in India*, 1863. A report by the two first-named writers, which includes the stations of Western Africa, St. Helena, the Cape of Good Hope, and the Mauritius, shows that in Western Africa, on the average of eighteen years, every soldier was thrice under medical treatment annually, and nearly half the force annually perished; and when the mortality was at its height, three-fourths of the troops perished annually. About 300 white troops were landed at different times in 1825, and in detachments: nearly every one died, or was shattered in constitution; and what is remarkable, during the whole of this dreadful mortality, a detachment of from forty to fifty black soldiers of the 2d West India Regiment lost only one man, and had seldom any sick in the hospital. No length of residence acclimates the whites in Africa; on the contrary, it tends to their extermination. In like manner, it has been shown that the *native* troops on the Bengal establishment are particularly healthy, while the imported English are the reverse. The Report on India shows most forcibly that much of the sickness and mortality in that country is due to the unhygienic practices which prevail, and to the unsanitary conditions which are suffered to exist in the towns, cantonments, and bazaars of India—where the *causes* of disease are in “a state of absolute perfection, but which have been removed with entire success in this country.” There can be no acclimation to causes of disease; and climate, *per se*, has been made to play the part of a scape-goat for the neglect of sanitary precautions ever since we set foot in India. “Every evil from which British troops have suffered has been laid at its door” (SIR CHARLES NAPIER).

These stern realities must one day or another be grappled with face to face. With the existing provisions for the sanitary state of our army, “the field of distinction in India has hitherto been a great grave-ground—a British Juggernaut.” But, as Miss Nightingale justly observes, “there is not a shadow of proof that India was created to be the grave of the British race. The evidence, on the contrary, is rather in the other direction, and shows that all that the climate requires is that men shall adapt their social habits and customs to it; as, indeed, they must do to the requirements of every other climate under heaven.” The Science of Medicine and Sanitary Science teach us that much may yet be done; and the voice of public opinion will insure that the recommendations of the Commissioners contained in the Report (*Report of the Royal Commission on the Sanitary State of the Army in India*) be eventually carried out. The great work of civilization in India has yet to be commenced. The climate of the East merely exaggerates the causes of disease which exist there, and which have been fostered rather than obviated by the military economy of our troops. The annals of warfare and of campaigns teem with abundant illustrations

which show that the humanizing influence of the Science of Medicine is not sufficiently appreciated by the commanders of armies. Lord Macaulay, in his *History of England*, observes of William Henry Prince of Orange, "that it was too much the habit even of the most humane and generous soldiers to think very lightly of the bloodshed and devastation inseparable from great martial exploits; and the heart becomes steeled, not only by professional insensibility, but by the sterner insensibility which is the effect of a sense of duty." From the days of Nero, the most ferocious, to those of Lord Raglan, the most humane commander that lived, this historical truth has been sufficiently verified. The sufferings of the soldier are apt to be forgotten and lost sight of in the excitement which attends our brilliant martial achievements. The humanizing influence of the medical profession may yet effect a change for the better, when it enjoys a higher position in the organization of the army than it has hitherto done; when a high standard of professional education is demanded of the student of medicine who would enter the service; when the head of the medical department of the army shall have a higher official position than has yet been accorded to him; and when his representative in the field shall have a seat in those councils of war, and a voice in that assembly, where the sanitary position of an army is always in danger of being compromised.

**Persistent Pernicious Influence of Malarious Climates.**—Sufficiently authenticated examples are now on record, which prove the persistent pernicious influences of malaria, both on races of mankind and on bodies of men subjected to their influences for periods of time beyond six or eight weeks, and especially during a temperature above 60°. It is known that after the Walcheren expedition our troops continued to suffer from paludal fevers for five, six, eight, and eleven months after their return to this country, although located in as salubrious quarters as could be procured for them. M. Boudin, also, in his *Lettres sur l'Algérie*, fully shows the persistent pernicious influence of paludal poison on the French and English colonists there. During the recent war against Russia the persistent pernicious influence of the residence of the troops in Bulgaria, during a period of three of the hottest months of the year, continued to make itself more or less manifest throughout the whole of the campaign in the Crimea.

The more chronic and enduring influence of marsh malaria on a race finds abundant illustration amongst the people who inhabit Campagna, Maremma, Pontines, and other insalubrious localities in classic Italy; and in France, in Forez, La Brenne, Sologne, Berry, Dombée, and La Bresse.

Every page of Sir Ranald Martin's classic work *On Climate* goes to establish the belief in the *degrading* influence of the Indian climate, *where malaria prevails*, on European constitutions. The slow, increasing influence of a fever which no acclimation overcomes maintains the high death-rate; and Twining also long since noticed that, "after careful inquiry, he was unable anywhere to find a sample of the third generation from unmixed European stock." Thus upon

European colonization, where malaria is permitted to exist, in India "Nature has set her ban—a blighting interdiction" till the progress of sanitary measures shall make this fine country the great garden of the world.

The characteristics of degeneracy, as shown by paludal races and inhabitants of such regions, may be shortly summed up as follow: A more or less intense state of cachexia, stunted growth, engorgement of the chief viscera, especially of the spleen, languor, and inertia of all the functions—aggravation of ordinary diseases, the superaddition to them of lesions only explicable by the atony and diminished power of reaction of the nervous centres, and finally, a diminished longevity. The influence of the degenerating principle in the sphere of the intellectual and emotional faculties is no less remarkably manifested by the torpor of intelligence, the apathy, a kind of hebetude, passing on under some circumstances to a state of idiocy, and under all to the most extreme indifference (MORELL, and Reviewer in *Med.-Chir. Review*, January, 1858, p. 82).

The length to which this volume has extended will not permit of further remarks on the Geography of Disease. Enough, perhaps, has been written in illustration of some of the more important directions which the study may take, and to show that the subject presents a vast field for investigation, and claims the united exertions of every one in its exploration, who is interested in the progress of the Science of Medicine, of Politics, and of Social Health. The immediate object of the study is to ascertain the laws by which disease is distributed or propagated, or the manner in which certain conditions inimical to health are found to prevail in certain localities or regions, with a view to the prevention of disease by sanitary measures; and how many of the phenomena relative to the regional distribution of disease are elucidated by the facts of *physical geography*.

The sources of information on the subject exist in reliable tables of sickness and mortality, a knowledge of the physical conformation of the earth's surface, and the meteorological agencies to which it is exposed. A *statistical* branch has now been created in the Office of the Army Medical Department of this country, "thus at last affording an official recognition of the value of statistics in the investigation of those questions on which the efficacy of our army so much depends, and by the satisfactory solution of which so much sickness and mortality among soldiers may be prevented, and economy both in life and in money attained."

As the sun proceeds northward in the ecliptic, so the sickly season advances from the southern to the northern islands. In the Mediterranean the mortality is doubled in the hot season between July and October; and in the Northern States of North America the posts of the army are regularly abandoned as the hot or sickly season approaches. In temperate regions this order is reversed. Throughout Europe generally, the maximum mortality occurs at the end of winter, and the minimum in the middle of summer. The Registrar-General of England calculates that a fall of the mean temperature of the air from  $40^{\circ}$  to  $4^{\circ}$  or  $5^{\circ}$  below the freezing-point



destroys from 300 to 500 of the population of London. The agency of the wind is manifested in the distribution of heat and moisture, and in the comparative density of the air, as well as by its direct influence as a distributor of malarial poison. The absence of wind was uniformly noted as a concomitant of cholera, which, in Britain and elsewhere, has been observed to be developed, and to be most virulent, when the calm was the greatest, and often began to abate when the wind rose.

It may not be out of place merely to indicate some definite and practically important subjects of study relative to this interesting department of medical science:

1. A study of the climatology and the diseases of the different quarters of the globe illustrate most clearly that the morbid conditions produced by certain pathological states, while they are of a fixed character, are more intense in their severity, more continuous in their development, and more prolonged in their existence, in some places than in others.

2. A study of the climatology of towns, and such circumscribed districts, is of the greatest practical importance to the physician in all questions relative to change of air for the invalid. On this subject the work of Sir James Clark *On Climate*, with its inestimable series of meteorological tables, is the classic source of reference; while many monographs have also been written of late on particular localities.\* The reader will also consult with advantage an admirable account of the effects of change of climate on diseases of the lungs, by Dr. Walshe, in the appendix to his work *On Diseases of the Lungs*.

3. Our medical officers of health are industriously mapping out the realms of disease, which often too definitely manifest themselves amongst the vilest purlieus of our cities. Systematically and energetically, many of the causes of diseases are thus more effectually exterminated. Those who have to care for the sanitary state of our armies now believe that similar means of preventing disease may be successfully applied abroad. Sanitary officers are now appointed to great military expeditions, and a board of sanitary officers is recommended, and has been established, in all the Presidencies of India. For this purpose a study of the geographical distribution of diseases, and of the causes which lead to their special distribution, obviously becomes of the greatest practical use. By it we learn that certain classes of diseases, rather than

\* Of these the following may be referred to:

*Diseases of the New Zealanders*, by Dr. Arthur Thomson. *Medico-Chirurgical Review*, commencing April, 1854.

*On the Climate of Algiers*, by Dr. Arthur Mitchell. *Medico-Chirurgical Review*, commencing January, 1858.

*A Comparative Inquiry as to the Preventive and Curative Influence of the Climate of Pau and other Places*, by Dr. Alexander Taylor.

*Change of Climate, and an Account of the Climates of Spanish Towns*, by Dr. Francis.

*On the Climate of Spain and Australia*, by Dr. Burgess. *Medico-Chirurgical Review*, October, 1854.

*Climatology of the United States*, by Blodget.

others, are mainly under the influence of terrestrial and meteorological causes—namely, those of the Zymotic class, and which are stamped with special *miasmatic* characters. In proportion, therefore, as we become capable of knowing that particular diseases of this class are limited to certain portions of the earth, and can trace the meteorological laws of their geographical distribution and diffusion, we necessarily obtain clearer conceptions of their causes and modes of propagation, as well as more practical knowledge of the means of their prevention and of cure.

By such knowledge large masses of men may be more successfully cared for in foreign countries; and the topographical position and construction of habitations may be determined upon with a certain definite knowledge to guide the chooser. Military barracks and hospitals may be judiciously provided for abroad, and with all the aids of scientific knowledge, the diet, the clothing, and the military exercising of the troops may be arranged so as to suit the physical climate of the place in which armies are to campaign or garrisons to be located.

## CONCLUSION.

IN bringing these volumes to a close (for the *fifth* time), the Author is still painfully conscious of many sins of omission and of commission in the execution of his task. Much has been left unwritten "of the thousand ills that flesh is heir to," for which it might have been desirable to find a place in these pages; but if the work shall merely serve as a guide to the acquisition of knowledge, and the acquirement of practical skill in the Art of Medicine, the Author will have accomplished his design.

After the student has read and carefully studied all that is contained in books of this kind, he will still find that he has only made a commencement of the great study and real labor of his professional life. To extend his knowledge, and to acquire practical skill, he must possess, cultivate, and foster, above all things, the faculty of observation, combined with the exercise of a sound judgment. From such a work as this, which simply transmits to the future a sketch or record of our present knowledge of the natural history of well-defined diseases and their remedies, he may acquire a *book knowledge* of the Science of Medicine; and, by clinical instruction, with such a book as his guide, and experienced clinical teachers to point out to him the best methods of investigation, he may also acquire some *practical experience*. It is such a method of study which will give the young medical man an extensive view of the Science of Medicine; and at the same time, with exactness of knowledge, some experience in the art of investigation, as well as in the direct and intelligent management of disease. It is exactness of knowledge which alone can give directness and intelligence of purpose in the treatment of diseases; and such knowledge will eventually place him in the best position to begin the practice of his profession.

It has been well observed by a veteran physician—Dr. Joseph Bullar, of Southampton—that "the treatment of disease is an ART requiring daily and hourly practice; and nothing but actual practice, with the responsibility on a man's own shoulders, can make a practical man." As this practice requires close attention, reflection, and judgment, as well as prompt action, the more a man's mental powers are educated by his college training, the better a practitioner he must in time become, if he devotes himself to his duties. Those who succeed in a marked way in acquiring a knowledge of the more theoretical parts of medicine, and who show their power strikingly in obtaining a full and exact knowledge of the natural history of diseases, and of the literature of the Science of Medicine, have sometimes been set down as not likely to be practical men. But this is an unjust conclusion—a popular fallacy. It requires only time and

the proof of success to show that, when such men apply their powers to practice, they become the most successful physicians. They bring to the bedside of the sick, minds well stored with professional knowledge—vigorous, quick, and well-cultivated mental powers, which cannot fail to place them amongst the most useful and trustworthy of practical men; in the application of remedies for the cure or relief of diseases,—the chief end and object of all our study.

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